

CARDIOLOGY

Question 1

A 17-year-old female presents with recurrent attacks of collapse. These episodes typically occur without warning and have occurred whilst she was running for a bus. There is no significant past medical history and the only family history of note is that her father died suddenly when he was 38-years-old. What is the likely cause?

- A. AVaso-vagal attacks
- B. AAnxiety
- C. AEpilepsy
- D. ACardiac syncope
- E. AMalingering

Sudden death, unusual collapse in young person - ? HOCM

This is a rather vague question. However, a family history of sudden death should make you think of conditions such as hypertrophic obstructive cardiomyopathy

HOCM: features

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins

Features

dyspnoea, angina, syncope
sudden death (most commonly due to ventricular arrhythmias), arrhythmias,
heart failure
jerky pulse, large 'a' waves, double apex beat
ejection systolic murmur: increases with Valsalva manoeuvre and decreases on squatting

Associations

Friedreich's ataxia
WPW

ECHO

systolic anterior motion (SAM) of the anterior mitral valve leaflet
asymmetric hypertrophy (ASH)
mitral regurgitation

ECG

LVH
progressive T wave inversion
deep Q waves
AF

Question 2

A 2-day-old baby girl is noted to become cyanotic whilst feeding and crying. A diagnosis of congenital heart disease is suspected. What is the most likely cause?

- A. Transposition of the great arteries
- B. Coarctation of the aorta
- C. Patent ductus arteriosus
- D. Tetralogy of Fallot 1-2 months
- E. Ventricular septal defect

Congenital heart disease

cyanotic: TGA most common at birth, Fallot's most common overall

acyanotic: VSD most common cause

The key point to this question is that whilst tetralogy of Fallot is more common than transposition of the great arteries (TGA), Fallot's doesn't usually present until 1-2 months following the identification of a murmur or cyanosis. In the neonate, TGA is the most common presenting cause of cyanotic congenital heart disease. The other 3 options are causes of acyanotic congenital heart disease

Congenital heart disease: types

Acyanotic - most common causes

ventricular septal defects (VSD) - most common, accounts for 30% atrial septal defect (ASD)

patent ductus arteriosus (PDA)

coarctation of the aorta

aortic valve stenosis

VSDs are more common than ASDs. However, in adult patients ASDs are the more common new diagnosis as they generally presents later

Cyanotic - most common causes

tetralogy of Fallot

transposition of the great arteries (TGA)

tricuspid atresia

pulmonary valve stenosis

Fallot's is more common than TGA. However, at birth TGA is the more common lesion as patients with Fallot's generally presenting at around 1-2 months

Question 3

A 26-year-old female is admitted to hospital with palpitations. ECG shows a shortened PR interval and wide QRS complexes associated with a slurred upstroke seen in lead II. What is the definitive management of this condition?

A. Accessory pathway ablation

B. Lifelong aspirin

C. AV node ablation

D. Lifelong amiodarone

E. Permanent pacemaker

This patient has Wolff-Parkinson White syndrome, with accessory pathway ablation being the definitive treatment

Wolff-Parkinson White

Wolff-Parkinson White (WPW) syndrome is caused by a congenital accessory conducting pathway between the atria and ventricles leading to a atrioventricular re-entry tachycardia (AVRT). As the accessory pathway does not slow conduction AF can degenerate rapidly to VF

Possible ECG features include:

short PR interval

wide QRS complexes with a slurred upstroke - 'delta wave'

left axis deviation if right-sided accessory pathway*

right axis deviation if left-sided accessory pathway*

Differentiating between type A and type B

type A (left-sided pathway): dominant R wave in V1

type B (right-sided pathway): no dominant R wave in V1

Associations of WPW

HOCM

mitral valve prolapse

Ebstein's anomaly

thyrotoxicosis

secundum ASD

Management

definitive treatment: radiofrequency ablation of the accessory pathway

medical therapy: sotalol**, amiodarone, flecainide

***in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation**

**sotalol should be avoided if there is coexistent atrial fibrillation as prolonging the refractory period at the AV node may increase the rate of transmission through the accessory pathway, increasing the ventricular rate and potentially deteriorating into ventricular fibrillation

Question 4

A 54-year-old man is admitted following a myocardial infarction associated with ST elevation. What advice should he be given regarding driving?

- A. Can continue driving but must inform DVLA
- B. Cannot drive until an angiogram has been performed and reviewed by a cardiologist
- C. Cannot drive for 1 week
- D. Cannot drive for 4 weeks**
- E. Cannot drive for 12 weeks

DVLA advice post MI - cannot drive for 4 weeks

DVLA: cardiovascular disorders

The guidelines below relate to car/motorcycle use unless specifically stated.

For obvious reasons, the rules relating to drivers of heavy goods vehicles tend to be much stricter

Specific rules

angioplasty (elective) - 1 week off driving

CABG - 4 weeks off driving

myocardial infarction - 4 weeks off driving

angina - driving must cease if symptoms occur at rest/at the wheel

pacemaker insertion - 1 week off driving

Question 5

A 54-year-old man is admitted to the Emergency Department with a 15 minute history of crushing central chest pain. Which one of the following rises first following a myocardial infarction?

- A. AST
- B. Troponin I
- C. CK
- D. CK-MB
- E. Myoglobin**

Myoglobin rises first following a myocardial infarction

Cardiac enzymes

Interpretation of the various cardiac enzymes has now largely been superseded by the introduction of troponin T and I. Questions still however commonly appear in the MRCP

Key points for the exam

myoglobin is the first to rise

**CK-MB is useful to look for reinfarction as it returns to normal after 2-3 days
(troponin T remains elevated for up to 10 days)**

	Begins to rise	Peak value	Returns to normal
Myoglobin	1-2 hours	6-8 hours	1-2 days
CK-MB	2-6 hours	16-20 hours	2-3 days
CK	4-8 hours	16-24 hours	3-4 days
Trop T	4-6 hours	12-24 hours	7-10 days
AST	12- 24 hours	36-48 hours	3-4 days
LDH	24 -48 hours	72 hours	8-10 days

Question 6

A 54-year-old male with no past medical history is found to be in atrial fibrillation during a consultation regarding a sprained ankle. He reports no history of palpitations or dyspnoea. After discussing treatment options he elects not to be cardioverted. If the patient remains in chronic atrial fibrillation what is the most suitable treatment to offer?

A. Aspirin

B. Warfarin, target INR 2-3

C. No anticoagulation

D. Warfarin, target INR 3-4

E. Warfarin, target INR 2-3 for six months then aspirin

Young AF, no TIA or risk factors, just give aspirin

Atrial fibrillation: anticoagulation

The Royal College of Physicians and NICE published guidelines on the management of atrial fibrillation (AF) in 2006

The guidelines suggest a stroke risk stratification approach when determining how to anticoagulate a patient, as detailed below:

Low risk - annual risk of stroke = 1%

age < 65 years with no moderate or high risk factors

use aspirin

Moderate risk - annual risk of stroke = 4%

age > 65 years with no high risk factors, or:

age < 75 years with diabetes, hypertension or vascular disease (ischaemic heart disease or peripheral arterial disease)

use aspirin or warfarin depending on individual circumstances

High risk - annual risk of stroke = 8-12%

age > 75 years with diabetes, hypertension or vascular disease (ischaemic heart disease or peripheral arterial disease)

previous TIA, ischaemic stroke or thromboembolic event
valve disease, heart failure or impaired left ventricular function use warfarin

Question 7

Which one of the following agents is most likely to cardiovert atrial fibrillation to sinus rhythm?

- A. Sotalol
- B. Procainamide
- C. Flecainide**
- D. Disopyramide
- E. Digoxin

Atrial fibrillation - cardioversion: amiodarone + flecainide

Atrial fibrillation: pharmacological cardioversion

The Royal College of Physicians and NICE published guidelines on the management of atrial fibrillation (AF) in 2006. The following is also based on the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2002 guidelines

Agents with proven efficacy in the pharmacological cardioversion of atrial fibrillation

amiodarone
flecainide
others (less commonly used in UK): quinidine, dofetilide, ibutilide,
propafenone

Less effective agents

beta-blockers (including sotalol)
calcium channel blocks
digoxin
disopyramide
procainamide

Question 8

A 34-year-old woman is admitted to the Emergency Department following a collapse. An ECG shows a polymorphic ventricular tachycardia. Which one of the following is not associated with an increased risk of developing torsade de pointes?

- A. Tricyclic antidepressants
- B. Subarachnoid haemorrhage
- C. Hypercalcaemia**
- D. Romano-Ward syndrome
- E. Hypothermia

Hypocalcaemia, not hypercalcaemia, causes prolongation of the QT interval and hence may predispose to the development of torsade de pointes

Long QT syndrome

Long QT syndrome is associated with delayed repolarization of the ventricles. It is important to recognise as it may lead to ventricular tachycardia and can therefore cause collapse/sudden death. A normal corrected QT is less than 440 ms in males and 450 ms in females

Congenital

Jervell-Lange-Nielsen syndrome (includes deafness and is due to an abnormal potassium channel)

Romano-Ward syndrome (no deafness)

Drugs

amiodarone

sotalol

class Ia antiarrhythmic drugs

tricyclic antidepressants

chloroquine

terfenadine - a non-sedating antihistamine and classic cause of prolonged QT in a patient especially if also taking P450 enzyme inhibitor, e.g. patient with cold takes terfenadine and erythromycin at same time

erythromycin

Other causes

electrolyte: **hypo**calcaemia, **hypo**kalaemia, **hypo**magnesaemia

acute MI

myocarditis

hypothermia

subarachnoid haemorrhage

Management

beta-blockers*

implantable cardioverter defibrillators in high risk cases

*note sotalol may exacerbate long QT syndrome

Question 9

A 62-year-old female with a known history of a sigmoid adenocarcinoma is admitted to hospital with shortness of breath and pyrexia. On examination a murmur is heard and an echo reveals a vegetation on the aortic valve. Which one of the following organisms is most characteristically associated with causing infective endocarditis in patients with colorectal cancer?

- A. Escherichia coli
- B. Enterococcus faecalis
- C. Salmonella
- D. Campylobacter
- E. Streptococcus bovis**

Streptococcus bovis endocarditis is associated with colorectal cancer

Infective endocarditis

Patients affected by endocarditis

previously normal valves (50%, typically acute presentation)

rheumatic valve disease (30%)
prosthetic valves
congenital heart defects
intravenous drug users (IVDUs, e.g. typically causing tricuspid lesion)

Causes

Streptococcus viridans (most common cause - 40-50%)
Staphylococcus epidermidis (especially prosthetic valves)
Staphylococcus aureus (especially acute presentation, IVDUs)
Streptococcus bovis is associated with colorectal cancer
non-infective: systemic lupus erythematosus (Libman-Sacks), malignancy:
marantic endocarditis

Culture negative causes BCCL(chlamydia,,legionella)

prior antibiotic therapy
Coxiella burnetii
Bartonella
Brucella
HACEK: Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella)

Following prosthetic valve surgery Staphylococcus epidermidis is the most common organism in the first 2 months and is usually the result of perioperative contamination. After 2 months the spectrum of organisms which cause endocarditis return to normal, except with a slight increase in Staph aureus infections

Question 10

Which one of the following treatments have not been shown to improve mortality in patients with chronic heart failure?

- A. Beta-blockers
- B. Spironolactone
- C. Frusemide**
- D. Nitrates and hydralazine
- E. Enalapril

Whilst useful in managing the symptoms of acute and chronic heart failure frusemide offers no prognostic benefits.

Heart failure: drug management

A number of drugs have been shown to improve mortality in patients with chronic heart failure:

ACE inhibitors (SAVE, SOLVD, CONSENSUS)
spironolactone (RALES)
beta-blockers (CIBIS)
hydralazine with nitrates (VHEFT-1)

Whilst spironolactone has been shown to improve prognosis in patients with chronic heart failure, no long-term reduction in mortality has been demonstrated for loop diuretics such as frusemide

NICE produced guidelines on management in 2003, key points include:

all patients should be given an ACE inhibitor unless contradictions exist

once an ACE inhibitor has been introduced a beta-blocker should be started
regardless of whether the patient is still symptomatic
offer annual influenza vaccine
offer pneumococcal vaccine

Digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties. *Digoxin is strongly indicated if there is coexistent atrial fibrillation !!!!*

Question 11

Six weeks after having a prosthetic heart valve a patient develops infective endocarditis. What is the most likely causative organism?

- A. Streptococcus viridans
- B. Staphylococcus epidermidis**
- C. Staphylococcus aureus
- D. Streptococcus bovis
- E. One of the HACEK group

Most common cause of endocarditis:

Streptococcus viridans

Staphylococcus epidermidis if < 2 months post valve surgery

In the first two months following surgery for a prosthetic valve the most likely causative organism is Staphylococcus epidermidis

Infective endocarditis

Patients affected by endocarditis

previously normal valves (50%, typically acute presentation)

rheumatic valve disease (30%)

prosthetic valves

congenital heart defects

intravenous drug users (IVDUs, e.g. typically causing tricuspid lesion)

Causes

Streptococcus viridans (most common cause - 40-50%)

Staphylococcus epidermidis (especially prosthetic valves)

Staphylococcus aureus (especially acute presentation, IVDUs)

Streptococcus bovis is associated with colorectal cancer

non-infective: systemic lupus erythematosus (Libman-Sacks), malignancy:

marantic endocarditis

Culture negative causes

prior antibiotic therapy

Coxiella burnetii

Bartonella

Brucella

HACEK: Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella)

Following prosthetic valve surgery Staphylococcus epidermidis is the most common organism in the first 2 months and is usually the result of perioperative contamination. After 2 months the spectrum of organisms which cause endocarditis return to normal, except with a slight increase in Staph aureus infections

Question 12

A 59-year-old female is admitted to the Emergency Department with a 30 minute history of central chest pain radiating to her left arm. An ECG shows ST elevation in leads II, III, aVF. Which coronary artery is most likely to be affected?

- A. Right coronary
- B. Left anterior descending
- C. Left main stem
- D. Left circumflex
- E. Anterior interventricular

Inferior MI - right coronary artery lesion

ECG: coronary territories

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	Coronary artery
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary
Anterolateral	V4-6, I, aVL	Left main stem
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Tall R waves V1-2	Usually left circumflex, also right coronary

Question 13

Each one of the following is associated with right axis deviation on ECG, except:

- A. Right ventricular hypertrophy
- B. Pulmonary embolism
- C. Wolf-Parkinson-White syndrome with right-sided accessory pathway
- D. Chronic lung disease
- E. Left posterior hemiblock

Wolff-Parkinson-White syndrome is associated with a short PR interval and a wide QRS complex with a slurred upstroke, termed a delta wave. Axis deviation depends on the position of the accessory pathway

ECG: axis deviation

Causes of left axis deviation (LAD)

left anterior hemiblock
left bundle branch block
Wolff-Parkinson-White syndrome* - right-sided accessory pathway
hyperkalaemia
congenital: ostium *primum* ASD, *tricuspid atresia*
minor LAD in obese people

Causes of right axis deviation (RAD)

right ventricular hypertrophy
left posterior hemiblock
chronic lung disease

pulmonary embolism
ostium secundum ASD
Wolff-Parkinson-White syndrome* - left-sided accessory pathway
normal in infant < 1 years old
minor RAD in tall people

*in the majority of cases, or in a question without qualification,
Wolff-Parkinson-White syndrome is associated with left axis deviation

Question 14

A 58-year-old man with no past medical history of note is admitted to hospital with crushing central chest pain. ECG on arrival shows anterior ST elevation and he is subsequently thrombolysed with a good resolution of symptoms and ECG changes. Two months following discharge from hospital, which combination of drugs should he be taking?

- A. ACE inhibitor + beta-blocker + statin + aspirin
- B. Spironolactone + beta-blocker + statin + aspirin
- C. ACE inhibitor + beta-blocker + statin + aspirin + clopidogrel
- D. ACE inhibitor + statin + aspirin + clopidogrel
- E. Beta-blocker + statin + aspirin + clopidogrel

The current guidance is to continue clopidogrel for 4 weeks following a ST-elevation myocardial infarction

Myocardial infarction: secondary prevention
NICE produced guidelines on the management of patients following a myocardial infarction (MI) in 2007. Some key points are listed below

All patients should be offered the following drugs:

ACE inhibitor
beta-blocker
aspirin
statin

Clopidogrel

after an ST-segment-elevation MI, patients treated with a combination of aspirin and clopidogrel during the first 24 hours after the MI should continue this treatment for at least 4 weeks after a non-ST segment elevation myocardial infarction clopidogrel should be given for the first 12 months

Aldosterone antagonists

patients **who have had an acute MI and who have symptoms and/or signs of heart failure and left ventricular systolic dysfunction, treatment with an aldosterone antagonist licensed for post-MI treatment should be initiated within 3–14 days of the MI**, preferably after ACE inhibitor therapy

Question 15

A 55-year-old smoker with a past history of hypertension presents to the Emergency Department with a **sudden onset of breathlessness**. Examination reveals bibasal crackles whilst the CXR shows upper lobe diversion and **perihilar shadowing**. The ECG and cardiac enzymes are normal. What is the likely cause of

his breathlessness?

- A. Infective endocarditis
- B. Pheochromocytoma
- C. Fibromuscular dysplasia
- D. Renal artery stenosis**
- E. Anterior myocardial infarction

Flash pulmonary oedema, U&Es worse on ACE inhibitor, asymmetrical kidneys --> renal artery stenosis - do MR angiography

Renal artery stenosis may cause sudden onset or 'flash' pulmonary oedema. A myocardial infarction is unlikely given the normal ECG and cardiac enzymes. Chest pain would also be expected in a 55-year-old patient with no history of diabetes. Fibromuscular dysplasia is generally seen in young woman

Renal vascular disease

Renal vascular disease is most commonly due to atherosclerosis (> 95% of patients). It is associated with risk factors such as smoking and hypertension that cause atheroma elsewhere in the body. **It may present as hypertension, chronic renal failure or 'flash' pulmonary oedema. In younger patients however fibromuscular dysplasia (FMD) needs to be considered. FMD is more common in young women and characteristically has a 'string of beads' appearance on angiography.** Patients respond well to balloon angioplasty

Investigation

MR angiography is now the investigation of choice

CT angiography

conventional renal angiography is less commonly performed used nowadays, but may still have a role when planning surgery

Question 16

A 74-year-old man presents for a medication review. Blood pressure is recorded as 184/72 mmHg. This is confirmed on two further occasions. What is the most appropriate first line therapy?

- A. Ramipril
- B. Losartan
- C. Frusemide
- D. Bendroflumethiazide**
- E. Atenolol

The 2006 NICE guidelines recommended treating isolated systolic hypertension the same way as standard hypertension. **In this age group thiazides or calcium channel blockers would be first-line agents**

Isolated systolic hypertension

Isolated systolic hypertension (ISH) is common in the elderly, affecting around 10% of people older than 70 years old. The Systolic Hypertension in the Elderly Program (SHEP) back in 1991 established that **treating ISH reduced both strokes and ischaemic heart disease.** Drugs such as thiazides were recommended as first line agents. This approach is not contraindicated by the 2006 NICE guidelines which recommends treating ISH in the same stepwise fashion as standard hypertension

Question 17

A 55-year-old man is admitted to the Emergency Department with 'tearing' chest pain radiating through to his back. Examination reveals a pulse of 96 / min regular, blood pressure of 130/85 mmHg and oxygen saturations of 97% on room air. A chest x-ray shows mediastinal widening. A CT shows dissection of the ascending aorta. What is the most suitable initial management?

- A. IV sodium nitroprusside
- B. Oral verapamil
- C. Observe only
- D. IV labetalol**
- E. Surgical repair

Aortic dissection

type A - ascending aorta - control BP(IV labetalol) + surgery
type B - descending aorta - control BP(IV labetalol)

The question tests ability to apply textbook knowledge to real world situations. Whilst surgical referral should be made as soon as possible definite surgery will inevitably take time and the blood pressure should be controlled in the meantime

Aortic dissection: management

Classification

type A - ascending aorta (2/3 of cases)

type B - descending aorta, **distal to left subclavian origin (1/3 of cases)**

Type A

surgical management, but blood pressure should be controlled to a **target systolic of 100-120 mmHg whilst awaiting intervention**

Type B*

conservative management

bed rest

reduce blood pressure IV labetalol to prevent progression

*endovascular repair of type B aortic dissection may have a role in the future

Question 18

Which one of the following is not an indication for insertion of a temporary pacemaker?

- A. Complete heart block following an inferior MI - blood pressure normal**
- B. Complete heart block following an anterior MI - blood pressure normal
- C. Trifascicular block prior to surgery
- D. Mobitz type II heart block following an anterior MI - blood pressure normal
- E. Symptomatic bradycardia not responding to drug treatment

Complete heart block following an inferior MI is NOT an indication for pacing, unlike with an anterior MI

Post-inferior MI complete heart block is common and can be managed conservatively if the patient is asymptomatic and haemodynamically stable

Pacemakers: temporary

Indications for a temporary pacemaker

symptomatic/haemodynamically unstable bradycardia, not responding to atropine post-
ANTERIOR MI: type 2 or complete heart block* trifascicular block prior to surgery

*post-INFERIOR MI complete heart block is common and can be managed conservatively if asymptomatic and haemodynamically stable

Question 19

A 65-year-old man is found to have an ejection systolic murmur and narrow pulse pressure on examination. He has experienced no chest pain, breathlessness or syncope. An echo confirms aortic stenosis and shows an aortic valve gradient of 40 mmHg. How should this patient be managed?

- A. Routine aortic valve replacement
- B. Urgent aortic valve replacement
- C. Anticoagulation
- D. Aortic valvuloplasty
- E. Regular cardiology outpatient review

Aortic stenosis management: AVR if symptomatic, otherwise cut-off is gradient of 50 mmHg

No action should be taken at present as he is currently asymptomatic. If the aortic valve gradient > 50 mmHg or there is evidence of significant left ventricular dysfunction then surgery is sometimes considered in selected asymptomatic patients

Aortic stenosis

Features of severe aortic stenosis

narrow pulse pressure
slow rising pulse
delayed ESM
soft/absent S2
S4
thrill
duration of murmur
left ventricular hypertrophy or failure

Causes of aortic stenosis

degenerative calcification (most common cause in elderly patients)
bicuspid aortic valve (most common cause in younger patients)
William's syndrome (supravalvular aortic stenosis)
post-rheumatic disease
subvalvular: HOCM

Management

if asymptomatic then observe the patient is general rule
if symptomatic then valve replacement
if asymptomatic but valvular gradient > 50 mmHg and with features such as left

ventricular systolic dysfunction then consider surgery

balloon valvuloplasty is limited to patients with critical aortic stenosis who are not fit for valve replacement

Question 20

A 24-year-old male is diagnosed as having hypertrophic obstructive cardiomyopathy. Which one of the following markers is most useful in assessing risk of sudden death?

- A. Abnormal blood pressure changes on exercise
- B. Left ventricular outflow tract gradient
- C. QT interval
- D. Right atrial diameter
- E. QRS duration

HOCM: prognostic factors

Hypertrophic obstructive cardiomyopathy (HOCM) is an **autosomal dominant** disorder of muscle tissue caused by **defects in the genes encoding contractile proteins**. Mutations to various proteins including beta-myosin, alpha-tropomyosin and troponin T have been identified. Septal hypertrophy causes left ventricular outflow obstruction. It is an important cause of sudden death in apparently healthy individuals

Poor prognostic factors

syncope

family history of sudden death

young age at presentation

non-sustained ventricular tachycardia on 24 or 48-hour Holter monitoring

abnormal blood pressure changes on exercise

An increased septal wall thickness is also associated with a poor prognosis

Question 21

A 64-year-old man with a history of ischaemic heart disease and poor left ventricular function presents with a broad complex tachycardia of 140 bpm. On examination blood pressure is 110/74 mmHg. Fusion and capture beats are seen on the 12 lead ECG. What is the first line drug management?

- A. Sotalol
- B. Amiodarone
- C. Adenosine
- D. Flecainide
- E. Lidocaine

The history of ischaemic heart disease combined with the **presence of fusion and capture beats strongly suggests a diagnosis of ventricular tachycardia (VT)**. Whilst lidocaine can also be used in VT, amiodarone would be preferred given his history of poor left ventricular function. In the 2005 joint European Resuscitation Council and Resuscitation Council (UK) guidelines **amiodarone is also considered first-line in a peri-arrest situation**

Ventricular tachycardia: management

Whilst a broad complex tachycardia may result from a supraventricular rhythm with aberrant conduction, the European Resuscitation Council advise that in a peri-arrest situation it is assumed to be ventricular in origin

If the patient has adverse signs (systolic BP < 90 mmHg, chest pain, heart failure or rate > 150 beats/min) then immediate cardioversion is indicated. In the absence of such signs antiarrhythmics may be used. If these fail, then electrical cardioversion may be needed with synchronised DC shocks

Drug therapy

amiodarone: ideally administered through a central line

lidocaine: use with caution in severe left ventricular impairment

procainamide

Verapamil should NOT be used in VT

If drug therapy fails

electrophysiological study (EPS)

implantable cardioverter-defibrillator (ICD) - this is particularly indicated in patients with significantly impaired LV function

Question 22

Which one of the following features would indicate cardiac tamponade rather than constrictive pericarditis?

- A. Raised JVP
- B. Muffled heart sounds
- C. No Y descent on JVP
- D. Hypotension
- E. Tachycardia

In cardiac tamponade there is characteristically no Y descent on the JVP. The other four features are seen in both cardiac tamponade and constrictive pericarditis

Cardiac tamponade

Features

raised JVP, with an absent Y descent - this is due to the limited right ventricular filling

tachycardia

hypotension

muffled heart sounds

pulsus paradoxus

Kussmaul's sign (much debate about this)

ECG: electrical alternans

The key differences between constrictive pericarditis and cardiac tamponade are summarised in the table below:

	Cardiac tamponade	Constrictive pericarditis
JVP	Absent Y descent	X + Y present
Pulsus paradoxus	Present	Absent
Kussmaul's sign	Rare	Present
Characteristic features		Pericardial calcification on CXR

A commonly used mnemonic to remember the absent Y descent in cardiac tamponade is TAMponade = TAMpaX

Question 23

A 76-year-old man presents with lethargy and palpitations for the past four or five days. On examination his pulse is 123 bpm irregularly irregular, blood pressure is 118/70 mmHg and his chest is clear. An ECG confirms atrial fibrillation. What is the appropriate drug to control his heart rate?

- A. Amiodarone
- B. Atenolol**
- C. Digoxin
- D. Amlodipine
- E. Flecainide

The NICE guidelines suggest either a beta-blocker or a rate limiting calcium channel blocker (i.e. not amlodipine) in this situation. Some clinicians would prefer to use a more cardio-selective beta-blocker such as bisoprolol, although this is not stipulated in current guidelines

Atrial fibrillation: rate control and maintenance of sinus rhythm

The Royal College of Physicians and NICE published guidelines on the management of atrial fibrillation (AF) in 2006. The following is also based on the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2002 guidelines

Agents used to maintain sinus rhythm in patients with atrial fibrillation

sotalol

amiodarone

flecainide

others (less commonly used in UK): disopyramide, dofetilide, procainamide, propafenone, quinidine

The table below indicates some of the factors which may be considered when considering either a rate control or rhythm control strategy

Factors favouring rate control

- Older than 65 years
- History of ischaemic heart disease

Factors favouring rhythm control

- Younger than 65 years
- Symptomatic
- First presentation
- Lone AF or AF secondary to a corrected precipitant (e.g. alcohol)
- Congestive heart failure

Question 24

A 61-year-old man is admitted with central crushing chest pain to the Emergency Department. An ECG taken immediately on arrival shows ST-elevation in leads II, III and aVF. His only past medical history of note is hypertension for which he takes ramipril, aspirin and simvastatin. What is the optimum management of this patient?

- A. Aspirin + clopidogrel + low-molecular weight heparin + repeat ECG in 20 minutes
- B. Clopidogrel + low-molecular weight heparin + alteplase
- C. Aspirin + clopidogrel + low-molecular weight heparin + tenecteplase
- D. Aspirin + clopidogrel + low-molecular weight heparin + alteplase

E. Aspirin + clopidogrel + heparin + immediate percutaneous coronary intervention

Primary percutaneous coronary intervention (PCI) is the gold-standard treatment for ST-elevation myocardial infarction

Myocardial infarction: management

A number of studies over the past 10 years have provided an evidence for the management of ST-elevation myocardial infarction (STEMI)

In the absence of contraindications, all patients should be given

aspirin

clopidogrel: the two major studies (CLARITY and COMMIT) both confirmed benefit but used different loading doses (300mg and 75mg respectively)

low molecular weight heparin:

Primary percutaneous coronary intervention (PCI) has emerged as the gold-standard treatment for STEMI but is not available in all centres. Thrombolysis should be performed in patients without access to primary PCI

With regards to thrombolysis:

tissue plasminogen activator (tPA) has been shown to offer clear mortality benefits over streptokinase tenecteplase is easier to administer and has been shown to have non-inferior efficacy to alteplase with a similar adverse effect profile

An ECG should be performed 90 minutes following thrombolysis to assess whether there has been a greater than 50% resolution in the ST elevation

if there has not been adequate resolution then rescue PCI is superior to repeat thrombolysis for patients successfully treated with thrombolysis PCI within 24 h has been shown to be beneficial

Question 25

A 52-year-old female with a known history of systemic sclerosis presents for annual review to the rheumatology clinic. Which one of the following symptoms is most characteristic in patients who have developed pulmonary arterial hypertension?

A. Exertional dyspnoea

B. Paroxysmal nocturnal dyspnoea

C. Cough

D. Early morning dyspnoea

E. Orthopnoea

Pulmonary arterial hypertension: features and management

Pulmonary arterial hypertension (PAH) may be defined as a sustained elevation in mean pulmonary arterial pressure of greater than 25 mmHg at rest or 30 mmHg after exercise.

Features

exertional dyspnoea is the most frequent symptom

chest pain and syncope may also occur

loud P2

left parasternal heave (due to right ventricular hypertrophy)

Management should first involve treating any underlying conditions, for example with anticoagulants or oxygen. Following this, it has now been shown that acute vasodilator testing is central to deciding on the appropriate management strategy. Acute vasodilator testing aims to decide which patients show a significant fall in pulmonary arterial pressure following the administration of vasodilators such as intravenous epoprostenol or inhaled nitric oxide

If there is a positive response to acute vasodilator testing
oral calcium channel blockers

If there is a negative response to acute vasodilator testing
prostacyclin analogues: treprostinil, iloprost
endothelin receptor antagonists: bosentan
phosphodiesterase inhibitors: sildenafil

Question 26

A 1-year-old girl is noted to have a continuous murmur, loudest at the left sternal edge. She is not cyanosed. A diagnosis of patent ductus arteriosus is suspected. What pulse abnormality is most associated with this condition?

- A. Collapsing pulse
- B. Bisferiens pulse
- C. Pulsus paradoxus
- D. 'Jerky' pulse
- E. Pulsus alternans

Patent ductus arteriosus

Overview

acyanotic congenital heart defect connection between the pulmonary trunk and descending aorta more common in premature babies, born at high altitude or maternal rubella infection in the first trimester

Features

left subclavicular thrill
continuous 'machinery' murmur
large volume, collapsing pulse
wide pulse pressure
heaving apex beat

Management

indomethacin closes the connection in the majority of cases
if associated with another congenital heart defect amenable to surgery then
prostaglandin E1 is useful to keep the duct open until after surgical repair

Question 27

A 54-year-old schizophrenic man is found to have torsade de pointes on his ECG recording. Blood pressure is 110/70 mmHg with a pulse of 170 bpm. What is the most appropriate management?

- A. IV beta-blocker
- B. IV verapamil
- C. IV magnesium
- D. IV amiodarone
- E. Synchronised DC cardioversion

The ALS guidelines make specific recommendations regarding the use of magnesium in torsade de pointes

Peri-arrest rhythms: tachycardia

The joint European Resuscitation Council and Resuscitation Council (UK) 2005 guidelines have simplified the advice given for the management of peri-arrest tachycardias. Separate algorithms for the management of broad-complex tachycardia, narrow complex tachycardia and atrial fibrillation have been replaced by one unified treatment algorithm

Following basic ABC assessment, patients are classified as being stable or unstable according to the presence of any adverse signs:

systolic BP < 90 mmHg
reduced conscious level
chest pain
heart failure

If any of the above adverse signs are present then synchronised DC shocks should be given

Treatment following this is given according to whether the QRS complex is narrow or broad and whether the rhythm is regular or irregular. The full treatment algorithm can be found at the Resuscitation Council website, below is a very limited summary:

Broad-complex tachycardia

Regular

assume ventricular tachycardia (unless previously confirmed SVT with bundle branch block)
loading dose of amiodarone followed by 24 hour infusion

Irregular

- 1. AF with bundle branch block** - treat as for narrow complex tachycardia
- 2. Polymorphic VT (e.g. torsade de pointes) - IV magnesium**

Narrow-complex tachycardia

Regular

vagal manoeuvres followed by IV adenosine
if above unsuccessful consider diagnosis of atrial flutter and control rate (e.g. beta-blockers)

Irregular

probable atrial fibrillation
if onset < 48 hr consider electrical or chemical cardioversion
rate control (e.g. beta-blocker or digoxin) and anticoagulation

Question 28

Which of the following congenital heart defects may progress to Eisenmenger's syndrome?

- A. Tetralogy of Fallot
- B. Coarctation of the aorta
- C. Patent ductus arteriosus**

- D. Tricuspid atresia
- E. Transposition of the great arteries

Although patients with tetralogy of Fallot have, by definition, a ventricular septal defect they do not go on to develop Eisenmenger's syndrome

Eisenmenger's syndrome

Describes the reversal of a left to right shunt in a congenital heart defect due to pulmonary hypertension

Associated with

VSD

ASD

PDA

Features

- original murmur may disappear
- cyanosis
- clubbing
- right ventricular failure
- haemoptysis, embolism

Management

heart-lung transplantation is required

Question 29

A 61-year-old woman who is normally fit and well is admitted with chest pain. An ECG shows anterolateral T wave inversion. The troponin T value at 12 hours is 0.54. On discharge her medications include aspirin, atorvastatin, bisoprolol and ramipril. Which one of the following statements best describes the role of clopidogrel in this situation?

- A. Is only given if aspirin is contraindicated
- B. Should be prescribed for life for patients < 65 years old
- C. Should be prescribed for the next 12 months for all patients**
- D. Should be prescribed for the next 12 months for patients < 65 years old
- E. Should be prescribed for life for all patients

Myocardial infarction: secondary prevention

NICE produced guidelines on the management of patients following a myocardial infarction (MI) in 2007. Some key points are listed below

All patients should be offered the following drugs:

- ACE inhibitor
- beta-blocker
- aspirin
- statin

Clopidogrel

after an ST-segment-elevation MI, patients treated with a combination of aspirin and clopidogrel during the first 24 hours after the MI should continue this treatment for at least 4 weeks after a non-ST segment elevation myocardial infarction clopidogrel should be given for the first 12 months

Aldosterone antagonists

patients who have had an acute MI and who have symptoms and/or signs of heart failure and left ventricular systolic dysfunction, treatment with an aldosterone antagonist licensed for post-MI treatment should be initiated within 3–14 days of the MI, preferably after ACE inhibitor therapy

Question 30

Which of the following is responsible for the rapid depolarisation phase of the myocardial action potential?

- A. Rapid sodium influx
- B. Slow sodium efflux
- C. Slow efflux of calcium
- D. Efflux of potassium
- E. Rapid calcium influx

Electrical activity of the heart
Myocardial action potential

<i>Phase</i>	<i>Description</i>	<i>Mechanism</i>
0	Rapid depolarization	Rapid sodium influx
1	Early repolarisation	These channels automatically deactivate after a few ms
2	Plateau	Slow influx of calcium
3	Final repolarisation	Efflux of potassium
4	Restoration of ionic concentrations	Resting potential is restored by Na ⁺ /K ⁺ ATPase There is slow entry of Na ⁺ into the cell decreasing the potential difference until the threshold potential is reached, triggering a new action potential

NB cardiac muscle remains contracted 10-15 times longer than skeletal muscle

Conduction velocity

Atrial conduction Spreads along ordinary atrial myocardial fibres at 1 m/sec

AV node conduction 0.05 m/sec

Ventricular conduction Purkinje fibres are of large diameter and achieve velocities of 2-4 m/sec (this allows a rapid and coordinated contraction of the ventricles)

passmedicine.comReference ranges End session

Question 31

Which one of the following cardiac conditions is most associated with a louder murmur following the Valsalva manoeuvre?

- A. Mitral stenosis
- B. Aortic stenosis
- C. Ventricular septal defect
- D. Hypertrophic obstructive cardiomyopathy
- E. Aortic regurgitation

HOCM: features

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins

Features

dyspnoea, angina, syncope

sudden death (most commonly due to ventricular arrhythmias), arrhythmias,

heart failure

jerky pulse, large 'a' waves, double apex beat

ejection systolic murmur: increases with Valsalva manoeuvre and decreases on squatting

Associations

Friedreich's ataxia

WPW

ECHO

systolic anterior motion (SAM) of the anterior mitral valve leaflet

asymmetric hypertrophy (ASH)

mitral regurgitation

ECG

LVH

progressive T wave inversion

deep Q waves

AF

Question 32

A 28-year-old female with a history of primary amenorrhoea and short stature is reviewed in clinic. On examination blood pressure in her right arm is 175/84 mmHg and 170/82 mmHg in her left. What is the most likely cause for her elevated blood pressure?

A. Coarctation of the aorta

B. Conn's syndrome

C. Essential hypertension

D. Renal aplasia

E. Renal artery stenosis

This patient has Turner's syndrome which is associated with coarctation of the aorta. **The site of the coarctation, for example if it involves the origin of the left subclavian artery, determines whether there is a difference between the right and left arm blood pressure readings.** There is no significant difference in this case.

Another cause worth considering in a young hypertensive patient with primary amenorrhoea would be congenital adrenal hyperplasia

Essential hypertension would be unusual in a 28-year-old

Turner's syndrome

Turner's syndrome is a **chromosomal disorder affecting around 1 in 2,500 females. It is caused by either the presence of only one sex chromosome (X) or a deletion of the short arm of one of the X chromosomes.** Turner's syndrome is denoted as 45,XO or 45,X

Features

short stature
shield chest, widely spaced nipples
webbed neck
coarctation of the aorta
primary amenorrhoea
high-arched palate
short fourth metacarpal

multiple pigmented naevi

lymphoedema in neonates (especially feet)

There is also an increased incidence of autoimmune disease (especially autoimmune thyroiditis) and Crohn's disease

Question 33

A 57-year-old man presents to the Emergency Department with palpitations for the past 36 hours. He has no past history of note. There is no associated chest pain or shortness of breath. Clinical examination is unremarkable other than an irregular tachycardia. An ECG shows atrial fibrillation at a rate of 126 bpm with no other changes. What is the most appropriate management?

- A. Beta-blocker + warfarin
- B. Immediate cardioversion in the Emergency Department
- C. Heparinise + transthoracic echo followed by electrical cardioversion during admission
- D. Beta-blocker + aspirin
- E. Warfarinise + transthoracic echo with elective electrical cardioversion in 4 weeks

This patient is a prime Candidate for electrical cardioversion

Atrial fibrillation: cardioversion

Onset < 48 hours

If atrial fibrillation (AF) is of less than 48 hours onset patients should be heparinised and a transthoracic echocardiogram and can be cardioverted immediately, either:

electrical - 'DC cardioversion'

pharmacology - amiodarone if structural heart disease, flecainide in those without structural heart disease

Following electrical cardioversion if AF is confirmed as being less than 48 hours duration then further anticoagulation is unnecessary

Onset > 48 hours

If AF is of greater than 48 hours then patients should have therapeutic anticoagulation for at least 3 weeks. If there is a high risk of cardioversion failure (e.g. previous failure or AF recurrence) then it is recommended to have at least 4 weeks amiodarone or sotalol prior to electrical cardioversion

Following electrical cardioversion patients should be anticoagulated for at least 4 weeks. After this time decisions about anticoagulation should be taken

Question 34

A 61-year-old woman is admitted to the Emergency Department with central chest pain. It feels like her previous angina but is not relieved by nitrates. She has a history of ischaemic heart disease and 4 weeks ago underwent a percutaneous coronary intervention during which a stent was placed. This is her first episode of angina since the procedure. What is the most likely diagnosis?

- A. Pericarditis
- B. Aortic dissection
- C. Coronary artery dissection
- D. Restenosis
- E. Stent thrombosis

Percutaneous coronary intervention

Percutaneous coronary intervention (PCI) is a technique used to restore myocardial perfusion in patients with ischaemic heart disease, both in patients with stable angina and acute coronary syndromes. Stents are implanted in around 95% of patients - it is now rare for just balloon angioplasty to be performed

Following stent insertion migration and proliferation of smooth muscle cells and fibroblasts occur to the treated segment. The stent struts eventually become covered by endothelium. Until this happens there is an increased risk of platelet aggregation leading to thrombosis.

Two main complications may occur

stent thrombosis: due to platelet aggregation as above. Occurs in 1-2% of patients, most commonly in the first month. *Usually presents with acute myocardial infarction*

restenosis: due to excessive tissue proliferation around stent. Occurs in around 5-20% of patients, most commonly in the first 3-6 months. Usually presents with the *recurrence of angina symptoms*. Risk factors include diabetes, renal impairment and stents in venous bypass grafts

Types of stent

bare-metal stent (**BMS**)

drug-eluting stents (**DES**): stent coated with paclitaxel or rapamycin which inhibit local tissue growth. Whilst this reduces restenosis rates the stent thrombosis rates are increased as the process of stent endothelialisation is slowed

Following insertion the most important factor in preventing stent thrombosis is antiplatelet therapy. Aspirin should be continued indefinitely. The length of clopidogrel treatment depends on the type of stent, reason for insertion and consultant preference

Question 35

A 17-year-old male is taken to the Emergency Department due to alcohol intoxication. On examination he is noted to be tachycardic with a rate of 140bpm. An ECG shows atrial fibrillation. The following morning he is noted to be in sinus rhythm. What is the most appropriate management?

- A. Sotalol and aspirin
- B. Sotalol and warfarin
- C. Refer for accessory pathway ablation
- D. Amiodarone and aspirin
- E. Discharge**

Supraventricular arrhythmias secondary to acute alcohol intake are well characterised and have been termed '**holiday heart syndrome**'. No specific treatment is required

Atrial fibrillation: classification

An attempt was made in the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2002 guidelines to simplify and clarify the classification of atrial fibrillation (AF).

It is recommended that AF be classified into 3 patterns:

first detected episode (irrespective of whether it is symptomatic or self-terminating)
recurrent episodes, when a patient has 2 or more episodes of AF. If episodes of AF terminate spontaneously then the term paroxysmal AF is used. Such episodes last less than 7 days (typically < 24 hours). If the arrhythmia is not self-terminating then the term persistent AF is used. Such episodes usually last greater than 7 days
in permanent AF there is continuous atrial fibrillation which cannot be cardioverted or if attempts to do so are deemed inappropriate. Treatment goals are therefore rate control and anticoagulation if appropriate

Question 36

What is the most common cardiac defect seen in patients with Down's syndrome?

- A. Ventricular septal defect
- B. Endocardial cushion defect**
- C. Secundum atrial septal defect
- D. Tetralogy of Fallot
- E. Patent ductus arteriosus

Endocardial cushion defects account for about 40% of congenital heart disease seen in patients with Down's syndrome

Down syndrome: features

Clinical features

face: upslanting palpebral fissures, epicanthic folds, Brushfield spots in iris, protruding tongue, small ears, round/flat face

flat occiput

single palmar crease, pronounced 'sandal gap' between big and first toe

hypotonia

congenital heart defects (40-50%, see below)

duodenal atresia

Hirschsprung's disease

Cardiac complications

multiple cardiac problems may be present

endocardial cushion defect (c. 40%, also known as atrioventricular septal canal defects)

ventricular septal defect (c. 30%)

secundum atrial septal defect (c. 10%)

tetralogy of Fallot (c. 5%)

isolated patent ductus arteriosus (c. 5%)

Later complications

subfertility: males are almost always infertile due to impaired spermatogenesis. Females are usually subfertile, and have an increased incidence of problems with pregnancy and labour

learning difficulties

short stature

repeated respiratory infections (+hearing impairment from glue ear)

acute lymphoblastic leukaemia

hypothyroidism

Alzheimer's

atlantoaxial instability

Question 37

A 62-year-old man is referred from the Emergency Department with a pulse of 40 beats/min. Which one of the following factors carries the least risk of asystole when risk stratifying the patient?

A. Ventricular pause of 5 seconds

B. Recent asystole

C. Complete heart block with a narrow complex QRS

D. Mobitz type II AV block

E. Complete heart block with a broad complex QRS

Complete heart block with a narrow complex QRS complex carries the least risk of asystole as the atrioventricular junctional pacemaker may provide an haemodynamically acceptable and stable heart rate. The other four factors are indications for transvenous pacing

Peri-arrest rhythms: bradycardia

The joint European Resuscitation Council and Resuscitation Council (UK) 2005 guidelines emphasise that the management of bradycardia depends on:

1. identifying the presence of signs indicating haemodynamic compromise - 'adverse signs'
2. identifying the potential risk of asystole

Adverse signs

The following factors indicate haemodynamic compromise and hence the need for treatment:

heart rate < 40 bpm

systolic blood pressure < 100 mmHg

heart failure

ventricular arrhythmias requiring suppression

Atropine is the first line treatment in this situation. If this fails to work, or there is the potential risk of asystole then transvenous pacing is indicated

Potential risk of asystole

The following indicate a potential risk of asystole and hence the need for treatment with transvenous pacing:

complete heart block with broad complex QRS
recent asystole
Mobitz type II AV block
ventricular pause > 3 seconds

If there is a delay in the provision of transvenous pacing the following interventions may be used:

atropine, up to maximum of 3mg
transcutaneous pacing
adrenaline infusion titrated to response
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Question 38

Which one of the following features is not part of the modified Duke criteria used in the diagnosis of infective endocarditis?

- A. Fever > 38°C
- B. Positive molecular assays for specific gene targets
- C. Indwelling central line**
- D. Elevated CRP or ESR
- E. Janeway lesions

The modified Duke criteria have now been adopted in the latest guidelines from the European Society of Cardiology. Details can be found in the link below

Infective endocarditis: Modified Duke criteria

Infective endocarditis diagnosed if

pathological criteria positive, or
2 major criteria, or
1 major and 3 minor criteria, or
5 minor criteria

Pathological criteria

Positive histology or microbiology of pathological material obtained at autopsy or cardiac surgery (valve tissue, vegetations, embolic fragments or intracardiac abscess content)

Major criteria

Positive blood cultures

*two positive blood cultures showing typical organisms consistent with infective endocarditis, such as Streptococcus viridans and the HACEK group, or persistent bacteraemia from two blood cultures taken > 12 hours apart or **three or more positive blood cultures where the pathogen is less specific such as Staph aureus and Staph epidermidis,** or positive serology for Coxiella burnetii, Bartonella species or Chlamydia psittaci, or positive molecular assays for specific gene targets*

Evidence of endocardial involvement

positive echocardiogram (**oscillating structures, abscess formation, new valvular regurgitation or dehiscence of prosthetic valves**), or new valvular regurgitation

Minor criteria

predisposing heart disease

microbiological evidence does not meet major criteria

fever $> 38^{\circ}\text{C}$

vascular phenomena: major emboli, splenomegaly, clubbing, splinter haemorrhages, petechiae or purpura

immunological phenomena: glomerulonephritis, Osler's nodes, Roth spots

elevated CRP or ESR

Question 39

A 45-year-old man is diagnosed with endocarditis of the aortic valve. He is treated with intravenous benzylpenicillin and gentamicin. What is the most important ECG change to monitor for?

- A. Left ventricular hypertrophy (by voltage criteria)
- B. Reflex tachycardia
- C. ST segment depression
- D. Prolonged QT interval
- E. Prolonged PR interval**

A prolonged PR interval could indicate the development of an aortic abscess, an indication for surgery

ECG: PR interval

Causes of a prolonged PR interval

idiopathic

ischaemic heart disease

digoxin toxicity

hypokalaemia*

rheumatic fever

aortic root pathology e.g. abscess secondary to endocarditis

Lyme disease

sarcoidosis

myotonic dystrophy

A prolonged PR interval may also be seen in athletes

*hyperkalaemia can rarely cause a prolonged PR interval, but this is a much less common association than hypokalaemia

Question 40

A 65-year-old female with a known history of heart failure presents for an annual check-up. She is found to have a blood pressure of 170/100 mmHg. Her current medications are frusemide and aspirin. What is the most appropriate medication to add?

- A. Bendroflumethiazide
- B. Spironolactone
- C. Bisoprolol
- D. Verapamil

E. Enalapril

Both enalapril and spironolactone have been shown to improve prognosis in patients with heart failure. Enalapril however would also treat the hypertension. The SOLVD and CONSENSUS studies have demonstrated the benefit of enalapril in patients with heart failure. **NICE guidelines recommend the introduction of an ACE inhibitor prior to a beta-blocker in patients with chronic heart failure**

Heart failure: drug management

A number of drugs have been shown to improve mortality in patients with chronic heart failure:

ACE inhibitors (SAVE, SOLVD, CONSENSUS)
spironolactone (RALES)
beta-blockers (CIBIS)
hydralazine with nitrates (VHEFT-1)

Whilst spironolactone has been shown to improve prognosis in patients with chronic heart failure, no long-term reduction in mortality has been demonstrated for loop diuretics such as frusemide

NICE produced guidelines on management in 2003, key points include:

all patients should be given an ACE inhibitor unless contradictions exist once an ACE inhibitor has been introduced a beta-blocker should be started regardless of whether the patient is still symptomatic
offer annual influenza vaccine
offer pneumococcal vaccine

Digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties. Digoxin is strongly indicated if there is coexistent atrial fibrillation

Question 41

Which one of the following is not a recognised treatment in primary pulmonary hypertension?

- A. Endothelin-1 receptor agonists**
- B. Heart-lung transplant
- C. IV prostaglandins
- D. Diuretics
- E. Calcium channel blockers

Endothelin-1 receptor antagonists such as bosentan, not agonists may be used

Primary pulmonary hypertension

The classification of pulmonary hypertension is currently changing with the term idiopathic pulmonary arterial hypertension (IPAH) becoming more widely used

Primary pulmonary hypertension (PPH, now IPAH)

pulmonary arterial pressure > 25 mmHg at rest, > 30mmHg with exercise PPH is diagnosed when no underlying cause can be found around 10% of cases are familial: autosomal dominant endothelin thought to play a key role in pathogenesis associated with HIV, cocaine and anorexigens (e.g. fenfluramine)

Features

more common in females, typically presents at 20-40 years old
progressive SOB
cyanosis
right ventricular heave, loud P2, raised JVP with **prominent 'a' waves**,
tricuspid regurgitation

Management

diuretics if right heart failure
anticoagulation
vasodilator therapy: calcium channel blocker, IV prostaglandins, bosentan:
endothelin-1 receptor antagonist
heart-lung transplant

Question 42

Which one of the following conditions is most associated with a bisferiens pulse?

- A. Cardiac tamponade
- B. Severe left ventricular failure
- C. Aortic stenosis
- D. Patent ductus arteriosus
- E. Mixed aortic valve disease

Pulsus paradoxus

greater than the normal (10 mmHg) fall in systolic blood pressure during inspiration --> faint or absent pulse in inspiration
severe asthma, cardiac tamponade

Slow-rising/plateau

aortic stenosis

Collapsing

aortic regurgitation
patent ductus arteriosus
hyperkinetic (anaemia, thyrotoxic, fever, exercise/pregnancy) hyperdynamic circulation

Pulsus alternans

regular alternation of the force of the arterial pulse
severe LVF

Bisferiens pulse

'double pulse' - two systolic peaks
mixed aortic valve disease

'Jerky' pulse

hypertrophic obstructive cardiomyopathy*

*HOCM may occasionally be associated with a bisferiens pulse

Question 43

Which one of the following drugs causes shortening of the QT interval?

- A. Digoxin
- B. Sotalol
- C. Amiodarone
- D. Tricyclic antidepressants
- E. Chloroquine

Digoxin causes shortening of the QT interval whilst the other four drugs cause QT prolongation

Digoxin toxicity

Features

generally unwell, lethargy, N/V, confusion, yellow-green vision
arrhythmias (e.g. AV block, bradycardia)

Precipitating factors

classically: **hypokalaemia***

myocardial ischaemia

hypomagnesaemia, hypercalcaemia, hypernatraemia, acidosis

hypoalbuminaemia

hypothermia

hypothyroidism

drugs: amiodarone, quinidine, verapamil, spironolactone (compete for secretion in distal convoluted tubule therefore reduce excretion)

Management

Digibind

correct arrhythmias

monitor K⁺

*hyperkalaemia may also worsen digoxin toxicity

Question 44

Which one of the following would not be considered a normal variant on the ECG of an athletic 28-year-old man?

- A. Wenckebach phenomenon
- B. Sinus bradycardia
- C. Junctional rhythm
- D. First degree heart block
- E. LBBB

ECG: normal variants

The following ECG changes are considered normal variants in an athlete:

sinus bradycardia

junctional rhythm

first degree heart block

Wenckebach phenomenon

Question 45

Which one of the following statements regarding B-type natriuretic peptide is incorrect?

- A. Effective treatment for heart failure lowers a patients BNP level
- B. Acts as a diuretic
- C. A hormone produced mainly by the left ventricular myocardium in response to strain
- D. Is a good marker of prognosis in patients with chronic heart failure
- E. The positive predictive value of BNP is greater than the negative predictive value

BNP has a good negative predictive value rather than positive predictive value

B-type natriuretic peptide (BNP) hormone produced mainly by the left ventricular myocardium in response to strain

Whilst heart failure is the most obvious cause of raised BNP levels any cause of left ventricular dysfunction such as **myocardial ischaemia or valvular disease may raise levels.**

Raised levels may also be seen due to reduced excretion in patients with **chronic kidney disease**. Factors which **reduce BNP levels** include treatment with **ACE inhibitors, angiotensin-2 receptor blockers and diuretics.**

Effects of BNP

vasodilator

diuretic and natriuretic

suppresses both sympathetic tone and the renin-angiotensin system

Clinical uses of BNP

Diagnosing patients with acute dyspnoea

a low concentration of BNP(< 100pg/ml) makes a diagnosis of heart failure unlikely, but raised levels should prompt further investigation to confirm the diagnosis

NICE currently recommends BNP as a helpful test to rule out a diagnosis of heart failure

Prognosis in patients with chronic heart failure

initial evidence suggests BNP is an extremely useful marker of prognosis

Guiding treatment in patients with chronic heart failure effective treatment lowers BNP levels

Screening for cardiac dysfunction

not currently recommended for population screening

Question 46

Which of the following is responsible for the plateau phase of the myocardial action potential?

- A. Slow calcium efflux
- B. Efflux of potassium
- C. Rapid sodium influx
- D. Slow influx of calcium
- E. Slow sodium efflux

Slow influx of calcium is responsible for the plateau phase of the action potential

Electrical activity of the heart

Myocardial action potential

Phase	Description	Mechanism
0	Rapid depolarization	Rapid sodium influx These channels automatically after few ms

- | | | |
|---|-------------------------------------|---|
| 1 | Early repolarisation | Efflux of potassium |
| 2 | Plateau | Slow influx of calcium |
| 3 | Final repolarisation | Efflux of potassium |
| 4 | Restoration of ionic concentrations | Resting potential is restored by Na ⁺ /K ⁺ ATPase
There is slow entry of Na ⁺ into the cell decreasing the potential difference until the threshold potential is reached, triggering a new action potential |

NB cardiac muscle remains contracted 10-15 times longer than skeletal muscle Conduction velocity

Atrial conduction Spreads along ordinary atrial myocardial fibres at 1 m/sec

AV node conduction 0.05 m/sec

Ventricular conduction Purkinje fibres are of large diameter and achieve velocities of 2-4 m/sec (this allows a rapid and coordinated contraction of the ventricle).

Question 47

A patient with severe aortic stenosis is noted to have a fourth heart sound. Which part of the ECG does this best correlate with?

- A. U wave
- B. QRS complex
- C. P wave
- D. ST segment
- E. T wave

Heart sounds

The first heart sound (S1) is caused by closure of the mitral and tricuspid valves whilst the second heart sound (S2) is due to aortic and pulmonary valve closure

S1

closure of mitral and tricuspid valves

soft if long PR or mitral regurgitation

loud in mitral stenosis

S2

closure of aortic and pulmonary valves

soft in aortic stenosis

splitting during inspiration is normal

S3

caused by diastolic filling of the ventricle

considered normal if < 30 years old (may persist in women up to 50 years old)

heard in left ventricular failure, constrictive pericarditis

S4

may be heard in aortic **stenosis, HOCM, hypertension**
caused by atrial contraction against a stiff ventricle
in HOCM a double apical impulse may be felt as a result of a palpable S4
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Question 48

Which part of the jugular venous waveform is associated with the closure of the tricuspid valve?

- A. a wave
- B. c wave**
- C. x descent
- D. y descent
- E. v wave

JVP: C wave - closure of the tricuspid valve

Jugular venous pulse

As well as providing information on right atrial pressure, the jugular vein waveform may provide clues to underlying valvular disease. **A non-pulsatile JVP is seen in superior vena caval obstruction. Kussmaul's sign describes a paradoxical rise in JVP during inspiration seen in constrictive pericarditis**

'a' wave = **a**trial contraction

large if atrial pressure e.g. *tricuspid stenosis, pulmonary stenosis, pulmonary hypertension*

absent if in atrial fibrillation

Cannon 'a' waves giant a wave

caused by atrial contractions against a closed tricuspid valve

are seen in **complete heart block, ventricular tachycardia/ectopics, nodal rhythm, single chamber ventricular pacing**

'c' wave

closure of tricuspid valve
not normally visible

'v' wave

giant v waves in tricuspid regurgitation

'x' descent = fall in atrial pressure during ventricular systole

'y' descent = opening of tricuspid valve

Question 49

A 76-year-old female is admitted after being found on the floor at her home. On examination she has a core temperature of 30°C. Her serum electrolytes are within normal range. Which one of the ECG findings is most likely to be seen?

- A. Long QT interval
- B. 'U' waves
- C. Short PR interval
- D. Second degree heart block
- E. Flattened T waves

ECG: hypothermia

The following ECG changes may be seen in hypothermia

bradycardia

'J' wave - small hump at the end of the QRS complex

first degree heart block

long QT interval

atrial and ventricular arrhythmias

Question 50

A 54-year-old man with angina has a percutaneous coronary intervention with insertion of a drug-eluting stent. What is the single most important risk factor for stent thrombosis?

- A. Age of patient
- B. Premature withdrawal of antiplatelet therapy
- C. Failing to adhere to cardiac rehabilitation program
- D. Duration of procedure
- E. History of diabetes mellitus

PCI: stent thrombosis - withdrawal of antiplatelets biggest risk factor

Diabetes mellitus is a risk factor for restenosis rather than stent thrombosis

Percutaneous coronary intervention

Percutaneous coronary intervention (PCI) is a technique used to restore myocardial perfusion in patients with ischaemic heart disease, both in patients with stable angina and acute coronary syndromes. Stents are implanted in around 95% of patients - it is now rare for just balloon angioplasty to be performed

Following stent insertion migration and proliferation of smooth muscle cells and fibroblasts occur to the treated segment. The stent struts eventually become covered by endothelium. Until this happens there is an increased risk of platelet aggregation leading to thrombosis.

Two main complications may occur

stent thrombosis: due to platelet aggregation as above. Occurs in 1-2% of patients, most commonly in the first month. Usually presents with acute myocardial infarction

restenosis: due to excessive tissue proliferation around stent. Occurs in around 5-20% of patients, most commonly in the first 3-6 months. Usually presents with the recurrence of angina symptoms. Risk factors include diabetes, renal impairment and stents in venous bypass grafts

Types of stent

bare-metal stent (BMS)

drug-eluting stents (DES): stent coated with paclitaxel or rapamycin which inhibit local tissue growth. Whilst this reduces restenosis rates the stent thrombosis rates are increased as the process of stent endothelialisation is slowed

Following insertion the most important factor in preventing stent thrombosis is antiplatelet therapy. Aspirin should be continued indefinitely. The length of clopidogrel treatment depends on the type of stent, reason for insertion and consultant preference

Question 51

Each one of the following physiological changes occur during exercise, except:

- A. Increased myocardial contractibility
- B. 50% increase in stroke volume
- C. Up to 3-fold increase in heart rate
- D. Rise in diastolic blood pressure**
- E. Venous constriction

Exercise: physiological changes

Blood pressure

systolic increases, diastolic decreases leads to increased pulse pressure in healthy young people the increase in MABP is only slight

Cardiac output

increase in cardiac output may be 3-5 fold results from venous constriction, vasodilation and increased myocardial contractibility, as well as from the maintenance of right atrial pressure by an increase in venous return heart rate up to 3-fold increase stroke volume up to 1.5-fold increase

Question 52

Pulmonary arterial hypertension may be seen in each one of the following conditions, except:

- A. Hepatitis B**
- B. Eisenmenger's syndrome
- C. Sickle cell anaemia
- D. HIV
- E. Sarcoidosis

Hepatitis B is not a recognised cause of pulmonary arterial hypertension

Pulmonary arterial hypertension: causes and classification

Pulmonary arterial hypertension (PAH) may be defined as a sustained elevation in mean pulmonary arterial pressure of greater than 25 mmHg at rest or 30 mmHg after exercise. PAH has recently been reclassified by the WHO:

Group 1: Pulmonary arterial hypertension (PAH)

- idiopathic*
- familial
- associated conditions: collagen vascular disease, congenital heart disease with systemic to pulmonary shunts, HIV**, drugs and toxins, sickle cell disease
- persistent pulmonary hypertension of the newborn

Group 2: Pulmonary hypertension with left heart disease

- left-sided atrial, ventricular or valvular disease such as left ventricular systolic and diastolic dysfunction, mitral stenosis and mitral regurgitation

Group 3: Pulmonary hypertension secondary to lung disease/hypoxia

- COPD
- interstitial lung disease
- sleep apnoea
- high altitude

Group 4: Pulmonary hypertension due to thromboembolic disease

Group 5: Miscellaneous conditions

– **lymphangiomatosis e.g. secondary to carcinomatosis or sarcoidosis**

*previously termed primary pulmonary hypertension

**the mechanism by which HIV infection produces pulmonary hypertension remains unknown

Question 53

A 34-year-old man is noted to have a pan-systolic murmur associated with large V waves in the JVP and pulsatile hepatomegaly. Which one of the following types of congenital heart disease is most associated with tricuspid regurgitation?

- A. Atrial septal defect
- B. Ebstein's anomaly**
- C. Coarctation of the aorta
- D. Patent ductus arteriosus
- E. Ventricular septal defect

Tricuspid regurgitation

Signs

pan-systolic murmur
giant V waves in JVP
pulsatile hepatomegaly
left parasternal heave

Causes

right ventricular dilation
pulmonary hypertension e.g. COPD
rheumatic heart disease
infective endocarditis (especially intravenous drug users)
Ebstein's anomaly
carcinoid syndrome

Question 54

A 64-year-old man is admitted to the Emergency Department with chest pain radiating through to his back. On examination pulse 90 regular, BP 140/90. A CXR shows mediastinal widening. A CT shows dissection of the descending aorta. What is the most suitable initial management?

- A. Observe only
- B. IV labetalol**
- C. IV sodium nitroprusside
- D. Immediate surgical referral
- E. Oral verapamil

Aortic dissection

type A - ascending aorta - control BP(IV labetalol) + surgery

type B - descending aorta - control BP(IV labetalol)

Dissection of the descending aorta indicates a type B dissection, which should be managed medically with IV labetalol

Aortic dissection: management

Classification

type A - ascending aorta (2/3 of cases)

type B - descending aorta, distal to left subclavian origin (1/3 of cases)

Type A

surgical management, but blood pressure should be controlled to a target systolic of 100-120 mmHg whilst awaiting intervention

Type B*

conservative management

bed rest

reduce blood pressure IV labetalol to prevent progression

*endovascular repair of type B aortic dissection may have a role in the future

Question 55

A 72-year-old man presents to the Emergency Department with a broad complex tachycardia. Which of the following features would make it more likely that this was due to a supraventricular tachycardia rather than a ventricular tachycardia?

A. History of ischaemic heart disease

B. Left axis deviation

C. Capture beats

D. Absence of QRS concordance in chest leads

E. QRS complex greater than 160 ms

Positive QRS concordance in the chest leads is associated with ventricular tachycardia

Broad complex tachycardia

Features suggesting VT rather than SVT with aberrant conduction

AV dissociation

fusion or capture beats

positive QRS concordance in chest leads

marked left axis deviation

history of IHD

lack of response to adenosine or carotid sinus massage

QRS > 160 ms

Question 56

Which one of the following ECG findings is least associated with digoxin use?

A. Bradycardia

B. Down-sloping ST depression

- C. Flattened T waves
- D. Prolonged QT interval**
- E. AV block

ECG: digoxin

ECG features

down-sloping ST depression ('reverse tick')
flattened/inverted T waves
short QT interval
arrhythmias e.g. AV block, bradycardia

Question 57

Which one of the following conditions is most associated with pulsus alternans?

- A. Cardiac tamponade
- B. Hypertrophic obstructive cardiomyopathy
- C. Aortic stenosis
- D. Severe left ventricular failure**
- E. Mixed aortic valve disease

Pulses

Pulsus paradoxus

greater than the normal (10 mmHg) fall in systolic blood pressure during inspiration --> faint or absent pulse in inspiration
severe asthma, cardiac tamponade

Slow-rising/plateau

aortic stenosis

Collapsing

aortic regurgitation
patent ductus arteriosus
hyperkinetic (anaemia, thyrotoxic, fever, exercise/pregnancy)

Pulsus alternans

regular alternation of the force of the arterial pulse
severe LVF

Bisferiens pulse

'double pulse' - two systolic peaks
mixed aortic valve disease

'Jerky' pulse

hypertrophic obstructive cardiomyopathy*

*HOCM may occasionally be associated with a bisferiens pulse

Question 58

Which one of the following is least likely to cause dilated cardiomyopathy?

- A. Wilson's disease**
- B. Haemochromatosis
- C. Coxsackie A
- D. Hypertension

E. Alcohol

Haemochromatosis is more commonly associated with restrictive cardiomyopathy but a dilated pattern may also be seen. There is a known association between Wilson's disease and cardiomyopathy but this is extremely rare and not often clinically significant

Dilated cardiomyopathy

Basics

dilated heart leading to systolic (+/- diastolic) dysfunction
all 4 chambers affected but LV more so than RV
features include arrhythmias, emboli, mitral regurgitation
absence of congenital, valvular or ischaemic heart disease

Causes often considered separate entities

alcohol: may improve with thiamine

postpartum

hypertension

Other causes

infections e.g. coxsackie A and B, HIV, diphtheria, parasitic
endocrine e.g. hyperthyroidism
infiltrative* e.g. haemochromatosis, sarcoidosis
neuromuscular e.g. Duchenne muscular dystrophy
nutritional e.g. kwashiorkor, pellagra, thiamine/selenium deficiency
drugs e.g. doxorubicin

*these causes may also lead to restrictive cardiomyopathy

Question 59

Which of the following is a cause of a soft second heart sound?

- A. Aortic stenosis**
- B. Aortic regurgitation
- C. Mitral stenosis
- D. Mitral regurgitation
- E. Pulmonary hypertension

Second heart sound (S2)

loud: hypertension

soft: AS

fixed split: ASD

reversed split: LBBB

S2 is soft in severe aortic stenosis

Heart sounds: S2

S2 is caused by the closure of the aortic valve (A2) closely followed by that of the pulmonary valve (P2)

Causes of a loud S2

hypertension: systemic (loud A2) or pulmonary (loud P2)

hyperdynamic states

atrial septal defect without pulmonary hypertension

Causes of a soft S2
aortic stenosis

Causes of fixed split S2
atrial septal defect

Causes of a widely split S2
deep inspiration
RBBB
pulmonary stenosis
severe mitral regurgitation

Causes of a reversed (paradoxical) split S2 (P2 occurs before A2)
LBBB
severe aortic stenosis
right ventricular pacing
WPW type B (causes early P2)
patent ductus arteriosus

Question 60

Which one of the following is least associated with ST depression on ECG?

- A. Myocardial ischaemia
- B. Syndrome X
- C. Acute pericarditis**
- D. Hypokalaemia
- E. Digoxin

ECG: ST depression

Causes of ST depression

normal if upward sloping

ischaemia
digoxin
hypokalaemia
syndrome X

Question 61

A 72-year-old man with a history of chronic heart failure secondary to ischaemic cardiomyopathy is reviewed. Despite his current treatment with frusemide, ramipril, carvedilol, aspirin and simvastatin he remains short of breath on minimal exertion such as walking 30 metres. On examination his chest is clear and there is minimal peripheral oedema.

What is the most appropriate next management step?

- A. Stop aspirin
- B. Refer for cardiac resynchronisation therapy
- C. Switch carvedilol to bisoprolol
- D. Add angiotensin-2 receptor blocker
- E. Add spironolactone**

NICE guidelines would suggest adding spironolactone in this situation

Heart failure: drug management

A number of drugs have been shown to improve mortality in patients with chronic heart failure:

- ACE inhibitors (SAVE, SOLVD, CONSENSUS)
- spironolactone (RALES)
- beta-blockers (CIBIS)
- hydralazine with nitrates (VHEFT-1)

Whilst spironolactone has been shown to improve prognosis in patients with chronic heart failure, no long-term reduction in mortality has been demonstrated for loop diuretics such as frusemide

NICE produced guidelines on management in 2003, key points include:

- all patients should be given an ACE inhibitor unless contradictions exist once an ACE inhibitor has been introduced a beta-blocker should be started regardless of whether the patient is still symptomatic
- offer annual influenza vaccine
- offer pneumococcal vaccine

Digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties.

Digoxin is strongly indicated if there is coexistent atrial fibrillation

Question 62

A 51-year-old female presents to the Emergency Department following an episode of transient right sided weakness lasting 5-10 minutes. Examination reveals the patient to be in atrial fibrillation. If the patient remains in chronic atrial fibrillation what is the most suitable form of anticoagulation?

- A. Aspirin
- B. Warfarin, target INR 2-3**
- C. No anticoagulation
- D. Warfarin, target INR 3-4
- E. Warfarin, target INR 2-3 for six months then aspirin

Atrial fibrillation: anticoagulation

The Royal College of Physicians and NICE published guidelines on the management of atrial fibrillation (AF) in 2006

The guidelines suggest a stroke risk stratification approach when determining how to anticoagulate a patient, as detailed below:

Low risk - annual risk of stroke = 1%

- age < 65 years with no moderate or high risk factors
- use aspirin

Moderate risk - annual risk of stroke = 4%

- age > 65 years with no high risk factors, or:
- age < 75 years with diabetes, hypertension or vascular disease (ischaemic heart disease or peripheral arterial disease)
- use aspirin or warfarin depending on individual circumstances

High risk - annual risk of stroke = 8-12%

age > 75 years with diabetes, hypertension or vascular disease (ischaemic heart disease or peripheral arterial disease) previous TIA, ischaemic stroke or thromboembolic event valve disease, heart failure or impaired left ventricular function use warfarin

Question 63

You are called to assess a man who has collapsed in the clinic waiting room. The receptionist has already called 999. On arrival the man is laid on his back. You open the airway with a head-tilt chin lift - after assessing for 10 seconds there are no signs of breathing. What is the most appropriate next step?

- A. Start chest compressions at a ratio of 15:2
- B. Place in the recovery position
- C. Check for a carotid pulse for 10 seconds
- D. Give 2 rescue breaths
- E. Start chest compressions at a ratio of 30:2**

The 2005 guidelines removed the concept of 'checking for circulation'; absence of breathing, in a non-responsive individual, is now used as the main sign of cardiac arrest. In reality most medical professionals will check for a carotid pulse whilst assessing circulation, but in this scenario to wait a further 10 seconds before starting chest compressions is not justifiable. Please see the link to the BLS guidelines

Adult advanced life support

The joint European Resuscitation Council and Resuscitation Council (UK) 2005 guidelines propose radical changes in the provision of advanced life support (ALS)

Major changes include

**2 initial 'rescue breaths' are no longer given
ratio of chest compressions to ventilation changed from 15:2 to 30:2
a single shock for VF/pulseless VT followed by 2 minutes of CPR, rather than a series of 3 shocks followed by 1 minute of CPR
asystole/pulseless-electrical activity should be treated with 2 minutes of CPR, rather than 3, prior to reassessment of the rhythm**

Question 64

A 67-year-old man with a history of chronic obstructive pulmonary disease and ischaemic heart disease is taken to the Emergency Department with dyspnoea. On examination his respiratory rate is 24 / min, JVP is not elevated and crackles are heard in both lung bases. Which other finding would most strongly indicate that his dyspnoea is secondary to isolated left ventricular failure?

- A. Pulsus alternans
- B. Gallop rhythm**
- C. Tachycardia
- D. Peripheral oedema
- E. Cardiomegaly on chest x-ray

Gallop rhythm (S3) is an early sign of LVF

Whilst all of the above features may be seen in patients with left ventricular failure a **gallop rhythm is one of the most specific and early signs**

Heart sounds

The first heart sound (S1) is caused by closure of the mitral and tricuspid valves whilst the second heart sound (S2) is due to aortic and pulmonary valve closure

S1

closure of mitral and tricuspid valves
soft if long PR or mitral regurgitation
loud in mitral stenosis

S2

closure of aortic and pulmonary valves
soft in aortic stenosis
splitting during inspiration is normal

S3

caused by diastolic filling of the ventricle
considered normal if < 30 years old (may persist in women up to 50 years old)
heard in left ventricular failure, constrictive pericarditis

S4

may be heard in aortic stenosis, HOCM, hypertension
caused by atrial contraction against a stiff ventricle
in HOCM a double apical impulse may be felt as a result of a palpable S4

Question 65

A 57-year-old man who had a prosthetic mitral valve replacement 7 years ago presents with fever. An urgent echocardiogram shows features consistent with endocarditis. What is the most suitable antibiotic therapy until blood culture results are known?

- A. IV ceftriaxone + benzylpenicillin
 - B. IV vancomycin + rifampicin + gentamicin**
 - C. IV benzylpenicillin + gentamicin
 - D. IV flucloxacillin + gentamicin
 - E. IV vancomycin + benzylpenicillin
- Infective endocarditis: prognosis and management

Poor prognostic factors

Staph aureus infection (see below)
prosthetic valve (especially 'early', acquired during surgery)
culture negative endocarditis
low complement levels

Mortality according to organism

staphylococci - 30%
bowel organisms - 15%
streptococci - 5%

Current management guidelines (source: British National Formulary)

initial blind therapy - flucloxacillin + gentamicin (benzylpenicillin + gentamicin if symptoms less severe)

initial blind therapy if prosthetic valve is present or patient is penicillin

allergic - **vancomycin + rifampicin + gentamicin**

endocarditis caused by staphylococci - flucloxacillin (vancomycin + rifampicin if penicillin allergic or MRSA)

endocarditis caused **by streptococci - benzylpenicillin + gentamicin**
(vancomycin + gentamicin if penicillin allergic)

Question 66

Each one of the following is associated with atrial myxoma, except:

- A. Clubbing
- B. Mid-diastolic murmur
- C. Pyrexia
- D. 'J' wave on ECG**
- E. Atrial fibrillation

A 'J' wave is seen in hypothermia

Atrial myxoma

Overview

75% occur in left atrium
more common in females

Features

systemic: weight loss, fever, clubbing
emboli
atrial fibrillation
mid-diastolic murmur, 'tumour plop'

Question 67

Which of the following congenital heart defects is associated with a bicuspid aortic valve

- A. Tetralogy of Fallot
- B. Ventricular septal defect
- C. Atrial septal defect
- D. Coarctation of the aorta**
- E. Transposition of the great arteries

Bicuspid aortic valve

Overview

occurs in 1-2% of the population
usually asymptomatic in childhood
the majority eventually develop aortic stenosis or regurgitation
associated with a **left dominant coronary circulation** (the posterior descending artery arises from the circumflex instead of the right coronary artery)
around **5% of patients also have coarctation of the aorta**

Complications

aortic stenosis/regurgitation as above
higher risk for aortic dissection and aneurysm formation of the ascending aorta

Question 68

Which of the following is most associated with a good prognosis in infective endocarditis?

- A. Staphylococcus aureus infection
- B. Culture negative endocarditis
- C. Streptococcus viridans infection**

- D. Low complement levels
- E. Prosthetic valve endocarditis

Infective endocarditis: prognosis and management

Poor prognostic factors

- Staph aureus infection (see below)
- prosthetic valve (especially 'early', acquired during surgery)
- culture negative endocarditis
- low complement levels

Mortality according to organism

- staphylococci - 30%
- bowel organisms - 15%
- streptococci - 5%

Current management guidelines (source: British National Formulary)

- initial blind therapy - flucloxacillin + gentamicin (benzylpenicillin + gentamicin if symptoms less severe)
- initial blind therapy if prosthetic valve is present or patient is penicillin allergic - vancomycin + rifampicin + gentamicin
- endocarditis caused by staphylococci - flucloxacillin (vancomycin + rifampicin if penicillin allergic or MRSA)
- endocarditis caused by streptococci - benzylpenicillin + gentamicin (vancomycin + gentamicin if penicillin allergic)

Question 69

Which of the following arrhythmias is most likely to produce regular cannon waves?

- A. VT with 1:1 ventricular-atrial conduction
- B. Atrial fibrillation
- C. Atrial flutter
- D. Complete heart block
- E. Ventricular fibrillation

VT with 1:1 ventricular-atrial conduction and nodal rhythms may produce regular cannon waves. Complete heart block causes intermittent cannon waves

JVP: cannon waves

Caused by the right atrium contracting against a closed tricuspid valve. May be subdivided into regular or intermittent

Regular cannon waves

- VT** (with 1:1 ventricular-atrial conduction)
- atrio-ventricular nodal re-entry tachycardia (**AVNRT**)

Intermittent cannon waves

- complete heart block

Question 70

A 71-year-old man is reviewed in the coronary care unit. He was admitted with an anterior ST-elevation myocardial infarction and received thrombolysis with alteplase. Ninety minutes following this an ECG shows a 25% resolution in the ST elevation. What is the most appropriate management?

- A. Percutaneous coronary intervention

- B. Repeat thrombolysis with tenecteplase
- C. Repeat thrombolysis with alteplase
- D. Start a nitrate infusion
- E. Inform his relatives that further intervention is futile and ensure adequate pain relief

Myocardial infarction: management

A number of studies over the past 10 years have provided an evidence for the management of ST-elevation myocardial infarction (STEMI)

In the absence of contraindications, all patients should be given

aspirin

clopidogrel: the two major studies (CLARITY and COMMIT) both confirmed benefit but used different loading doses (300mg and 75mg respectively)

low molecular weight heparin:

Primary percutaneous coronary intervention (PCI) has emerged as the gold-standard treatment for STEMI but is not available in all centres.

Thrombolysis should be performed in patients without access to primary PCI

With regards to thrombolysis:

tissue plasminogen activator (tPA) has been shown to offer clear mortality benefits over streptokinase

tenecteplase is easier to administer and has been shown to have non-inferior efficacy to alteplase with a similar adverse effect profile

An ECG should be performed 90 minutes following thrombolysis to assess whether there has been a greater than 50% resolution in the ST elevation

if there has not been adequate resolution then rescue PCI is superior to repeat thrombolysis

for patients successfully treated with thrombolysis PCI within 24 h has been shown to be beneficial

Question 71

Which one of the following is least associated with myocarditis?

- A. Chagas' disease
- B. Lyme disease
- C. Leishmaniasis
- D. Coxsackie virus
- E. Toxoplasmosis

Myocarditis

Causes

viral: coxsackie, HIV

bacteria: diphtheria, clostridia

spirochaetes: Lyme disease

protozoa: Chagas' disease, toxoplasmosis

autoimmune

drugs

Presentation

usually young patient with acute history
chest pain, SOB,

Question 72

A 71-year-old woman is admitted with acute dyspnoea to the Emergency Department. Oxygen saturations are 94% on 28% supplementary oxygen and her respiratory rate is 30/min. A rapid B-type natriuretic peptide (BNP) assay is reported as follows: BNP62 pg/ml

What is the best interpretation of this result?

- A. No conclusion can be drawn from this result
- B. Pulmonary embolism is the most likely cause of her symptoms
- C. If a further BNP level is above 50 pg/ml after one hour then this is diagnostic of heart failure
- D. Heart failure is unlikely to be the cause of her dyspnoea**
- E. Heart failure is highly likely to be the cause of her dyspnoea

B-type natriuretic peptide

B-type natriuretic peptide (BNP) hormone produced mainly by the left ventricular myocardium in response to strain

Whilst heart failure is the most obvious cause of raised BNP levels any cause of left ventricular dysfunction such as myocardial ischaemia or valvular disease may raise levels. Raised levels may also be seen due to reduced excretion in patients with chronic kidney disease. Factors which reduce BNP levels include treatment with ACE inhibitors, angiotensin-2 receptor blockers and diuretics.

Effects of BNP

vasodilator

diuretic and natriuretic

suppresses both sympathetic tone and the renin-angiotensin system

Clinical uses of BNP

Diagnosing patients with acute dyspnoea

a low concentration of BNP(< 100pg/ml) makes a diagnosis of heart failure unlikely, but raised levels should prompt further investigation to confirm the diagnosis

NICE currently recommends BNP as a helpful test to rule out a diagnosis of heart failure

Prognosis in patients with chronic heart failure

initial evidence suggests BNP is an extremely useful marker of prognosis

Guiding treatment in patients with chronic heart failure effective treatment lowers BNP levels

Screening for cardiac dysfunction

not currently recommended for population screening

Question 73

A 62-year-old man is admitted to hospital following a myocardial infarction. Four days after admission he develops a further episode of central crushing chest pain. Which is the best cardiac marker to investigate his chest pain?

- A. LDH
- B. Troponin I
- C. Troponin T
- D. CK-MB**
- E. AST

By day four the CK-MB levels should have returned to normal from the initial myocardial infarction. If the CK-MB levels are elevated it would indicate a further coronary event

Cardiac enzymes

Interpretation of the various cardiac enzymes has now largely been superseded by the introduction of troponin T and I. Questions still however commonly appear in the MRCP

Key points for the exam

myoglobin is the first to rise

CK-MB is useful to look for reinfarction as it returns to normal after 2-3 days (troponin T remains elevated for up to 10 days)

	Begins to rise	Peak value	Returns to normal
Myoglobin	1-2 hours	6-8 hours	1-2 days
CK-MB	2-6 hours	16-20 hours	2-3 days
CK	4-8 hours	16-24 hours	3-4 days
Trop T	4-6 hours	12-24 hours	7-10 days
AST	12-24 hours	36-48 hours	3-4 days
LDH	24-48 hours	72 hours	8-10 days

Question 74

A 53-year-old man is reviewed in the cardiology clinic with a history of chest pain and syncope. On examination he has an ejection systolic murmur radiating to the carotid area. What is the most likely cause of his symptoms?

- A. Bicuspid aortic valve**
- B. Aortic root abscess
- C. Post rheumatic fever
- D. Posterior myocardial infarction
- E. Calcification of the aortic valve

Aortic stenosis - most common cause:

young patients: bicuspid aortic valve

elderly patients: calcification

Aortic stenosis

Features of severe aortic stenosis

narrow pulse pressure

slow rising pulse

delayed ESM

soft/absent S2

S4

thrill

duration of murmur

left ventricular hypertrophy or failure

Causes of aortic stenosis

degenerative calcification (most common cause in elderly patients)

bicuspid aortic valve (most common cause in younger patients)

William's syndrome (supraaortic stenosis)

post-rheumatic disease

subaortic: HOCM

Management

if asymptomatic then observe the patient is general rule

if symptomatic then valve replacement

if asymptomatic but valvular gradient > 50 mmHg and with features such as left ventricular systolic dysfunction then consider surgery

balloon valvuloplasty is limited to patients with critical aortic stenosis who are not fit for valve replacement

Question 75

A 57-year-old man presents to the Emergency Department with a 15 minute history of severe central chest pain radiating to his left arm. ECG shows T-wave inversion in leads V5 and V6. Which coronary artery is likely to be affected?

A. Left circumflex

B. Posterior interventricular

C. Left main stem

D. Right coronary

E. Left anterior descending

ECG: coronary territories

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	Coronary artery
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary
Anterolateral	V4-6, I, aVL	Left main stem
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Tall R waves V1-2	Usually left circumflex, also right coronary

Question 76

A 28-year-old man with hypertrophic obstructive cardiomyopathy is investigated for palpitations. A 24 hour ECG reveals runs of non-sustained ventricular tachycardia. What is the most appropriate management?

A. AV node ablation

B. Accessory pathway ablation

C. Amiodarone

D. Implantable cardioverter defibrillator

E. Sotalol

Most cardiologists would now proceed to inserting an implantable cardioverter defibrillator to lower the risk of sudden cardiac death

HOCM: management

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins.

Management

Amiodarone

Beta-blockers or verapamil for symptoms

Cardioverter defibrillator

Dual chamber pacemaker

Endocarditis prophylaxis

Drugs to avoid

nitrates

ACE-inhibitors

inotropes

Question 77

Which one of the following conditions is most associated with aortic dissection?

- A. Acromegaly
- B. Actinomycosis
- C. Sarcoidosis
- D. Bicuspid aortic valve**
- E. Adult polycystic kidney disease

A bicuspid aortic valve increases the risk of aortic dissection six-fold

Aortic dissection

Aortic dissection may be classified as type A or B:

type A - ascending aorta, 2/3 of cases

type B - descending aorta, distal to left subclavian origin, 1/3 of cases

Associations

hypertension

trauma

bicuspid aortic valve

collagens: Marfan's syndrome, Ehlers-Danlos syndrome

Turner's and Noonan's syndrome

pregnancy

syphilis

Complications of backward tear

aortic incompetence/regurgitation

MI: inferior pattern often seen due to right coronary involvement

Complications of forward tear

unequal arm pulses and BP

stroke

renal failure

Question 78

Which one of the following features would best indicate severe aortic stenosis?

- A. Valvular gradient of 35 mmHg
- B. Quiet first heart sound
- C. Loudness of ejection systolic murmur
- D. Fourth heart sound**
- E. Development of an opening snap

Aortic stenosis

Features of severe aortic stenosis

- narrow pulse pressure
- slow rising pulse
- delayed ESM
- soft/absent S2
- S4
- thrill
- duration of murmur
- left ventricular hypertrophy or failure

Causes of aortic stenosis

- degenerative calcification (most common cause in elderly patients)
- bicuspid aortic valve (most common cause in younger patients)
- William's syndrome (supravalvular aortic stenosis)
- post-rheumatic disease
- subvalvular: HOCM

Management

- if asymptomatic then observe the patient is general rule
- if symptomatic then valve replacement
- if asymptomatic but valvular gradient > 50 mmHg and with features such as left ventricular systolic dysfunction then consider surgery
- balloon valvuloplasty is limited to patients with critical aortic stenosis who are not fit for valve replacement

Question 79

What is the most common cardiac defect seen in Marfan's syndrome

- A. Mitral valve prolapse
- B. Coarctation of the aorta
- C. Bicuspid aortic valve
- D. Dilation of the aortic sinuses**
- E. Ventricular septal defect

Whilst mitral valve prolapse is seen in Marfan's syndrome, dilation of the aortic sinuses is more common

Marfan's syndrome is an autosomal dominant connective tissue disorder. It is caused by a defect in the fibrillin-1 gene on chromosome 15

Features

- tall stature with arm span > height ratio > 1.05
- high-arched palate
- arachnodactyly
- pectus excavatum
- pes planus

scoliosis of > 20 degrees

heart: dilation of the aortic sinuses (seen in 90%) which may lead to aortic regurgitation, mitral valve prolapse (75%), aortic dissection

lungs: repeated pneumothoraces

eyes: upwards lens dislocation (superotemporal ectopia lentis), blue sclera

Question 80

How long should a patient stop driving for following an elective cardiac angioplasty?

A. No restriction

B. 1 week

C. 2 weeks

D. 4 weeks

E. 8 weeks

DVLA advice following angioplasty - cannot drive for 1 week

DVLA: cardiovascular disorders

The guidelines below relate to car/motorcycle use unless specifically stated.

For obvious reasons, the rules relating to drivers of heavy goods vehicles tend to be much stricter

Specific rules

angioplasty (elective) - 1 week off driving

CABG - 4 weeks off driving

myocardial infarction - 4 weeks off driving

angina - driving must cease if symptoms occur at rest/at the wheel

pacemaker insertion - 1 week off driving

Question 81

Which of the following is least associated with mitral valve prolapse?

A. Osteogenesis imperfecta

B. Pseudoxanthoma elasticum

C. Turner's syndrome

D. Marfan's syndrome

E. Acromegaly

Whilst some patients with acromegaly have mitral valve prolapse (MVP) it is not a common association. It should be remembered that the prevalence of MVP in a standard population is around 5-10%

Mitral valve prolapse is common, occurring in around 5-10 % of the population.

It is usually idiopathic but may be associated with a wide variety of cardiovascular disease and other conditions

Associations

congenital heart disease: PDA, ASD

cardiomyopathy

Turner's syndrome

Marfan's syndrome, Fragile X

osteogenesis imperfecta

pseudoxanthoma elasticum

Wolff-Parkinson White syndrome

long-QT syndrome

Features

patients may complain of atypical chest pain or palpitations

mid-systolic click (occurs later if patient squatting)
late systolic murmur (longer if patient standing)
complications: mitral regurgitation, arrhythmias (including long QT), emboli,
sudden death

Question 82

Which one of the following complications is least associated with ventricular septal defects?

- A. Right heart failure
- B. Aortic regurgitation
- C. Eisenmenger's complex
- D. Infective endocarditis
- E. Atrial fibrillation

Atrial fibrillation is associated more with atrial septal defects

Ventricular septal defects

Ventricular septal defects are the most common cause of congenital heart disease. They close spontaneously in around 50% of cases. Non-congenital causes include post myocardial infarction

Features

classically a pan-systolic murmur which is louder in smaller defects

Complications

aortic regurgitation*
infective endocarditis
Eisenmenger's complex
right heart failure

*aortic regurgitation is due to a poorly supported right coronary cusp resulting in cusp prolapse

Question 83

A 47-year-old man is admitted to hospital following an acute coronary syndrome. He has a history peptic ulcer disease and his cardiologist decides to use clopidogrel. What is the mechanism of action of clopidogrel?

- A. Non-selective phosphodiesterase inhibitor
- B. Phosphodiesterase V inhibitor
- C. Inhibits ATP binding to its platelet receptor
- D. Inhibits ADP binding to its platelet receptor
- E. Glycoprotein IIb/IIIa inhibitor

Clopidogrel inhibits ADP binding to platelet receptors

Clopidogrel

Mechanism

inhibits ADP binding to its platelet receptor

Question 84

A 62-year-old female with a history of mitral regurgitation attends her dentist, who intends to perform dental polishing. She is known to be penicillin allergic.

What prophylaxis against infective endocarditis should be given?

- A. Oral doxycycline
- B. Oral erythromycin
- C. No antibiotic prophylaxis needed**
- D. Oral ofloxacin
- E. Oral clindamycin

The 2008 NICE guidelines have fundamentally changed the approach to infective endocarditis prophylaxis. What is not yet clear is if there are any circumstances in which NICE would recommend using antibiotic prophylaxis

Infective endocarditis: prophylaxis

New 2008 guidelines from NICE relating have radically changed the list of procedures for which antibiotic prophylaxis is recommended

NICE recommends the following procedures **do not require prophylaxis**:

dental procedures

upper and lower gastrointestinal tract procedures

genitourinary tract; this includes urological, gynaecological and obstetric procedures and childbirth

upper and lower respiratory tract; this includes ear, nose and throat procedures and bronchoscopy

It was previously thought that the use of antibiotic prophylaxis depended on the type of cardiac lesion, as below:

Conditions potentially at risk

prosthetic valves

previous endocarditis

acquired valve disease

congenital heart lesions (see right)

mitral prolapse with regurgitation

Conditions not at risk

mitral prolapse with no regurgitation

isolated atrial septal defect

repaired ventricular septal defect

repaired patent ductus arteriosus

Question 85

A patient with known heart failure has slight limitation of physical activity. She is comfortable at rest but housework results in fatigue, palpitations or dyspnoea. What New York Heart Association class best describes the severity of their disease?

- A. NYHA Class 0
- B. NYHA Class I
- C. NYHA Class II**
- D. NYHA Class III
- E. NYHA Class IV

Heart failure: NYHA classification

The New York Heart Association (NYHA) classification is widely used to classify the severity of heart failure:

NYHA Class I

no symptoms

no limitation: ordinary physical exercise does not cause undue fatigue, dyspnoea or palpitations

NYHA Class II

mild symptoms

slight limitation of physical activity: comfortable at rest but ordinary activity results in fatigue, palpitations or dyspnoea

NYHA Class III

moderate symptoms

marked limitation of physical activity: comfortable at rest but less than ordinary activity results in symptoms

NYHA Class IV

severe symptoms

unable to carry out any physical activity without discomfort: symptoms of heart failure are present even at rest with increased discomfort with any physical activity

Question 86

In patients with atrial fibrillation (AF), which one of the following factors would make a rate control strategy, rather than rhythm control, more suitable?

- A. Congestive heart failure
- B. AF secondary to a corrected precipitant
- C. Symptomatic
- D. Age > 65 years**
- E. First presentation

Atrial fibrillation: rate control and maintenance of sinus rhythm

The Royal College of Physicians and NICE published guidelines on the management of atrial fibrillation (AF) in 2006. The following is also based on the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2002 guidelines

Agents used to maintain sinus rhythm in patients with atrial fibrillation

sotalol

amiodarone

flecainide

others (less commonly used in UK): disopyramide, dofetilide, procainamide, propafenone, quinidine

The table below indicates some of the factors which may be considered when considering either a rate control or rhythm control strategy

Factors favouring rate control

- Older than 65 years
- History of ischaemic heart disease

Factors favouring rhythm control

- Younger than 65 years
- Symptomatic
- First presentation
- Lone AF or AF secondary to a corrected precipitant (e.g. alcohol)

- Congestive heart failure

Question 87

Which of the following is least associated with primary pulmonary hypertension?

- A. HIV
- B. Fenfluramine
- C. Recurrent pulmonary embolism**
- D. Loud P2
- E. Right ventricular heave

Recurrent pulmonary embolism is a cause of secondary pulmonary hypertension

Some Candidates have questioned whether HIV, cocaine and fenfluramine should be regarded as secondary causes of pulmonary hypertension. This is a fair point and the situation should be improved with the new classification of pulmonary hypertension. However, faced with this question in the exam the correct answer would be recurrent pulmonary embolism - a classical cause of secondary pulmonary hypertension

Primary pulmonary hypertension

The classification of pulmonary hypertension is currently changing with the term idiopathic pulmonary arterial hypertension (IPAH) becoming more widely used

Primary pulmonary hypertension (PPH, now IPAH)

pulmonary arterial pressure > 25 mmHg at rest, > 30mmHg with exercise

PPH is diagnosed when no underlying cause can be found around 10% of cases are familial: autosomal dominant endothelin thought to play a key role in pathogenesis associated with HIV, cocaine and anorexigens (e.g. fenfluramine)

Features

more common in females, typically presents at 20-40 years old
 progressive SOB
 cyanosis
 right ventricular heave, loud P2, raised JVP with prominent 'a' waves,
 tricuspid regurgitation

Management

diuretics if right heart failure
 anticoagulation
 vasodilator therapy: calcium channel blocker, IV prostaglandins, bosentan:
 endothelin-1 receptor antagonist
 heart-lung transplant

Question 88

A 44-year-old female is investigated for suspected idiopathic pulmonary hypertension. Which one of the following is the best method for deciding upon management strategy?

- A. Genetic testing
- B. Acute vasodilator testing**
- C. Trial of endothelin receptor antagonists
- D. Serial echocardiography
- E. Trial of calcium channel blockers

Pulmonary arterial hypertension: features and management

Pulmonary arterial hypertension (PAH) may be defined as a sustained elevation in mean pulmonary arterial pressure of greater than 25 mmHg at rest or 30 mmHg after exercise.

Features

- exertional dyspnoea is the most frequent symptom
- chest pain and syncope may also occur
- loud P2
- left parasternal heave (due to right ventricular hypertrophy)

Management should first involve treating any underlying conditions, for example with anticoagulants or oxygen. **Following this, it has now been shown that acute vasodilator testing is central to deciding on the appropriate management strategy.** Acute vasodilator testing aims to decide which patients show a significant fall in pulmonary arterial pressure following the administration of vasodilators such as intravenous epoprostenol or inhaled nitric oxide

If there is a positive response to acute vasodilator testing
oral calcium channel blockers

If there is a negative response to acute vasodilator testing
prostacyclin analogues: treprostinil, iloprost
endothelin receptor antagonists: bosentan
phosphodiesterase inhibitors: sildenafil

Question 89

A 23-year-old man with a family history of sudden cardiac death is diagnosed as having hypertrophic obstructive cardiomyopathy. Which one of the following is the strongest marker of poor prognosis?

- A. Mitral regurgitation
- B. Apical hypertrophy
- C. Systolic anterior motion of the anterior mitral valve leaflet
- D. Septal wall thickness of > 3cm**
- E. Asymmetric hypertrophy

HOCM: prognostic factors

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins. Mutations to various proteins including beta-myosin, alpha-tropomyosin and troponin T have been identified. Septal hypertrophy causes left ventricular outflow obstruction. It is an important cause of sudden death in apparently healthy individuals

Poor prognostic factors

- syncope
- family history of sudden death
- young age at presentation
- non-sustained ventricular tachycardia on 24 or 48-hour Holter monitoring**
- abnormal blood pressure changes on exercise**
- An increased septal wall thickness is also associated with a poor prognosis**

Question 90

A 55-year-old man is admitted with central chest pain. His ECG shows ST depression in the inferior leads and the chest pain requires intravenous morphine to settle. Past medical history

includes a thrombolysed myocardial infarction 2 years ago, asthma and type 2 diabetes mellitus. Treatment with oxygen, aspirin, clopidogrel and enoxaparin is commenced. Which one of the following factors should determine if an intravenous glycoprotein IIb/IIIa receptor antagonist is given?

- A. High TIMI (thrombolysis in myocardial infarction) risk score
- B. Degree of ST depression
- C. Whether a percutaneous coronary intervention is to be performed
- D. Should be avoided in asthmatics
- E. The presence of recurrent cardiac chest pain

High-risk patients benefit from, and should be given, glycoprotein IIb/IIIa receptor antagonists whether they are due to undergo a percutaneous coronary intervention or not

Acute coronary syndrome: management

The management of non-ST elevation acute coronary syndrome is based upon the calculation of a risk score, for example TIMI (Thrombolysis In Myocardial Infarction)

All patients should receive

- oxygen
- aspirin 300mg
- nitrates or morphine to relieve chest pain if required

Clopidogrel 300mg and low molecular weight heparin (principally enoxaparin) should also be added to higher risk patients

For patients with the highest risk score an intravenous glycoprotein IIb/IIIa receptor antagonist should be used. Whilst studies have shown that patients who undergo percutaneous coronary interventions (PCI) benefit most, there is also a benefit for high risk patients who do not go on to have a PCI.

Question 91

Which of the following conditions is not associated with the development of aortic regurgitation?

- A. Rheumatic fever
- B. Ankylosing spondylitis
- C. Marfan's syndrome
- D. Syphilis
- E. Dilated cardiomyopathy

Dilated cardiomyopathy is associated with the development of mitral regurgitation, not aortic regurgitation

Aortic regurgitation

Features

early diastolic murmur

collapsing pulse

wide pulse pressure

mid-diastolic Austin-Flint murmur in severe AR - due to partial closure of the anterior mitral valve cusps caused by the regurgitation streams

Causes (due to valve disease)

rheumatic fever

infective endocarditis

connective tissue diseases e.g. RA/SLE

bicuspid aortic valve

Causes (due to aortic root disease)

aortic dissection

spondylarthropathies (e.g. ankylosing spondylitis)

hypertension

syphilis

Marfan's, Ehler-Danlos syndrome

Question 92

Which one of the following types of hyperlipidaemia are palmar crease xanthoma most commonly associated with?

A. Familial combined hyperlipidaemia

B. Lipoprotein lipase deficiency

C. Familial hypertriglyceridaemia

D. Remnant hyperlipidaemia

E. Familial hypercholesterolaemia

Palmar crease xanthoma are most strongly associated with remnant hyperlipidaemia

Hyperlipidaemia: xanthomata

Characteristic xanthomata seen in hyperlipidaemia:

Palmar xanthoma

remnant hyperlipidaemia (III)

may less commonly be seen in familial hypercholesterolaemia (IIa)

Eruptive xanthoma are due to high triglyceride levels and present as multiple red/yellow vesicles on the extensor surfaces (e.g. elbows, knees)

Causes of eruptive xanthoma

familial hypertriglyceridaemia (IV)

lipoprotein lipase deficiency (I)

familial hypertriglyceridaemia (V)

Tendon xanthoma, tuberous xanthoma, xanthelasma

familial hypercholesterolaemia (IIa)

remnant hyperlipidaemia (III)

Xanthelasma are also seen without lipid abnormalities

Question 93

Which one of the following features would indicate cardiac tamponade rather than constrictive pericarditis?

A. Pulsus paradoxus

B. Tachycardia

C. Raised JVP

D. Hypotension

E. Muffled heart sounds

Cardiac tamponade

Features

raised JVP, **with an absent Y descent** - this is due to the limited right ventricular filling
 tachycardia
 hypotension
 muffled heart sounds
pulsus paradoxus
 Kussmaul's sign (much debate about this)
 ECG: **electrical alternans**

The key differences between constrictive pericarditis and cardiac tamponade are summarised in the table below:

	Cardiac tamponade	Constrictive pericarditis
JVP	Absent Y descent	X + Y present
Pulsus paradoxus	Present	Absent
Kussmaul's sign	Rare	Present
Characteristic features		Pericardial calcification on CXR

A commonly used mnemonic to remember the absent Y descent in cardiac tamponade is TAMponade = TAMpaX

Question 94

A 43-year-old man who is known to have Wolff-Parkinson White syndrome presents to the Emergency Department with palpitations. He has no other significant history of note. The palpitations started around 4 hours ago and are not associated with chest pain or shortness of breath. On examination blood pressure is 124/80 mmHg and the chest is clear on auscultation. An ECG show atrial fibrillation at a rate of 154 bpm. Of the following options, what is the most appropriate management?

- A. Adenosine
- B. Flecainide**
- C. Verapamil
- D. Digoxin
- E. Sotalol

Adenosine should be avoided as blocking the AV node can paradoxically increase ventricular rate resulting in fall in cardiac output. **Verapamil and digoxin should also be avoided in patients with Wolff-Parkinson White as they may precipitate VT or VF.**

Another option to consider in this situation would be DC cardioversion

Wolff-Parkinson White

Wolff-Parkinson White (WPW) syndrome is caused by a congenital accessory conducting pathway between the atria and ventricles leading to a atrioventricular re-entry tachycardia (AVRT). As the accessory pathway does not slow conduction AF can degenerate rapidly to VF

Possible ECG features include:

- short PR interval
- wide QRS complexes with a slurred upstroke - 'delta wave'
- left axis deviation if right-sided accessory pathway*
- right axis deviation if left-sided accessory pathway*

Differentiating between type A and type B

type A (left-sided pathway): dominant R wave in V1

type B (right-sided pathway): no dominant R wave in V1

Associations of WPW

HOCM

mitral valve prolapse

Ebstein's anomaly

thyrotoxicosis

secundum ASD

Management

definitive treatment: radiofrequency ablation of the accessory pathway

medical therapy: sotalol**, amiodarone, flecainide

*in the majority of cases, or in a question without qualification,
Wolff-Parkinson-White syndrome is associated with left axis deviation

**sotalol should be avoided if there is coexistent atrial fibrillation as prolonging the refractory period at the AV node may increase the rate of transmission through the accessory pathway, increasing the ventricular rate and potentially deteriorating into ventricular fibrillation

Question 95

You are asked to urgently review a 61-year-old female on the cardiology ward due to difficulty in breathing. On examination she has a raised JVP with bilateral fine crackles to the mid zones. Blood pressure is 100/60 mmHg and the pulse is 140-150 and irregular. ECG confirms atrial fibrillation. What is the most appropriate management?

A. IV amiodarone

B. IV digoxin

C. Urgent synchronised DC cardioversion

D. Oral digoxin

E. IV flecainide

Heart failure is one of the adverse signs indicating the need for urgent synchronised DC cardioversion

Peri-arrest rhythms: tachycardia

The joint European Resuscitation Council and Resuscitation Council (UK) 2005 guidelines have simplified the advice given for the management of peri-arrest tachycardias. Separate algorithms for the management of broad-complex tachycardia, narrow complex tachycardia and atrial fibrillation have been replaced by one unified treatment algorithm

Following basic ABC assessment, patients are classified as being stable or unstable according to the presence of any adverse signs:

systolic BP < 90 mmHg

reduced conscious level

chest pain

heart failure

If any of the above adverse signs are present then synchronised DC shocks should be given

Treatment following this is given according to whether the QRS complex is narrow or broad and whether the rhythm is regular or irregular. The full treatment algorithm can be found at the Resuscitation Council website, below is a very limited summary:

Broad-complex tachycardia

Regular

assume ventricular tachycardia (unless previously confirmed SVT with bundle branch block)
loading dose of amiodarone followed by 24 hour infusion

Irregular

1. AF with bundle branch block - treat as for narrow complex tachycardia
2. Polymorphic VT (e.g. torsade de pointes) - IV magnesium

Narrow-complex tachycardia

Regular

vagal manoeuvres followed by IV adenosine
if above unsuccessful consider diagnosis of atrial flutter and control rate (e.g. beta-blockers)

Irregular

probable atrial fibrillation
if onset < 48 hr consider electrical or chemical cardioversion
rate control (e.g. beta-blocker or digoxin) and anticoagulation

Question 96

Which part of the jugular venous waveform may be exaggerated in tricuspid regurgitation?

- A. x descent
- B. v wave**
- C. y descent
- D. a wave
- E. c wave

JVP: giant v waves in tricuspid regurgitation

Jugular venous pulse

As well as providing information on right atrial pressure, the jugular vein waveform may provide clues to underlying valvular disease. A non-pulsatile JVP is seen in superior vena caval obstruction. Kussmaul's sign describes a paradoxical rise in JVP during inspiration seen in constrictive pericarditis

'a' wave = atrial contraction

large if atrial pressure e.g. tricuspid stenosis, pulmonary stenosis,
pulmonary hypertension
absent if in atrial fibrillation

Cannon 'a' waves

caused by atrial contractions against a closed tricuspid valve
are seen in complete heart block, ventricular tachycardia/ectopics, nodal rhythm, single chamber ventricular pacing

'c' wave

closure of tricuspid valve
not normally visible

'v' wave

giant v waves in tricuspid regurgitation

'x' descent = fall in atrial pressure during ventricular systole

'y' descent = opening of tricuspid valve

Question 97

A 72-year-old male is admitted to the Emergency Room following a collapse at church. ECG reveals dissociation between the P and QRS complexes with a rate of 40 / minute. Which one of the following clinical findings may also be found?

- A. Loud S1
- B. Narrow pulse pressure
- C. Giant v waveforms in the JVP
- D. Variable intensity of S1**
- E. Soft S2

Complete heart block

Features

syncope

heart failure

regular bradycardia (30-50 bpm)

wide pulse pressure

JVP: cannon waves in neck

variable intensity of S1

Question 98

Which one of the following is least associated with prolongation of the PR interval?

- A. Digoxin toxicity
- B. Hypocalcaemia**
- C. Lyme disease
- D. Rheumatic fever
- E. Ischaemic heart disease

Hypocalcaemia is associated with a prolonged QT interval. **Hypokalaemia is associated with a prolonged PR interval**

ECG: PR interval

Causes of a prolonged PR interval

idiopathic

ischaemic heart disease

digoxin toxicity

hypokalaemia*

rheumatic fever

aortic root pathology e.g. abscess secondary to endocarditis

Lyme disease

sarcoidosis

myotonic dystrophy

A prolonged PR interval may also be seen in athletes

*hyperkalaemia can rarely cause a prolonged PR interval, but this is a much less common association than hypokalaemia

Question 99

A 52-year-old female is referred from the Emergency Department with a pulse of 36 beats/min. The ECG shows complete heart block with a narrow QRS complex. Blood pressure is 110/70 and there is no evidence of heart failure. What is the most appropriate management?

- A. Transvenous pacing
- B. Transcutaneous pacing
- C. Isoprenaline infusion, titrated to heart rate
- D. No intervention but cardiac monitoring
- E. Intravenous atropine**

Peri-arrest rhythms: bradycardia

The joint European Resuscitation Council and Resuscitation Council (UK) 2005 guidelines emphasise that the management of bradycardia depends on:

1. identifying the presence of signs indicating haemodynamic compromise - 'adverse signs'
2. identifying the potential risk of asystole

Adverse signs

The following factors indicate haemodynamic compromise and hence the need for treatment:

heart rate < 40 bpm
systolic blood pressure < 100 mmHg
heart failure
ventricular arrhythmias requiring suppression

Atropine is the first line treatment in this situation. If this fails to work, or there is the potential risk of asystole then transvenous pacing is indicated

Potential risk of asystole

The following indicate a potential risk of asystole and hence the need for treatment with transvenous pacing:

complete heart block with broad complex QRS
recent asystole

Mobitz type II AV block
ventricular pause > 3 seconds

If there is a delay in the provision of transvenous pacing the following interventions may be used:

atropine, up to maximum of 3mg
transcutaneous pacing
adrenaline infusion titrated to response

Question 100

Each one of the following may cause secondary hypertension, except:

- A. Patent ductus arteriosus
- B. Cushing's syndrome
- C. Liddle's syndrome
- D. 11-beta hydroxylase deficiency
- E. Primary hyperparathyroidism

Hypertension: secondary causes

Renal - c. 80% of secondary hypertension

glomerulonephritis
pyelonephritis
congenital polycystic kidneys
renal artery stenosis

Endocrine disorders

Cushing's syndrome

Conn's syndrome

Liddle's syndrome

congenital adrenal hyperplasia (11-beta hydroxylase deficiency)

phaeochromocytoma

acromegaly

primary hyperparathyroidism

Others

pregnancy

coarctation of the aorta

the Pill

steroids

MAOI

alcohol

Question 101

Which one of the following is least associated with Tetralogy of Fallot?

- A. Right ventricular outflow tract obstruction
- B. Overriding aorta
- C. Ejection systolic murmur
- D. Left-to-right shunt
- E. Right ventricular hypertrophy

Right-to-left shunting is characteristic of Fallot's. It is however known that a small number of asymptomatic infants may initially have a degree of left-to-right shunting through the ventricular septal defect

Tetralogy of Fallot

Tetralogy of Fallot (TOF) is the most common cause of cyanotic congenital heart disease*. It typically presents at around 1-2 months, although may not be picked up until the baby is 6 months old

The four characteristic features are:

- ventricular septal defect (VSD)
- right ventricular hypertrophy
- right ventricular outflow tract obstruction, pulmonary stenosis
- overriding aorta

The severity of the right ventricular outflow tract obstruction determines the degree of cyanosis and clinical severity

Other features

- cyanosis
- causes a right-to-left shunt
- ejection systolic murmur due to pulmonary stenosis (the VSD doesn't usually cause a murmur)
- a right-sided aortic arch is seen in 25% of patients
- chest x-ray shows a 'boot-shaped' heart, ECG shows right ventricular hypertrophy

Management

- surgical repair is often undertaken in two parts
- cyanotic episodes may be helped by beta-blockers to reduce infundibular spasm

*however, at birth TGA is the more common lesion as patients with TOF generally present at around 1-2 months

Question 102

Which one of the following types of hyperlipidaemia are eruptive xanthoma most commonly associated with?

- A. Familial hypertriglyceridaemia
- B. Familial hypercholesterolaemia
- C. Familial combined hyperlipidaemia
- D. Remnant hyperlipidaemia
- E. Hyperlipidaemia secondary to nephrotic syndrome

Hyperlipidaemia: xanthomata

Characteristic xanthomata seen in hyperlipidaemia:

Palmar xanthoma

remnant hyperlipidaemia (III)

may less commonly be seen in familial hypercholesterolaemia (IIa)

Eruptive xanthoma are due to high triglyceride levels and present as multiple red/yellow vesicles on the extensor surfaces (e.g. elbows, knees)

Causes of eruptive xanthoma

- familial hypertriglyceridaemia (IV)
- lipoprotein lipase deficiency (I)
- familial hypertriglyceridaemia (V)

Tendon xanthoma, tuberous xanthoma, xanthelasma

familial hypercholesterolaemia (IIa)
remnant hyperlipidaemia (III)

Xanthelasma are also seen without lipid abnormalities

Question 103

A 68-year-old woman with no past medical history of note is admitted with palpitations and shortness of breath, having been unwell for the past three days. Examination reveals an irregularly irregular pulse of 130 bpm, blood pressure of 108/70 mmHg, oxygen saturations of 96% on air and bibasal lung crepitations. What is the most appropriate therapy to control her heart rate?

- A. Amiodarone
- B. Flecainide
- C. Verapamil
- D. Digoxin**
- E. Bisoprolol

Digoxin is strongly indicated for coexistent atrial fibrillation and heart failure. Beta-blockers should not be introduced until any heart failure has been stabilised. Giving amiodarone or flecainide may result in cardioversion before the patient has been adequately anticoagulated

If there was a more acute history and the patient was in significant heart failure then DC cardioversion would be appropriate, as per Advanced Life Support guidelines

Atrial fibrillation: rate control and maintenance of sinus rhythm

The Royal College of Physicians and NICE published guidelines on the management of atrial fibrillation (AF) in 2006. The following is also based on the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2002 guidelines

Agents used to maintain sinus rhythm in patients with atrial fibrillation

- sotalol
- amiodarone
- flecainide
- others (less commonly used in UK): disopyramide, dofetilide, procainamide, propafenone, quinidine

The table below indicates some of the factors which may be considered when considering either a rate control or rhythm control strategy

Factors favouring rate control

- Older than 65 years
- History of ischaemic heart disease

Factors favouring rhythm control

- Younger than 65 years
- Symptomatic
- First presentation
- Lone AF or AF secondary to a corrected precipitant (e.g. alcohol)

- Congestive heart failure

Question 104

Which one of the following is an example of a centrally acting antihypertensive?

- A. Minoxidil
- B. Hydralazine
- C. Sodium nitroprusside
- D. Moxonidine**
- E. Diazoxide

Centrally acting antihypertensives

Examples of centrally acting antihypertensives include:

methyldopa: used in the management of hypertension during pregnancy

moxonidine: used in the management of essential hypertension when conventional antihypertensives have failed to control blood pressure

clonidine: the antihypertensive effect is mediated through **stimulating alpha-2 adrenoceptors** in the vasomotor centre

Question 105

Which of the following statements concerning the third heart sound is correct?

- A. Caused by systolic filling of the ventricle
- B. May be heard in constrictive pericarditis**
- C. Associated with atrial septal defects
- D. Is characteristically soft in aortic stenosis
- E. Caused by atrial contraction against a stiff ventricle

A third heart sound is often heard in left ventricular failure and constrictive pericarditis

Heart sounds

The first heart sound (S1) is caused by closure of the mitral and tricuspid valves whilst the second heart sound (S2) is due to aortic and pulmonary valve closure

S1

closure of mitral and tricuspid valves
soft if long PR or mitral regurgitation
loud in mitral stenosis

S2

closure of aortic and pulmonary valves
soft in aortic stenosis
splitting during inspiration is normal

S3

caused by diastolic filling of the ventricle
considered normal if < 30 years old (may persist in women up to 50 years old)
heard in left ventricular failure, constrictive pericarditis

S4

may be heard in aortic stenosis, HOCM, hypertension
caused by atrial contraction against a stiff ventricle

in HOCM a double apical impulse may be felt as a result of a palpable S4

Question 106

A 14-year-old boy is admitted with palpitations and is noted to have a long QT interval. His only past medical history is deafness. What is the likely diagnosis?

- A. Leriche's syndrome
- B. Wolff-Parkinson White syndrome
- C. Jervell-Lange-Nielsen syndrome
- D. Romano-Ward syndrome
- E. Osler-Weber-Rendu syndrome

Jervell-Lange-Nielsen syndrome is associated with profound deafness and a prolonged QT interval

Long QT syndrome

Long QT syndrome is associated with delayed repolarization of the ventricles. It is important to recognise as it may lead to ventricular tachycardia and can therefore cause collapse/sudden death. A normal corrected QT is less than 440 ms in males and 450 ms in females

Congenital

Jervell-Lange-Nielsen syndrome (includes deafness and is due to an abnormal

potassium channel)

Romano-Ward syndrome (no deafness)

Drugs

amiodarone

sotalol

class 1a antiarrhythmic drugs

tricyclic antidepressants

chloroquine

terfenadine - a non-sedating antihistamine and classic cause of prolonged QT

in a patient especially if also taking P450 enzyme inhibitor, e.g. patient with cold takes terfenadine and erythromycin at same time
erythromycin

Other causes

electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia

acute MI

myocarditis

hypothermia

subarachnoid haemorrhage

Management

beta-blockers*

implantable cardioverter defibrillators in high risk cases

*note sotalol may exacerbate long QT syndrome

Question 107

A 45-year-old man presents with pleuritic central chest pain. Which one of the following ECG findings is most specific for pericarditis?

- A. PR depression
- B. T wave inversion
- C. Short PR interval
- D. U waves
- E. ST elevation

ST elevation is seen but is not specific as it may also indicate ischaemia

Pericarditis

Causes

viral infections (Coxsackie)

TB

uraemia (causes 'fibrinous' pericarditis)

trauma

post MI, Dressler's syndrome

connective tissue disease

hypothyroidism

ECG changes

widespread 'saddle-shaped' ST elevation

PR depression

Question 108

Which of the following features is not associated with patent ductus arteriosus?

- A. Continuous 'machinery' murmur
- B. Bisferiens pulse
- C. Heaving apex beat
- D. Wide pulse pressure
- E. Left subclavicular thrill

PDA is associated with a collapsing pulse

Patent ductus arteriosus

Overview

acyanotic congenital heart defect

connection between the pulmonary trunk and descending aorta

more common in premature babies, born at high altitude or maternal rubella

infection in the first trimester

Features

left subclavicular thrill

continuous 'machinery' murmur

large volume, collapsing pulse

wide pulse pressure

heaving apex beat

Management

indomethacin closes the connection in the majority of cases
if associated with another congenital heart defect amenable to surgery then
prostaglandin E1 is useful to keep the duct open until after surgical repair
passmedicine.comReference ranges End session

Question 109

Which one of the following is least associated with aortic regurgitation?

- A. Rheumatic fever
- B. William's syndrome**
- C. Syphilis
- D. Bicuspid aortic valve
- E. Post-rheumatic disease

Aortic regurgitation

Features

early diastolic murmur
collapsing pulse
wide pulse pressure
mid-diastolic Austin-Flint murmur in severe AR - due to partial closure of the
anterior mitral valve cusps caused by the regurgitation streams

Causes (due to valve disease)

rheumatic fever
infective endocarditis
connective tissue diseases e.g. RA/SLE
bicuspid aortic valve

Causes (due to aortic root disease)

aortic dissection
spondylarthropathies (e.g. ankylosing spondylitis)
hypertension
syphilis
Marfan's, Ehler-Danlos syndrome

Question 110

Which one of the following statements regarding percutaneous coronary intervention (PCI) is incorrect?

- A. Stent thrombosis usually occurs in the first month
- B. Restenosis is more common than stent thrombosis
- C. Around 95% of patients have a stent fitted during a PCI
- D. Renal impairment is a risk factor for restenosis
- E. Patients with drug-eluting stents require a shorter duration of clopidogrel therapy**

PCI - patients with drug-eluting stents require a longer duration of
clopidogrel therapy

Percutaneous coronary intervention

Percutaneous coronary intervention (PCI) is a technique used to restore myocardial perfusion in patients with ischaemic heart disease, both in patients with stable angina and acute coronary syndromes. Stents are implanted in around 95% of patients - it is now rare for just balloon angioplasty to be performed

Following stent insertion migration and proliferation of smooth muscle cells and fibroblasts occur to the treated segment. The stent struts eventually become covered by endothelium. Until this happens there is an increased risk of platelet aggregation leading to thrombosis.

Two main complications may occur

stent thrombosis: due to platelet aggregation as above. Occurs in 1-2% of patients, most commonly in the first month. Usually presents with acute myocardial infarction
restenosis: due to excessive tissue proliferation around stent. Occurs in around 5-20% of patients, most commonly in the first 3-6 months. **Usually presents with the recurrence of angina symptoms. Risk factors include diabetes, renal impairment** and stents in venous bypass grafts

Types of stent

bare-metal stent (BMS)

drug-eluting stents (DES): stent coated with paclitaxel or rapamycin which inhibit local tissue growth. Whilst this reduces restenosis rates the stent thrombosis rates are increased as the process of stent endothelialisation is slowed

Following insertion the most important factor in preventing stent thrombosis is antiplatelet therapy. Aspirin should be continued indefinitely. The length of clopidogrel treatment depends on the type of stent, reason for insertion and consultant preference

Question 111

Which of the following congenital heart defects may progress to Eisenmenger's syndrome?

- A. Tetralogy of Fallot
- B. Coarctation of the aorta
- C. Patent ductus arteriosus**
- D. Tricuspid atresia
- E. Transposition of the great arteries

Although patients with tetralogy of Fallot have, by definition, a ventricular septal defect they do not go on to develop Eisenmenger's syndrome

Eisenmenger's syndrome

Describes the reversal of a left to right shunt in a congenital heart defect due to pulmonary hypertension

Associated with

VSD
ASD
PDA

Features

original murmur may disappear
cyanosis

clubbing
right ventricular failure
haemoptysis, embolism

Management

heart-lung transplantation is required

Question 112

A 61-year-old woman who is normally fit and well is admitted with chest pain. An ECG shows anterolateral T wave inversion. The troponin T value at 12 hours is 0.54. On discharge her medications include aspirin, atorvastatin, bisoprolol and ramipril. Which one of the following statements best describes the role of clopidogrel in this situation?

- A. Is only given if aspirin is contraindicated
- B. Should be prescribed for life for patients < 65 years old
- C. Should be prescribed for the next 12 months for all patients
- D. Should be prescribed for the next 12 months for patients < 65 years old
- E. Should be prescribed for life for all patients

Myocardial infarction: secondary prevention

NICE produced guidelines on the management of patients following a myocardial infarction (MI) in 2007. Some key points are listed below

All patients should be offered the following drugs:

ACE inhibitor
beta-blocker
aspirin
statin

Clopidogrel

after an ST-segment-elevation MI, patients treated with a combination of aspirin and clopidogrel during the first 24 hours after the MI should continue this treatment for at least 4 weeks

after a non-ST segment elevation myocardial infarction clopidogrel should be given for the first 12 months

Aldosterone antagonists

patients who have had an acute MI and who have symptoms and/or signs of heart failure and left ventricular systolic dysfunction, treatment with an aldosterone antagonist licensed for post-MI treatment should be initiated within 3–14 days of the MI, preferably after ACE inhibitor therapy

External links

Question 113

Which of the following is responsible for the rapid depolarisation phase of the myocardial action potential?

- A. Rapid sodium influx
- B. Slow sodium efflux
- C. Slow efflux of calcium
- D. Efflux of potassium
- E. Rapid calcium influx

Electrical activity of the heart

Myocardial action potential

Phase	Description	Mechanism
0	Rapid depolarization	Rapid sodium influx These channels automatically deactivate after a few ms
1	Early repolarisation	Efflux of potassium
2	Plateau	Slow influx of calcium
3	Final repolarisation	Efflux of potassium
4	Restoration of ionic concentrations	Resting potential is restored by Na ⁺ /K ⁺ ATPase There is slow entry of Na ⁺ into the cell decreasing the potential difference until the threshold potential is reached, triggering a new action potential

NB cardiac muscle remains contracted 10-15 times longer than skeletal muscle

Conduction velocity

Atrial conduction Spreads along ordinary atrial myocardial fibres at 1 m/sec

AV node conduction 0.05 m/sec

Ventricular conduction Purkinje fibres are of large diameter and achieve velocities of 2-4 m/sec (this allows a rapid and coordinated contraction of the ventricles)

Question 114

Which one of the following is least associated with Wolff-Parkinson White syndrome?

- A. Mitral valve prolapse
- B. Ebstein's anomaly
- C. Thyrotoxicosis
- D. Coarctation of the aorta**
- E. Hypertrophic cardiomyopathy

Wolff-Parkinson White

Wolff-Parkinson White (WPW) syndrome is caused by a congenital accessory conducting pathway between the atria and ventricles leading to a atrioventricular re-entry tachycardia (AVRT). As the accessory pathway does not slow conduction AF can degenerate rapidly to VF

Possible ECG features include:

- short PR interval
- wide QRS complexes with a slurred upstroke - 'delta wave'
- left axis deviation if right-sided accessory pathway*
- right axis deviation if left-sided accessory pathway*

Differentiating between type A and type B

type A (left-sided pathway): dominant R wave in V1

type B (right-sided pathway): no dominant R wave in V1

Associations of WPW

HOCM

mitral valve prolapse

Ebstein's anomaly

thyrotoxicosis

secundum ASD

Management

definitive treatment: radiofrequency ablation of the accessory pathway

medical therapy: sotalol**, amiodarone, flecainide

*in the majority of cases, or in a question without qualification,

Wolff-Parkinson-White syndrome is associated with left axis deviation

**sotalol should be avoided if there is coexistent atrial fibrillation as prolonging the refractory period at the AV node may increase the rate of transmission through the accessory pathway, increasing the ventricular rate and potentially deteriorating into ventricular fibrillation

Question 115

Which one of the following cardiac conditions is most associated with a louder murmur following the Valsalva manoeuvre?

A. Mitral stenosis

B. Aortic stenosis

C. Ventricular septal defect

D. Hypertrophic obstructive cardiomyopathy

E. Aortic regurgitation

HOCM: features

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins

Features

dyspnoea, angina, syncope

sudden death (most commonly due to ventricular arrhythmias), arrhythmias, heart failure

jerky pulse, large 'a' waves, double apex beat

ejection systolic murmur: increases with Valsalva manoeuvre and decreases on squatting

Associations

Friedreich's ataxia

WPW

ECHO

systolic anterior motion (SAM) of the anterior mitral valve leaflet

asymmetric hypertrophy (ASH)

mitral regurgitation

ECG

LVH

progressive T wave inversion

deep Q waves

AF

Question 116

A 28-year-old female with a history of primary amenorrhoea and short stature is reviewed in clinic. On examination blood pressure in her right arm is 175/84 mmHg and 170/82 mmHg in her left. What is the most likely cause for her elevated blood pressure?

A. Coarctation of the aorta

- B. Conn's syndrome
- C. Essential hypertension
- D. Renal aplasia
- E. Renal artery stenosis

This patient has Turner's syndrome which is associated with coarctation of the aorta. The site of the coarctation, for example if it involves the origin of the left subclavian artery, determines whether there is a difference between the right and left arm blood pressure readings. There is no significant difference in this case.

Another cause worth considering in a young hypertensive patient with primary amenorrhoea would be congenital adrenal hyperplasia

Essential hypertension would be unusual in a 28-year-old

Turner's syndrome

Turner's syndrome is a chromosomal disorder affecting around 1 in 2,500 females. It is caused by either the presence of only one sex chromosome (X) or a deletion of the short arm of one of the X chromosomes. Turner's syndrome is denoted as 45,XO or 45,X

Features

- short stature
- shield chest, widely spaced nipples
- webbed neck
- coarctation of the aorta
- primary amenorrhoea
- high-arched palate
- short fourth metacarpal
- multiple pigmented naevi
- lymphoedema in neonates (especially feet)

There is also an increased incidence of autoimmune disease (especially autoimmune thyroiditis) and Crohn's disease

Question 117

Each one of the following is an indication for an implantable cardiac defibrillator, except:

- A. Previous myocardial infarction with non-sustained VT on 24 hr monitoring
- B. Wolff-Parkinson White syndrome**
- C. Hypertrophic obstructive cardiomyopathy
- D. Previous cardiac arrest due to VF
- E. Long QT syndrome

Implantable cardiac defibrillators

Indications

- long QT syndrome
- hypertrophic obstructive cardiomyopathy
- previous cardiac arrest due to VT/VF
- previous myocardial infarction with non-sustained VT on 24 hr monitoring,
- inducible VT on electrophysiology testing and ejection fraction < 35%

Question 118

What is the normal cross sectional area of the mitral valve?

- A. 1-2 sq cm
- B. 3-4 sq cm
- C. 4-6 sq cm
- D. 6-8 sq cm
- E. 8-10 sq cm

Mitral stenosis

It is said that the causes of mitral stenosis are rheumatic fever, rheumatic fever and rheumatic fever. Rarer causes that may be seen in the MRCP include mucopolysaccharidoses, carcinoid and endocardial fibroelastosis

Features

mid-diastolic murmur (best heard in expiration)
loud S1, opening snap
low volume pulse
malar flush
atrial fibrillation

Features of severe MS

length of murmur increases
opening snap becomes closer to S2

Echocardiography

the normal cross sectional area of the mitral valve is 4-6 sq cm. **A 'tight' mitral stenosis implies a cross sectional area of < 1 sq cm**

Question 119

A 57-year-old man with NYHA class III heart failure is currently treated with frusemide and ramipril. What is the most suitable beta-blocker to add to improve his long-term prognosis?

- A. Acebutolol
- B. Labetalol
- C. Bisoprolol
- D. Sotalol
- E. Esmolol

Both carvedilol and bisoprolol have been shown to reduce mortality in stable heart failure. The other beta-blockers have no evidence base to support their use

Heart failure: drug management

A number of drugs have been shown to improve mortality in patients with chronic heart failure:

ACE inhibitors (SAVE, SOLVD, CONSENSUS)
spironolactone (RALES)
beta-blockers (CIBIS)
hydralazine with nitrates (VHEFT-1)

Whilst spironolactone has been shown to improve prognosis in patients with chronic heart failure, no long-term reduction in mortality has been demonstrated for loop diuretics such as frusemide

NICE produced guidelines on management in 2003, key points include:

all patients should be given an ACE inhibitor unless contradictions exist once an ACE inhibitor has been introduced a beta-blocker should be started regardless of whether the patient is still symptomatic offer annual influenza vaccine offer pneumococcal vaccine. Digoxin has also not been proven to reduce mortality in patients with heart failure. It may however improve symptoms due to its inotropic properties. **Digoxin is strongly indicated if there is coexistent atrial fibrillation**

Question 120

A 62-year-old man is seen following a successful elective DC cardioversion for atrial fibrillation. Four weeks ago he presented in fast atrial fibrillation. A decision was made at the time to warfarinise him for four weeks after which he was to be cardioverted. During this time he had a normal transthoracic echocardiogram. He has no past medical history of note other than treatment for a basal cell carcinoma. What is the most appropriate plan regarding anticoagulation?

- A. Can stop immediately
- B. Continue warfarinisation for 1 week then review following
- C. Lifelong warfarin
- D. Lifelong aspirin
- E. Continue warfarinisation for 4 weeks then review following**

Atrial fibrillation: cardioversion

Onset < 48 hours

If atrial fibrillation (AF) is of less than 48 hours onset patients should be heparinised and a transthoracic echocardiogram and can be cardioverted immediately, either:

electrical - 'DC cardioversion'

pharmacology - amiodarone if structural heart disease, flecainide in those without structural heart disease

Following electrical cardioversion if AF is confirmed as being less than 48 hours duration then further anticoagulation is unnecessary

Onset > 48 hours

If AF is of greater than 48 hours then patients should have therapeutic anticoagulation for at least 3 weeks. If there is a high risk of cardioversion failure (e.g. previous failure or AF recurrence) then it is recommend to have at least 4 weeks amiodarone or sotalol prior to electrical cardioversion

Following electrical cardioversion patients should be anticoagulated for at least 4 weeks.

After this time decisions about anticoagulation should be taken on an individual basis depending on the risk of recurrence

Question 121

A 37-year-old woman presents for review. She is 26 weeks pregnant and has had no problems with her pregnancy to date. Blood pressure is 144/92 mmHg, a rise from her booking reading of 110/80 mmHg. Urine dipstick reveals the following:

Protein	negative
Leucocytes	negative
Blood	negative

What is the most appropriate description of her condition?

- A. Moderate pre-eclampsia
- B. Mild pre-eclampsia
- C. Gestational hypertension
- D. Normal physiological change in blood pressure
- E. Pre-existing hypertension

Hypertension in pregnancy

The classification of hypertension in pregnancy is complicated and varies.

Remember, in normal pregnancy:

blood pressure usually falls in the first trimester (particularly the diastolic), and continues to fall until 20-24 weeks after this time the blood pressure usually increases to pre-pregnancy levels by term

Hypertension in pregnancy is usually defined as:

systolic > 140 mmHg or diastolic > 90 mmHg or an increase above booking readings of > 30 mmHg systolic or > 15 mmHg diastolic

After establishing that the patient is hypertensive they should be categorised into one of the following groups

Pre-existing hypertension

A history of hypertension before 20 weeks gestation

or an elevated blood pressure $> 140/90$ mmHg on booking readings; No proteinuria, no oedema Occurs in 3-5% of pregnancies and is more common in older women.

Pregnancy-induced hypertension(PIH, also known as gestational hypertension) Hypertension (as defined above) occurring in the second half of pregnancy (i.e. after 20 weeks) No proteinuria, no oedema Occurs in around 5-7% of pregnancies Resolves following birth (typically after one month).

Pre-eclampsia

Pregnancy-induced hypertension in association with proteinuria (> 0.3 g / 24 hours)

Oedema may occur but is now less commonly used as a criteria

Occurs in around 5% of pregnancies

Question 122

Which part of the jugular venous waveform is associated with the fall in atrial pressure during ventricular systole?

- A. y descent
- B. v wave
- C. x descent
- D. c wave
- E. a wave

JVP: x descent = fall in atrial pressure during ventricular systole

Jugular venous pulse

As well as providing information on right atrial pressure, the jugular vein waveform may provide clues to underlying valvular disease. A non-pulsatile JVP is seen in superior vena

caval obstruction. Kussmaul's sign describes a paradoxical rise in JVP during inspiration seen in constrictive pericarditis

'a' wave = atrial contraction

large if atrial pressure e.g. tricuspid stenosis, pulmonary stenosis,
pulmonary hypertension
absent if in atrial fibrillation

Cannon 'a' waves

caused by atrial contractions against a closed tricuspid valve
are seen in complete heart block, ventricular tachycardia/ectopics, nodal
rhythm, single chamber ventricular pacing

'c' wave

closure of tricuspid valve
not normally visible

'v' wave

giant v waves in tricuspid regurgitation

'x' descent = fall in atrial pressure during ventricular systole

'y' descent = opening of tricuspid valve

Question 123

A 62-year-old patient presents to the Emergency Department with a 25 minute history of crushing central chest pain. ECG shows ST elevation in leads I and aVL. Which coronary territory is likely to be affected?

A. Lateral

B. Posterior

C. Anteroseptal

D. Anterolateral

E. Inferior

ECG: coronary territories

The table below shows the correlation between ECG changes and coronary territories:

	ECG changes	Coronary artery
Anteroseptal	V1-V4	Left anterior descending
Inferior	II, III, aVF	Right coronary
Anterolateral	V4-6, I, aVL	Left main stem
Lateral	I, aVL +/- V5-6	Left circumflex
Posterior	Tall R waves V1-2	Usually left circumflex, also right coronary

Question 124

A 74-year-old man is admitted with chest pain associated with ECG changes. A troponin T taken 12 hours after admission indicates an acute myocardial infarction. Which one of the following is most likely to predict a poor prognosis?

A. History of diabetes mellitus

B. Loss of heart rate variability

C. Left ventricular ejection fraction of 40%

- D. Diastolic blood pressure of 110 mmHg
- E. Male sex

Acute coronary syndrome: prognostic factors
 The 2006 Global Registry of Acute Coronary Events (GRACE) study has been used to derive regression models to predict death in hospital and death after discharge in patients with acute coronary syndrome

Poor prognostic factors

age

development (or history) of heart failure

peripheral vascular disease

systolic blood pressure

Killip class*

initial serum creatinine concentration

elevated initial cardiac markers

cardiac arrest on admission

ST segment deviation

***Killip class** - system used to stratify risk post myocardial infarction

Killip class	Features	30 day mortality
I	No clinical signs heart failure	6%
II	Lung crackles, S3	17%
III	Frank pulmonary oedema	38%
IV	Cardiogenic shock	81%

Question 125

Eight months after having a prosthetic heart valve a patient develops infective endocarditis. What is the most likely causative organism?

A. Streptococcus viridans

B. Staphylococcus aureus

C. Staphylococcus epidermidis

D. Coxiella burnetii

E. One of the HACEK group

Most common cause of endocarditis:

Streptococcus viridans

Staphylococcus epidermidis if < 2 months post valve surgery

Staphylococcus epidermidis is the most common causative organism in the first two months following surgery. After this time the spectrum of organisms causing endocarditis returns to normal, with Streptococcus viridans being the most common organism

Infective endocarditis

Patients affected by endocarditis

previously normal valves (50%, typically acute presentation)

rheumatic valve disease (30%)

prosthetic valves

congenital heart defects
intravenous drug users (IVDUs, e.g. typically causing tricuspid lesion)

Causes

Streptococcus viridans (most common cause - 40-50%)
Staphylococcus epidermidis (especially prosthetic valves)
Staphylococcus aureus (especially acute presentation, IVDUs)
Streptococcus bovis is associated with colorectal cancer
non-infective: systemic lupus erythematosus (Libman-Sacks), malignancy:
marantic endocarditis

Culture negative causes

prior antibiotic therapy
Coxiella burnetii
Bartonella
Brucella
HACEK: Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella)

Following prosthetic valve surgery Staphylococcus epidermidis is the most common organism in the first 2 months and is usually the result of perioperative contamination. After 2 months the spectrum of organisms which cause endocarditis return to normal, except with a slight increase in Staph aureus infections

Question 126

A 64-year-old female presents with central chest pain radiating down her left arm of 20 minutes duration. On examination the pulse is 90 bpm and regular and the BP is 205/110 mmHg. ECG shows 2 mm ST elevation in leads V2-6. Morphine and aspirin have already been given. What is the most appropriate next step?

- A. Observe
- B. IV streptokinase
- C. IV alteplase
- D. IV GTN**
- E. Temporary pacing

The elevated blood pressure would be a contraindication to giving thrombolysis in this patient

Contraindications to thrombolysis

active internal bleeding
recent haemorrhage, trauma or surgery (including dental extraction)
coagulation and bleeding disorders
intracranial neoplasm
stroke < 2 months
aortic dissection
recent head injury
pregnancy
severe hypertension

Question 127

Each one of the following is associated with left axis deviation on ECG, except:

- A. Left anterior hemiblock
- B. Ostium primum ASD
- C. Left posterior hemiblock**
- D. Obesity

E. Left bundle branch block

ECG: axis deviation

Causes of left axis deviation (LAD)

left anterior hemiblock

left bundle branch block

Wolff-Parkinson-White syndrome* - right-sided accessory pathway

hyperkalaemia

congenital: ostium primum ASD, tricuspid atresia

minor LAD in obese people

Causes of right axis deviation (RAD)

right ventricular hypertrophy

left posterior hemiblock

chronic lung disease

pulmonary embolism

ostium secundum ASD

Wolff-Parkinson-White syndrome* - left-sided accessory pathway

normal in infant < 1 years old

minor RAD in tall people

*in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation

Question 128

Which of the following conditions is least associated with coarctation of the aorta?

A. Neurofibromatosis

B. Bicuspid aortic valve

C. Prader-Willi syndrome

D. Turner's syndrome

E. Berry aneurysms

Coarctation of the aorta

Coarctation of the aorta describes a congenital narrowing of the descending aorta

Overview

more common in males (despite association with Turner's syndrome)

Features

infancy: heart failure

adult: hypertension

radio-femoral delay

mid systolic murmur, maximal over back

apical click from the aortic valve

notching of the inferior border of the ribs (due to collateral vessels) is not seen in young children

Associations

Turner's syndrome

bicuspid aortic valve

berry aneurysms

neurofibromatosis

Question 129

A 68-year-old man with a past history of aortic stenosis is reviewed in clinic. Which one of the following features would most guide the timing of surgery?

- A. Symptomatology of patient
- B. Aortic valve gradient of 50 mmHg
- C. Pulse pressure
- D. Loudness of murmur
- E. Left ventricular ejection fraction

Aortic stenosis management: AVR if symptomatic, otherwise cut-off is gradient of 50 mmHg

Aortic stenosis

Features of severe aortic stenosis

narrow pulse pressure
slow rising pulse
delayed ESM
soft/absent S2
S4
thrill
duration of murmur
left ventricular hypertrophy or failure

Causes of aortic stenosis

degenerative calcification (most common cause in elderly patients)
bicuspid aortic valve (most common cause in younger patients)
William's syndrome (supravalvular aortic stenosis)
post-rheumatic disease
subvalvular: HOCM

Management

if asymptomatic then observe the patient is general rule
if symptomatic then valve replacement
if asymptomatic but valvular gradient > 50 mmHg and with features such as left ventricular systolic dysfunction then consider surgery
balloon valvuloplasty is limited to patients with critical aortic stenosis who are not fit for valve replacement

Question 130

Each one of the following may cause left bundle branch block, except:

- A. Cardiomyopathy
- B. Atrial septal defect
- C. Hypertension
- D. Idiopathic fibrosis
- E. Ischaemic heart disease

Atrial septal defects, both primum and secundum, are associated with right rather than left bundle branch block

ECG: LBBB

Causes of LBBB

ischaemic heart disease

hypertension

cardiomyopathy

idiopathic fibrosis

Question 131

Which part of the ECG complex corresponds with the closure of the mitral valve?

A. P wave

B. PR interval

C. QRS complex

D. ST segment

E. T wave

A diagram of the cardiac cycle can be found on the external link

Heart sounds

The first heart sound (S1) is caused by closure of the mitral and tricuspid valves whilst the second heart sound (S2) is due to aortic and pulmonary valve closure

S1

closure of mitral and tricuspid valves

soft if long PR or mitral regurgitation

loud in mitral stenosis

S2

closure of aortic and pulmonary valves

soft in aortic stenosis

splitting during inspiration is normal

S3

caused by diastolic filling of the ventricle

considered normal if < 30 years old (may persist in women up to 50 years old)

heard in left ventricular failure, constrictive pericarditis

S4

may be heard in aortic stenosis, HOCM, hypertension

caused by atrial contraction against a stiff ventricle

in HOCM a double apical impulse may be felt as a result of a palpable S4

Question 132

Which one of the following clinical signs would best indicate severe calcified aortic stenosis?

A. Loudness of murmur

B. Loud second heart sound

C. Radiation to the carotids

D. Hypertension

E. Displaced apex beat

The apex beat is not normally displaced in aortic stenosis. Displacement would indicate left ventricular dilatation and hence severe disease

Features of severe aortic stenosis

- narrow pulse pressure
- slow rising pulse
- delayed ESM
- soft/absent S2
- S4
- thrill
- duration of murmur

left ventricular hypertrophy or failure

Causes of aortic stenosis

- degenerative calcification (most common cause in elderly patients)
- bicuspid aortic valve (most common cause in younger patients)
- William's syndrome (supravalvular aortic stenosis)
- post-rheumatic disease
- subvalvular: HOCM

Management

- if asymptomatic then observe the patient is general rule
- if symptomatic then valve replacement
- if asymptomatic but valvular gradient > 50 mmHg and with features such as left ventricular systolic dysfunction then consider surgery
- balloon valvuloplasty is limited to patients with critical aortic stenosis who are not fit for valve replacement

Question 133

Which one of the following patients should not automatically be prescribed aspirin 75 mg od in the absence of any contraindication?

- A. 62-year-old man who had a myocardial infarction 11 years ago
- B. 72-year-old man with intermittent claudication
- C. 44-year-old man with hypertension and a 10-year cardiovascular risk of 14%**
- D. 54-year-old man with well controlled type 2 diabetes mellitus with no evidence of end-organ damage
- E. 64-year-old smoker with a 10-year cardiovascular risk of 24%

Aspirin

Aspirin works by blocking the action of both cyclooxygenase-1 and 2.

Who should receive aspirin?

- all people with established cardiovascular disease** (stroke, TIA, ischaemic heart disease, peripheral arterial disease)
- all people aged 50 years and over with a 10-year cardiovascular risk $\geq 20\%$**
- all people with diabetes mellitus (type 1 or 2) who are ≥ 50 years old or who have: diabetes > 10 years, taking treatment for hypertension or evidence of target organ damage**
- all people with target organ damage from hypertension**

Potentiates

- oral hypoglycaemics
- warfarin
- steroids

Question 134

A 29-year-old man with myotonic dystrophy has an electrocardiogram. Which one of the following findings is most likely to be present?

- A. Wide QRS complex
- B. Atrial fibrillation
- C. Voltage criteria for left ventricular hypertrophy
- D. Right axis deviation
- E. Prolonged PR interval

A prolonged PR interval is seen in around 20-40% of patients

Myotonic dystrophy

Myotonic dystrophy (also called dystrophia myotonica) is an inherited myopathy with features developing at around 20-30 years old. It affects skeletal, cardiac and smooth muscle. There are two main types of myotonic dystrophy, DM1 and DM2.

Genetics

autosomal dominant

a trinucleotide repeat disorder

DM1 is caused by a CTG repeat at the end of the DMPK (Dystrophia Myotonica-Protein Kinase) gene on chromosome 19

DM2 is caused by a repeat expansion of the ZNF9 gene on chromosome 3

The key differences are listed in table below:

DM1	DM2
- DMPK gene on chromosome 19	ZNF9 gene on chromosome 3
- Distal weakness more prominent	Proximal weakness more prominent
	Severe congenital form not seen

General features

myotonic facies (long, 'haggard' appearance)
frontal balding
bilateral ptosis
cataracts
dysarthria

Other features

myotonia (tonic spasm of muscle)
weakness of arms and legs (distal initially)
mild mental impairment
diabetes mellitus
testicular atrophy
cardiac involvement: heart block, cardiomyopathy
dysphagia

Question 135

Where is the most common site for primary cardiac tumours to occur in adults?

- A. Left atrium
- B. Right ventricle
- C. Right atrium
- D. Left atrial appendage
- E. Left ventricle

The most common site of atrial myxomas is at the fossa ovalis border in the left atrium

Atrial myxoma

Overview

75% occur in left atrium
more common in females

Features

systemic: weight loss, fever, clubbing
emboli
atrial fibrillation
mid-diastolic murmur, 'tumour plop'

Question 136

A 45-year-old man presents with fever. On examination he is noted to have a pan-systolic murmur and splinter haemorrhages. He is generally unwell with a blood pressure of 100/60 mmHg and a temperature of 38.8°C. What is the most suitable antibiotic therapy until blood culture results are known?

- A. IV flucloxacillin + gentamicin
- B. IV benzylpenicillin + gentamicin
- C. IV vancomycin + gentamicin
- D. IV vancomycin + benzylpenicillin
- E. IV ceftriaxone + benzylpenicillin

Infective endocarditis: prognosis and management

Poor prognostic factors

Staph aureus infection (see below)
prosthetic valve (especially 'early', acquired during surgery)
culture negative endocarditis
low complement levels

Mortality according to organism

staphylococci - 30%
bowel organisms - 15%
streptococci - 5%

Current management guidelines (source: British National Formulary)

initial blind therapy - flucloxacillin + gentamicin (benzylpenicillin + gentamicin if symptoms less severe)

initial blind therapy if prosthetic valve is present or patient is penicillin allergic - **vancomycin + rifampicin + gentamicin**

endocarditis caused by **staphylococci - flucloxacillin** (vancomycin + rifampicin if penicillin allergic or MRSA)

endocarditis caused by **streptococci - benzylpenicillin + gentamicin** (vancomycin + gentamicin if penicillin allergic)

Question 137

Which part of the jugular venous waveform is associated with the opening of the tricuspid valve?

- A. x descent
- B. v wave

- C. a wave
- D. c wave
- E. y descent

JVP: y descent = opening of tricuspid valve

Jugular venous pulse

As well as providing information on right atrial pressure, the jugular vein waveform may provide clues to underlying valvular disease. A non-pulsatile JVP is seen in superior vena caval obstruction. Kussmaul's sign describes a paradoxical rise in JVP during inspiration seen in constrictive pericarditis

'a' wave = atrial contraction

large if atrial pressure e.g. tricuspid stenosis, pulmonary stenosis,
pulmonary hypertension
absent if in atrial fibrillation

Cannon 'a' waves

caused by atrial contractions against a closed tricuspid valve
are seen in complete heart block, ventricular tachycardia/ectopics, nodal
rhythm, single chamber ventricular pacing

'c' wave

closure of tricuspid valve
not normally visible

'v' wave

giant v waves in tricuspid regurgitation

'x' descent = fall in atrial pressure during ventricular systole

'y' descent = opening of tricuspid valve

Question 138

A 65-year-old man is discharged from hospital following a thrombolysed ST-elevation myocardial infarction. Other than a history of depression he has no past medical history of note. His stay on the coronary care unit was complicated by the development of dyspnoea and an echo show a reduced left ventricular ejection fraction. The patient was not given clopidogrel during his hospital admission. Other than standard treatment with an ACE inhibitor, beta-blocker, aspirin and statin, what other type of drug should he be taking?

- A. Angiotensin 2 receptor antagonist
- B. Potassium channel activator
- C. Aldosterone antagonist
- D. Thiazide diuretic
- E. Clopidogrel

An aldosterone antagonist is recommended by current NICE guidelines as the patient has a reduced left ventricular ejection fraction. If clopidogrel was given during the first 24 hours then it should be continued for the next 4 weeks

Myocardial infarction: secondary prevention

NICE produced guidelines on the management of patients following a myocardial infarction (MI) in 2007. Some key points are listed below

All patients should be offered the following drugs:

ACE inhibitor
beta-blocker
aspirin
statin

Clopidogrel

after an ST-segment-elevation MI, patients treated with a combination of aspirin and clopidogrel during the first 24 hours after the MI should continue this treatment for at least 4 weeks

after a non-ST segment elevation myocardial infarction clopidogrel should be given for the first 12 months

Aldosterone antagonists

patients who have had an acute MI and who have symptoms and/or signs of heart failure and left ventricular systolic dysfunction, treatment with an aldosterone antagonist licensed for post-MI treatment should be initiated within 3–14 days of the MI, preferably after ACE inhibitor therapy

Question 139

A 24-year-old female develops transient slurred speech following a flight from Australia to the United Kingdom. Both a CT head and ECG are normal. Which one of the following tests is most likely to reveal the underlying cause?

- A. Transoesophageal echo
- B. MRI brain
- C. Carotid USS Doppler
- D. Cerebral angiogram
- E. Transthoracic echo

Paradoxical embolus - PFO most common cause - do TOE

Transesophageal echocardiography provides superior views of the atrial septum and therefore is preferred to transthoracic echocardiography for detecting patent foramen ovale

Paradoxical embolisation

For a right-sided thrombus (e.g. DVT) to cause a left-sided embolism (e.g. stroke) it must obviously pass from the right-to-left side of the heart

The following cardiac lesions may cause such events

- patent foramen ovale - present in around 20% of the population
- atrial septal defect - a much less common cause

Question 140

The most common cause of restrictive cardiomyopathy in the UK is:

- A. Diabetes mellitus
- B. Systemic lupus erythematosus
- C. Haemochromatosis
- D. Tuberculosis
- E. Amyloidosis

Restrictive cardiomyopathy: amyloid (most common), haemochromatosis, Löffler's syndrome, sarcoidosis, scleroderma

Restrictive cardiomyopathy

Features

similar to constrictive pericarditis

Features suggesting restrictive cardiomyopathy rather than constrictive pericarditis

prominent apical pulse

absence of pericardial calcification on CXR

heart may be enlarged

ECG abnormalities e.g. bundle branch block, Q waves

Causes

amyloidosis (e.g. secondary to myeloma) - most common cause in UK

haemochromatosis

Löffler's syndrome

sarcoidosis

scleroderma

Question 141

A 65-year-old female is admitted with a suspected infective exacerbation of chronic obstructive pulmonary disease. On examination she is dyspnoeic with a blood pressure of 112/68 mmHg. Electrocardiogram shows an irregular, narrow-complex tachycardia with a rate of 130 bpm. At least three different P wave morphologies are seen. A diagnosis of multifocal tachycardia is suspected. What is the most appropriate management?

A. Adenosine

B. Digoxin

C. Verapamil

D. Atenolol

E. DC cardioversion

Multifocal atrial tachycardia

Multifocal atrial tachycardia (MAT) may be defined as a irregular cardiac rhythm caused by at least three different sites in the atria, which may be demonstrated by morphologically distinctive P waves. It is more common in elderly patients with chronic lung disease, for example COPD

Management

correction of hypoxia and electrolyte disturbances

rate-limiting calcium channel blockers are often used first-line

cardioversion and digoxin are not useful in the management of MAT

Question 142

A 42-year-old man of Afro-Caribbean origin is diagnosed as having hypertension. Secondary causes of hypertension have been excluded. What is the most appropriate initial drug therapy?

A. Losartan

B. Bisoprolol

C. Doxazosin

D. Perindopril

E. Amlodipine

ACE inhibitors have reduced efficacy in black patients and are therefore not used first-line

Hypertension: management

NICE published updated guidelines for the management of hypertension in June 2006

Initial drug choice

patients < 55-years-old: ACE inhibitor

patients > 55-years-old or of Afro-Caribbean origin: calcium channel blocker or thiazide diuretic

If this fails to control the blood pressure then use a combination of an ACE inhibitor plus either a calcium channel blocker or thiazide diuretic

If this still fails then a combination of an ACE inhibitor + calcium channel blocker + thiazide diuretic should be used

Question 143

A 52-year-old man is admitted to the Emergency Department. He was found collapsed by neighbours. An ECG on arrival shows torsades de pointes. Which one of his medications is most likely to have contributed to this presentation?

- A. Bisoprolol
- B. Cimetidine
- C. Risperidone**
- D. Phenytoin
- E. Doxycycline

Torsades de pointes

Torsades de pointes ('twisting of the points') is a rare arrhythmia associated with a long QT interval. It may deteriorate into ventricular fibrillation and hence lead to sudden death

Causes of long QT interval

congenital: Jervell-Lange-Nielsen syndrome, Romano-Ward syndrome

antiarrhythmics: amiodarone, sotalol, class 1a antiarrhythmic drugs

tricyclic antidepressants

antipsychotics

chloroquine

terfenadine

erythromycin

electrolyte: hypocalcaemia, hypokalaemia, hypomagnesaemia

myocarditis

hypothermia

subarachnoid haemorrhage

Management

IV magnesium sulphate

Question 144

Which one of the following drugs is best avoided in patients with hypertrophic obstructive cardiomyopathy?

- A. Amiodarone
- B. Verapamil
- C. Ramipril**
- D. Amoxicillin
- E. Atenolol

HOCM - drugs to avoid: nitrates, ACE-inhibitors, inotropes

Verapamil should however be avoided in patients with coexistent Wolff-Parkinson White as it may precipitate VT or VF

HOCM: management

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins.

Management

- Amiodarone
- Beta-blockers or verapamil for symptoms
- Cardioverter defibrillator
- Dual chamber pacemaker
- Endocarditis prophylaxis

Drugs to avoid

- nitrates
- ACE-inhibitors
- inotropes

Question 145

A 56-year-old man with a past history of ischaemic heart disease is admitted with central chest pain radiating to his left arm associated with nausea. On arrival in the Coronary Care Unit he is noted to be in complete heart block. Which coronary artery is likely to be affected?

- A. Circumflex
- B. Right coronary**
- C. Obtuse marginal
- D. Left anterior descending
- E. Posterior descending

The right coronary artery supplies the atrioventricular node in 90% of patients

Coronary circulation

Arterial supply of the heart

- posterior aortic sinus --> left coronary artery (LCA)
- anterior aortic sinus --> right coronary artery (RCA)
- LCA --> LAD + circumflex
- RCA --> posterior descending

RCA supplies SA node in 60%, AV node in 90%

Venous drainage of the heart

- coronary sinus drains into the right atrium

Question 146

Which of the following is a cause of a loud second heart sound?

- A. Aortic regurgitation
- B. Ventricular septal defect
- C. Systemic hypertension**
- D. Aortic stenosis

E. Mitral stenosis

Second heart sound (S2)

loud: hypertension

soft: AS

fixed split: ASD

reversed split: LBBB

Heart sounds: S2

S2 is caused by the closure of the aortic valve (A2) closely followed by that of the pulmonary valve (P2)

Causes of a loud S2

hypertension: systemic (loud A2) or pulmonary (loud P2)

hyperdynamic states

atrial septal defect without pulmonary hypertension

Causes of a soft S2

aortic stenosis

Causes of fixed split S2

atrial septal defect

Causes of a widely split S2

deep inspiration

RBBB

pulmonary stenosis

severe mitral regurgitation

Causes of a reversed (paradoxical) split S2 (P2 occurs before A2)

LBBB

severe aortic stenosis

right ventricular pacing

WPW type B (causes early P2)

patent ductus arteriosus

Question 147

Which one of the following features is not part of the modified Duke criteria used in the diagnosis of infective endocarditis?

A. Prolonged PR interval

B. Positive serology for *Coxiella burnetii*

C. Fever $> 38^{\circ}\text{C}$

D. Roth spots

E. Positive microbiology from embolic fragments

A prolonged PR interval is part of the diagnostic criteria of rheumatic fever. The modified Duke criteria have now been adopted in the latest guidelines from the European Society of Cardiology. Details can be found in the link below

Infective endocarditis: Modified Duke criteria

Infective endocarditis diagnosed if:-

- pathological criteria positive, or
- 2 major criteria, or

- 1 major and 3 minor criteria, or
- 5 minor criteria

Pathological criteria

Positive histology or microbiology of pathological material obtained at autopsy or cardiac surgery (valve tissue, vegetations, embolic fragments or intracardiac abscess content)

Major criteria

Positive blood cultures

two positive blood cultures showing typical organisms consistent with infective endocarditis, such as *Streptococcus viridans* and the HACEK group, or persistent bacteraemia from two blood cultures taken > 12 hours apart or three or more positive blood cultures where the pathogen is less specific such as *Staph aureus* and *Staph epidermidis*, or positive serology for *Coxiella burnetii*, *Bartonella* species or *Chlamydia psittaci*, or positive molecular assays for specific gene targets

Evidence of endocardial involvement

positive echocardiogram (oscillating structures, abscess formation, new valvular regurgitation or dehiscence of prosthetic valves), or new valvular regurgitation

Minor criteria

predisposing heart disease

microbiological evidence does not meet major criteria

fever > 38°C

vascular phenomena: major emboli, splenomegaly, clubbing, splinter

haemorrhages, petechiae or purpura

immunological phenomena: glomerulonephritis, Osler's nodes, Roth spots

elevated CRP or ESR

Question 148

A 49-year-old man with idiopathic pulmonary arterial hypertension has a negative acute vasodilator test. Which one of the following medications is least likely to be beneficial in his long-term management?

A. Nifedipine

B. Treprostinil

C. Bosentan

D. Sildenafil

E. Warfarin

Oral calcium channel blockers are unlikely to be beneficial following a negative acute vasodilator test

Pulmonary arterial hypertension: features and management

Pulmonary arterial hypertension (PAH) may be defined as a sustained elevation in mean pulmonary arterial pressure of greater than 25 mmHg at rest or 30 mmHg after exercise.

Features

exertional dyspnoea is the most frequent symptom

chest pain and syncope may also occur

loud P2

left parasternal heave (due to right ventricular hypertrophy)

Management should first involve treating any underlying conditions, for example with anticoagulants or oxygen. Following this, it has now been shown that acute vasodilator testing is central to deciding on the appropriate management strategy. Acute vasodilator testing aims to decide which patients show a significant fall in pulmonary arterial pressure following the administration of vasodilators such as intravenous epoprostenol or inhaled nitric oxide

If there is a positive response to acute vasodilator testing

oral calcium channel blockers

If there is a negative response to acute vasodilator testing

prostacyclin analogues: treprostinil, iloprost

endothelin receptor antagonists: bosentan

phosphodiesterase inhibitors: sildenafil

Question 149

Each one of the following is associated with aortic dissection, except:

A. Ventricular septal defect

B. Turner's syndrome

C. Noonan's syndrome

D. Pregnancy

E. Marfan's syndrome

Aortic dissection

Aortic dissection may be classified as type A or B:

type A - ascending aorta, 2/3 of cases

type B - descending aorta, distal to left subclavian origin, 1/3 of cases

Associations

hypertension

trauma

bicuspid aortic valve

collagens: Marfan's syndrome, Ehlers-Danlos syndrome

Turner's and Noonan's syndrome

pregnancy

syphilis

Complications of backward tear

aortic incompetence/regurgitation

MI: inferior pattern often seen due to right coronary involvement

Complications of forward tear

unequal arm pulses and BP

stroke

renal failure

Question 150

A 58-year-old man who is taking lithium for bipolar disorder presents for review. During routine examination he found to be hypertensive with a blood pressure of 166/82 mmHg. This is confirmed with two separate readings. Urine dipstick is negative and renal function is normal. What is the most appropriate medication to start?

A. Amlodipine

B. Ramipril

C. Losartan

- D. Bendroflumethiazide
- E. Doxazosin

Diuretics, ACE-inhibitors and angiotensin II receptor antagonists may cause lithium toxicity.

Looking at the NICE hypertension guidelines amlodipine wouldn't be a bad first choice, even if we ignore his lithium treatment

Lithium

Lithium is mood stabilising drug used most commonly prophylactically in bipolar disorder but also as an adjunct in refractory depression. It has a very narrow therapeutic range (0.5-1.2 mmol/L) and a long plasma half-life being excreted primarily by the kidneys

Mechanism of action - not fully understood, two theories:

- interferes with inositol triphosphate formation
- interferes with cAMP formation

Adverse effects

- nausea/vomiting, diarrhoea
- fine tremor
- polyuria
- thyroid enlargement, may lead to hypothyroidism
- ECG: T wave flattening/inversion
- weight gain

Lithium toxicity generally occurs following concentrations > 1.5 mmol/L.

Toxicity may be precipitated by dehydration, renal failure, diuretics (especially bendroflumethiazide) or ACE inhibitors

Features of toxicity

- coarse tremor (a fine tremor is seen in therapeutic levels)
- acute confusion
- seizure
- coma

Management

mild-moderate toxicity may respond to volume resuscitation with normal saline haemodialysis may be needed in severe toxicity sodium bicarbonate is sometimes used but there is limited evidence to support this. By increasing the alkalinity of the urine it promotes lithium excretion

Question 151

Which one of the following treatments is not appropriate in the management of Wolff-Parkinson White?

- A. Verapamil**
- B. Sotalol
- C. Amiodarone
- D. Flecainide
- E. Radiofrequency ablation of the accessory pathway

Verapamil and digoxin should be avoided in patients with Wolff-Parkinson White as they may precipitate VT or VF

Wolff-Parkinson White

Wolff-Parkinson White (WPW) syndrome is caused by a congenital accessory conducting pathway between the atria and ventricles leading to a atrioventricular re-entry tachycardia

(AVRT). As the accessory pathway does not slow conduction AF can degenerate rapidly to VF

Possible ECG features include:

- short PR interval
- wide QRS complexes with a slurred upstroke - 'delta wave'
- left axis deviation if right-sided accessory pathway*
- right axis deviation if left-sided accessory pathway*

Differentiating between type A and type B

type A (left-sided pathway): dominant R wave in V1

type B (right-sided pathway): no dominant R wave in V1

Associations of WPW

HOCM

mitral valve prolapse

Ebstein's anomaly

thyrotoxicosis

secundum ASD

Management

definitive treatment: radiofrequency ablation of the accessory pathway

medical therapy: sotalol**, amiodarone, flecainide

*in the majority of cases, or in a question without qualification, Wolff-Parkinson-White syndrome is associated with left axis deviation

**sotalol should be avoided if there is coexistent atrial fibrillation as prolonging the refractory period at the AV node may increase the rate of transmission through the accessory pathway, increasing the ventricular rate and potentially deteriorating into ventricular fibrillation

Question 152

Which of the following is least associated with a poor prognosis in hypertrophic cardiomyopathy?

- A. Non-sustained ventricular tachycardia on 24 or 48-hour Holter monitoring
- B. Reduced left ventricular outflow gradient
- C. Family history of sudden death
- D. Syncope
- E. Early age at presentation

There is no recognised prognostic association with left ventricular outflow gradient

HOCM: prognostic factors

Hypertrophic obstructive cardiomyopathy (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins. Mutations to various proteins including beta-myosin, alpha-tropomyosin and troponin T have been identified. Septal hypertrophy causes left ventricular outflow obstruction. It is an important cause of sudden death in apparently healthy individuals

Poor prognostic factors

- syncope
- family history of sudden death
- young age at presentation
- non-sustained ventricular tachycardia on 24 or 48-hour Holter monitoring

abnormal blood pressure changes on exercise

An increased septal wall thickness is also associated with a poor prognosis

Question 153

A 55-year old man with a history of ischaemic heart disease is presents to the Emergency Department with palpitations. Examination of his pulse reveals a rate of 130 bpm which is irregularly irregular. He has had one previous episode of atrial fibrillation 3 months ago which was terminated by elective cardioversion following warfarinisation. What term best describes his arrhythmia?

- A. Paroxysmal atrial fibrillation
- B. Atrial flutter
- C. Permanent atrial fibrillation
- D. Persistent atrial fibrillation**
- E. Secondary atrial fibrillation

Atrial fibrillation: classification

An attempt was made in the joint American Heart Association (AHA), American College of Cardiology (ACC) and European Society of Cardiology (ESC) 2002 guidelines to simplify and clarify the classification of atrial fibrillation (AF).

It is recommended that AF be classified into 3 patterns:

first detected episode (irrespective of whether it is symptomatic or self-terminating)
recurrent episodes, when a patient has 2 or more episodes of AF. If episodes of AF terminate spontaneously then the term paroxysmal AF is used. Such episodes last less than 7 days (typically < 24 hours). If the arrhythmia is not self-terminating then the term persistent AF is used. Such episodes usually **last greater than 7 days in permanent AF there is continuous atrial fibrillation which cannot be cardioverted** or if attempts to do so are deemed inappropriate. Treatment goals are therefore rate control and anticoagulation if appropriate

Question 154

When is the optimum time to perform a percutaneous coronary intervention following the successful thrombolysis of an anterior ST-elevation myocardial infarction?

- A. Immediately
- B. The next day**
- C. At 3-5 days
- D. At 7-10 days
- E. After 14 days

Myocardial infarction: management

A number of studies over the past 10 years have provided an evidence for the management of ST-elevation myocardial infarction (STEMI)

In the absence of contraindications, all patients should be given aspirin

clopidogrel: the two major studies (CLARITY and COMMIT) both confirmed benefit but used different loading doses (300mg and 75mg respectively)

low molecular weight heparin:

Primary percutaneous coronary intervention (PCI) has emerged as the gold-standard treatment for STEMI but is not available in all centres.

Thrombolysis should be performed in patients without access to primary PCI

With regards to thrombolysis:

tissue plasminogen activator (tPA) has been shown to offer clear mortality benefits over streptokinase

tenecteplase is easier to administer and has been shown to have non-inferior efficacy to alteplase with a similar adverse effect profile

An ECG should be performed 90 minutes following thrombolysis to assess whether there has been a greater than 50% resolution in the ST elevation

if there has not been adequate resolution then rescue PCI is superior to repeat thrombolysis
for patients successfully treated with thrombolysis PCI within 24 h has been shown to be beneficial

Question 155

A 69-year-old man is reviewed. He was recently admitted after being found to be in atrial fibrillation. This was his second episode of atrial fibrillation. He also takes ramipril for hypertension but has no other history of note. During admission he was warfarinised and discharged with planned follow-up in the cardiology clinic. However, on review today he is found to be in sinus rhythm. What should happen regarding anticoagulation?

- A. Stop warfarin
- B. Continue warfarin for 1 month
- C. Stop warfarin + start aspirin
- D. Continue lifelong warfarin**
- E. Continue warfarin for 6 months

Warfarin should be continued indefinitely as this is his second episode of atrial fibrillation and he has risk factors for stroke (age, hypertension)

Atrial fibrillation: anticoagulation

The Royal College of Physicians and NICE published guidelines on the management of atrial fibrillation (AF) in 2006

The guidelines suggest a stroke risk stratification approach when determining how to anticoagulate a patient, as detailed below:

Low risk - annual risk of stroke = 1%

age < 65 years with no moderate or high risk factors
use aspirin

Moderate risk - annual risk of stroke = 4%

age > 65 years with no high risk factors, or:
age < 75 years with diabetes, hypertension or vascular disease (ischaemic heart disease or peripheral arterial disease)
use aspirin or warfarin depending on individual circumstances

High risk - annual risk of stroke = 8-12%

age > 75 years with diabetes, hypertension or vascular disease (ischaemic heart disease or peripheral arterial disease)
previous TIA, ischaemic stroke or thromboembolic event
valve disease, heart failure or impaired left ventricular function
use warfarin

Question 156

A 60-year-old man is admitted with palpitations to the Emergency Department. An ECG on admission shows a broad complex tachycardia at a rate of 150 bpm. His blood pressure is 124/82 mmHg and there is no evidence of heart failure. Which one of the following is it least appropriate to give?

- A. Procainamide
- B. Lidocaine
- C. Synchronised DC shock
- D. Adenosine
- E. Verapamil

Ventricular tachycardia - verapamil is contraindicated

Verapamil should never be given to a patient with a broad complex tachycardia as it may precipitate ventricular fibrillation in patients with ventricular tachycardia. Adenosine is sometimes given in this situation as a 'trial' if there is a strong suspicion the underlying rhythm is a supraventricular tachycardia with aberrant conduction

Ventricular tachycardia: management

Whilst a broad complex tachycardia may result from a supraventricular rhythm with aberrant conduction, the European Resuscitation Council advise that in a peri-arrest situation it is assumed to be ventricular in origin

If the patient has adverse signs (**systolic BP < 90 mmHg, chest pain, heart failure or rate > 150 beats/min**) then immediate **cardioversion** is indicated. In the absence of such signs antiarrhythmics may be used. If these fail, then electrical cardioversion may be needed with synchronised DC shocks

Drug therapy

amiodarone: ideally administered through a central line
lidocaine: use with caution in severe left ventricular impairment
procainamide

Verapamil should NOT be used in VT

If drug therapy fails

electrophysiological study (EPS)
implantable cardioverter-defibrillator (ICD) - this is particularly indicated in patients with significantly impaired LV function

Question 157

What is the most appropriate dose of adrenaline to give during a cardiac arrest?

- A. 1ml 1:100,000 IV
- B. 10ml 1:1,000 IV
- C. 0.5ml 1:1,000 IM
- D. 1ml 1:10,000 IV
- E. 10ml 1:10,000 IV

Recommend Adult Life Support (ALS) adrenaline doses

anaphylaxis: 0.5ml 1:1,000 IM

cardiac arrest: 10ml 1:10,000 IV or 1ml of 1:1000 IV

10ml of the 1:10,000 preparation contains 1mg of adrenaline

Adrenaline

Adrenaline is a sympathomimetic amine with both alpha and beta adrenergic stimulating properties

Indications

anaphylaxis

cardiac arrest

Recommend Adult Life Support (ALS) adrenaline doses

anaphylaxis: 0.5ml 1:1,000 IM

cardiac arrest: 10ml 1:10,000 IV or 1ml of 1:1000 IV

Management of accidental injection

local infiltration of phentolamine

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