Cardiovascular

Rapid Emergency and Unscheduled Care, First Edition. Oliver Phipps and Jason Lugg.

Abdominal aortic aneurysm

Definition

An abdominal aortic aneurysm (AAA) is defined as an enlargement of the aorta by at least 1.5 times its normal diameter. The normal diameter of the aorta is ~2 cm and increases with age. Most AAA are small and not dangerous; however when they increase in size, they are prone to rupture causing a life-threatening condition.

Epidemiology

It is estimated that in 95% of patients, AAA is a complication of atherosclerosis. Risk factors include being male, hypertension, increasing age, smoking and a family history of AAA.

History

- Asymptomatic and often detected on routine abdominal imaging or NHS screening programme.
- Patient may feel pulsatile mass in abdomen.
- Backache.
- Aching pain in the epigastrium and central abdomen to the back.
- In rupture the patient will have severe abdominal pain, often epigastric and radiating to the back.
- May be accompanied by collapse.
- Symptoms can be similar to renal colic.

Examination

The patient should be assessed using the ABCDE approach with appropriate step interventions. Specific points to increase the likely diagnosis of a ruptured AAA include:

- Signs of shock
- Abdominal tenderness and guarding
- Palpable abdominal mass – often pulsatile
- Weak or absent lower limb pulses

Investigations

- Bloods:
  - FBC
  - U&Es
  - LFTs
  - Clotting screen
  - Cross-match
- Arterial blood gas
- ECG
- CXR and AXR
- CT abdomen
- FAST ultrasound scan

Management

- Transfer direct to the emergency department (ED) with pre-alert.
- ABCDE approach.
- Oxygen (set SpO_2 target).
- IV access × 2.
- Cautious IV fluid resuscitation to maintain blood pressure (systolic ~90 mmHg or radial pulse presence), ideally with blood products.
- Analgesia.
- Early discussion with appropriate surgeons.
- Prepare for theatre.

Acute coronary syndrome

Definition

Acute coronary syndrome (ACS) is an umbrella term that encompasses:

- Unstable angina
- Non-ST segment elevation myocardial infarction (NSTEMI)
- ST segment elevation myocardial infarction (STEMI)

Aetiology

ACS is commonly caused by rupture of an atheromatous plaque in a coronary artery. This results in the accumulation of fibrin and platelets to repair the damage. This results in a thrombus formation leading to partial or complete occlusion of the coronary artery and distal myocardial cell death.

Epidemiology

Around 114 000 patients with ACSs are admitted to the hospital each year in the United Kingdom. Coronary heart disease (CHD) is the most common cause of death in the United Kingdom with around one in five men and one in seven women dying each year from CHD.

History

- Consider the history of chest pain or discomfort.
- Cardiovascular (CVS) risk factors.
- Family history of CHD.
- History of CHD, previous treatment and investigations:
- Pain or discomfort in the chest and/or the arms, back or jaw lasting longer than 15 minutes
- Chest pain with nausea and vomiting, sweating and/or breathlessness
- Abrupt deterioration in stable angina, with recurring chest pain discomfort occurring more frequently with little or no exertion and often lasting longer than 15 minutes.

Examination

- Clinical examination is often of little value in diagnosing ACS.
- It can identify alternative causes of chest pain (localised tenderness).
- Look for evidence of the aforementioned symptoms (sweating, SOB, shock).
- Full CVS, respiratory and abdominal assessment.
- Look for signs of heart failure.
- Examine chest wall for local tenderness and other possible causes of chest pain (costochondritis).

Investigations

- Vital signs – RR, HR, BP (both arms) and SpO_2
- Cardiac monitoring – to identify underlying rhythm and arrhythmias
- 12-Lead ECG:
  - To confirm a cardiac basis for presentation and may show pre-existing structural or CHD.
  - ECG changes that occur during episodes of angina (ischaemia) T-wave inversion or ST segment depression.
  - Look for ST segment elevation suggestive of an STEMI.
- Bloods:
  - FBC, U&Es, LFTs, clotting screen and glucose
  - Troponin – should be taken immediately in suspected ACS, but negative result can only be used to rule ACS at 6 and 12 hours, respectively
- CXR – useful to show complications of ischaemia (e.g. pulmonary oedema) or to explore alternative diagnoses (e.g. pneumothorax, aortic aneurysm)

Acute coronary syndrome (continued)

Management

- Refer to local protocols and care pathways.
- 999 Ambulance is required for transfer direct to cardiology in cases of STEMI for primary coronary intervention (PCI) or ED in other cases of ACS.
- IV access.
- IV morphine (dose titrated to pain with antiemetic).
- Oxygen (as required to meet target oxygen saturation of 94–98%).
- Nitrates (GTN if systolic BP > 90 mmHg).
- Aspirin (stat dose of 300 mg).

TOP TIP:

- Chest pain relieved by GTN does not exclude ACS.
- A normal ECG does not exclude an ischaemic cause.

Anaphylaxis

Definition

Anaphylaxis is a severe, life-threatening and systemic hypersensitivity reaction to a foreign protein. Common examples include drugs, food products and insect stings. The resulting vasodilation and bronchospasm causes life-threatening symptoms.

Aetiology

True anaphylaxis does not occur on the first exposure to the allergen as the patient needs to have been exposed previously and therefore sensitised to the protein. Further repeated exposure leads to significant histamine release that increases on each subsequent exposure.

Epidemiology

The incidence of anaphylaxis is increasing in the United Kingdom and is suggested to be around 1–3 reactions per 10 000 population per annum. The overall prognosis of anaphylaxis is good. Mortality is increased within the asthmatic population, specifically those with poorly controlled asthma. Mortality rates from anaphylaxis in the United Kingdom are estimated at around 20 per annum.

History

- May be PMH of anaphylaxis or allergic response
- Sudden onset of symptoms (usually within minutes)
- Identifiable trigger (not always possible)

Examination

Patients with suspected anaphylaxis should be assessed using the ABCDE approach as follows:

Airway

- Hoarse voice
- Airway swelling
- Stridor

Breathing

- Shortness of breath
- Tachypnoea
- Tiredness/exhaustion
- Cyanosis
- Respiratory arrest

Anaphylaxis (continued)

Circulation

- Signs of shock (pale and clammy)
- Tachycardia
- Hypotension
- Cardiac arrest

Skin/Mucosal

- Often first feature
- Erythema
- Urticaria
- Angioedema

Others

- Gastrointestinal disturbance (abdominal pain, vomiting and diarrhoea)

Investigations

- Investigation should not delay resuscitation.
- Vital sign monitoring should be established (RR, SpO_2, HR and ECG monitoring).
- 12-Lead ECG.
- CXR.
- ABG.
- Bloods (including mast-cell tryptase to confirm anaphylaxis diagnosis).

Management

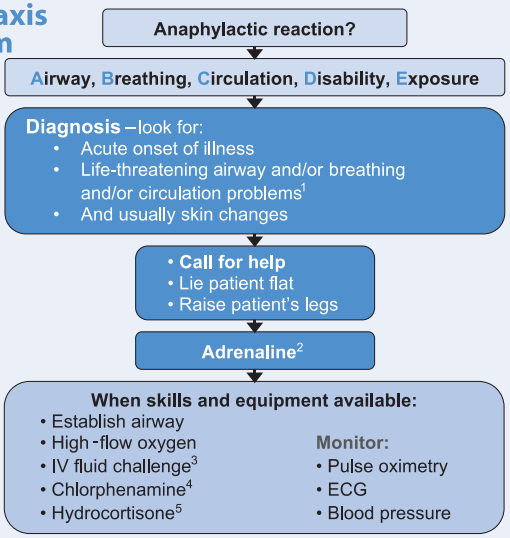
- Call for help.
- Lie flat and raise legs (some patients may benefit from sitting up if respiratory distress is the key feature, blood pressure is not compromised and the patient is not feeling dizzy or does not faint).
- Give intramuscular adrenaline.*
- High flow oxygen.
- IV access and fluid challenges of 500–1000 ml in adults and 20 ml/kg in children.*
- IV antihistamine.*
- IV steroids.*

*Please see the latest guidelines for specific drugs and doses.

Please refer to the latest guidelines from the Resuscitation Council (UK) available at www.resus.org.uk.

Resuscitation Council (UK)

Anaphylaxis algorithm



[1] Life-threatening problems:

Airway:	swelling, hoarseness, stridor
Breathing:	rapid breathing, wheeze, fatigue, cyanosis, SpO_2 < 92%, confusion
Circulation:	pale, clammy, low blood pressure, faintness, drowsiness/coma

[2] Adrenaline *(give IM unless experienced with IV adrenaline)*

IM doses of 1 : 1000 adrenaline (repeat after 5 minutes if no relief)

- Adult 500 µg IM (0.5 ml)
- Child > 12 years: 500 µg IM (0.5 ml)
- Child 6–12 years: 300 µg IM (0.3 ml)
- Child < 6 years: 150 µg IM (0.15 ml)

Adrenaline IV to be given **only by experienced specialists**

Titrate: Adults 50 µg ; children 1 µg/kg

[3] IV fluid challenge:

Adult – 500–1000 ml

Child – crystalloid 20 ml/kg

Stop IV colloid as this might be the cause of anaphylaxis

	[4] Chlorphenamine (IM or slow IV)	[5] Hydrocortisone (IM or slow IV)
Adult or child > 12 years	10 mg	200 mg
Child 6–12 years	5 mg	100 mg
Child 6 months to 6 years	2.5 mg	50 mg
Child < 6 months	250 µg/kg	25 mg

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Aortic dissection (thoracic)

Definition

Aortic dissection is the tearing within the thoracic aorta allowing for blood to create a false lumen between the inner and the outer tunica media. There are different types classified by location. Type A involves the ascending aorta and is most common, whereas type B involves the descending aorta. Aortic dissection can lead to occlusion of the aorta and its branches (carotid, coronary, subclavian, spinal, coeliac, renal), each presenting with associated symptoms.

Epidemiology

Thoracic aortic dissection more commonly occurs between the ages of 40 and 60 years of age, with morbidity twice as likely in males.

History

- Sudden onset of severe chest pain
- Pain often described as tearing in nature
- Pain often radiates to the back
- May be accompanied by collapse

Examination

The patient should be assessed using the ABCDE approach with appropriate step interventions. Specific points to strengthen the likely diagnosis of thoracic dissection include:

- A murmur heard below the left scapula
- BP discrepancy of >20 mmHg in both arms
- Widening pulse pressure
- Comprehensive CVS examination noting peripheral pulse

Investigations

- Bloods:
  - FBC
  - U&Es
  - LFTs
  - Clotting screen
  - Cross-match
- Arterial blood gas
- Chest X-ray – widened mediastinum bulge of the aortic arch but can be normal
- ECG – may be normal but may show left ventricular hypertrophy
- Thoracic CT
- Echocardiography

Management

- Transfer directly to the ED with pre-alert (ideally where cardiothoracic specialty is located – refer to local protocols).
- ABCDE approach.
- Oxygen (set SpO_2 target).
- IV access × 2.
- Cautious IV fluid resuscitation to maintain blood pressure (systolic ~90 mmHg or radial pulse presence), ideally with blood products.
- Early discussion with appropriate surgical specialty.

Atrial fibrillation

Definition

Atrial fibrillation (AF) is an arrhythmia. It results from irregular, disorganised electrical activity in the atria, leading to an inconsistent and irregular ventricular response. AF if left untreated can lead to complications that include stroke, TIA, thromboembolism, heart failure and tachycardia-induced cardiomyopathy.

Aetiology

In just over 10% of cases of AF, no cause is identified. However, common causes include heart valve deformities, rheumatic heart disease, heart failure, myocarditis, pericarditis, sepsis, electrolyte imbalance and excessive caffeine or alcohol consumption. Some drugs can also increase the risk of AF and include thyroxine or bronchodilators.

Epidemiology

AF is the most common cardiac arrhythmia in the United Kingdom with a morbidity of over 800 000 and an increasing prevalence.

History

- Palpitations
- Breathlessness
- Chest discomfort
- Syncope or dizziness
- Reduced exercise tolerance

Examination

- Often unremarkable
- Irregularly irregular pulse
- May be signs of heart failure

Investigations

- ECG – confirms diagnosis (no identifiable P waves with chaotic base line and irregular ventricular response). The ventricular rate is commonly between 160 and 180 bpm but can be less in asymptomatic patients.
- Bloods:
  - FBC (to identify anaemia and increased WCC in sepsis)
  - TFTs (hyperthyroidism is a cause of AF)
  - U&Es (to identify other metabolic causes)
  - CXR (if pulmonary cause is suspected and to assess heart failure or cause of sepsis)
  - Echocardiogram (to identify structural or valvular causes)

Management

- Refer to national or local guidelines.
- Admit if:
  - HR > 150 bpm or systolic BP < 90 mmHg
  - Any of the following are evident: LOC, dizziness, chest pain and SOB
  - Any signs of complications (CVA, TIA or heart failure) are present
  - Underlying cause requires inpatient management
- Treatment strategy focuses on either:
  - Treating and managing underlying cause.

Atrial fibrillation (continued)

- *Rhythm control* – this can be done acutely if patients present within 48 hours of onset or electively in patients who present later than 48 hours after onset who need anti-coagulation due to the risk of thrombotic emboli with prophylaxis against further episodes of AF with medication (β-blockers, amiodarone or flecainide).
- *Rate control* – for those patients in whom the time of onset is not known or who are not suitable for cardioversion (digoxin, β-blockers and verapamil).

Bradycardia

Definition

Bradycardia refers to a heart rate of <60 bpm; however for some individuals, this is not harmful and in fact is physiological for them. Extreme bradycardia is referred to when the heart rate is <40 bpm and is rarely physiologically tolerated.

History

- Chest pain
- Palpitations
- SOB
- Fatigue and exercise tolerance
- Dizziness, syncope or collapse
- Medication with focus on drugs that can cause bradycardia (β-blockers, calcium channel blockers, digoxin, amiodarone, verapamil)

Examination

- Does the patient look well?
- ABCDE assessment if patient is unwell.
- Full CVS, respiratory and abdominal assessment.
- Are there signs of poor cardiac output, for example, cold peripheries and cyanosis?
- Are there signs of cardiac failure?

Investigations

- ECG monitoring – to identify underlying rhythm and any arrhythmias
- Vital signs (HR, RR, BP, SpO_2, temperature) – to identify systemic effects
- 12-Lead ECG – to identify rhythm and other ECG abnormalities
- Bloods (FBC, U&Es, LFTs, glucose, calcium, cardiac enzyme, TFTs and digoxin level if indicated) to identify possible underlying causes
- CXR – as clinically indicated

Management

The focus for any patient with bradycardia is whether the rate is causing harmful adverse effects as follows:

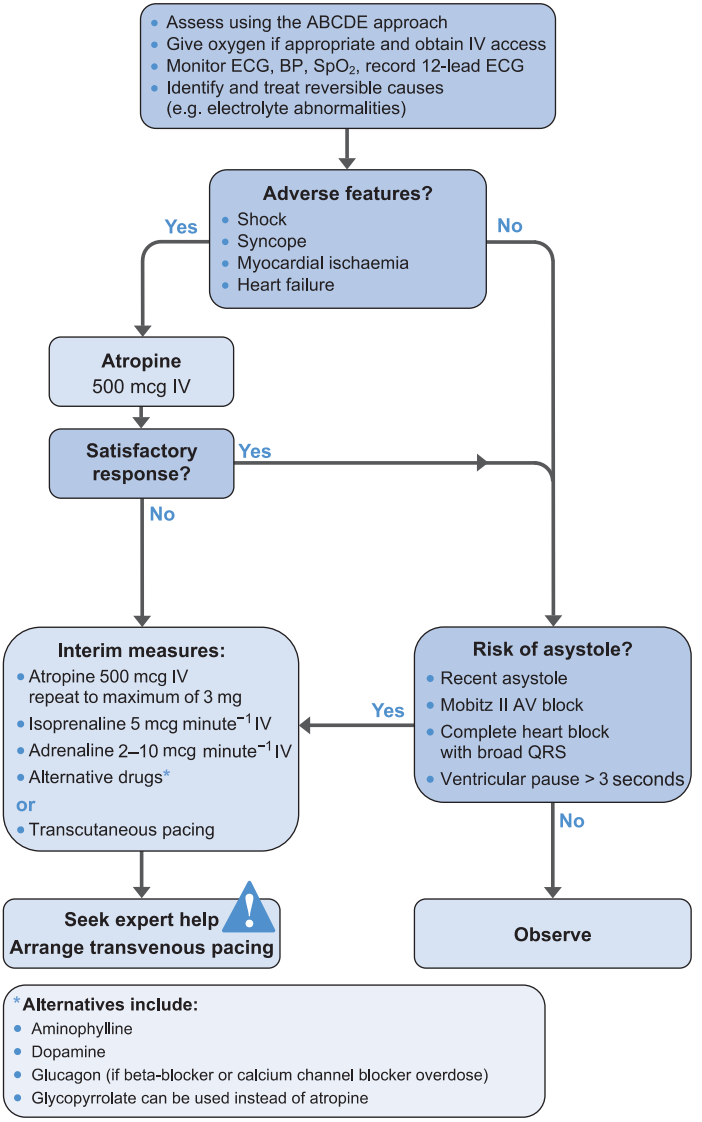
- Systolic <90 mmHg
- Heart rate <40 bpm
- Ventricular arrhythmias
- Heart failure

Acutely unwell patient will need to be referred to an ED for ongoing care. Stable patients with no adverse effects can be managed in primary care.

2010 Resuscitation Guidelines

Resuscitation Council (UK)

Adult bradycardia algorithm



Deep vein thrombosis

Definition

Deep vein thrombosis (DVT) refers to the formation of a thrombus (blood clot) in a deep vein leading to a partial or complete obstruction of blood flow. Most commonly this occurs in the deep veins of the leg or pelvis, but the upper limbs can also be affected, as can intracranial and abdominal veins. The thrombus can dislodge and enter the pulmonary arteries, causing a pulmonary embolism.

DVT is more likely to occur in the following:

- Age over 60 years
- Male sex
- Previous venous thromboembolism
- Cancer (known or undiagnosed)
- Being overweight or obese
- Patients with heart failure
- Acquired or familial thrombophilia
- Patients with vasculitis and hypoxia from venous stasis or undergoing chemotherapy
- Varicose veins
- Smokers

The transient risk of DVT is increased in the following:

- Immobility (e.g. following illness, the use of plaster cast or other limb immobilisation devices, surgery, hospitalisation or during long-distance travel)
- Significant trauma or direct trauma to a vein, for example, intravenous catheter or IV drug use
- Hormone treatment
- Pregnancy and the postpartum period
- Dehydration

Epidemiology

It is estimated that DVT occurs in around 1 in 1000 patients.

History

- Lower limb pain, swelling or tenderness
- Can be asymptomatic
- May present with signs of PE

Examination

- Examine the entire limb for swelling and tenderness and compare to other limb.
- Measure circumference of each calf at same point.
- Inspect for dilated superficial veins.
- CVS and respiratory assessment to identify signs of PE.

Wells DVT score	Score
Lower limb trauma or immobilisation	+1
Bedridden for >3 days within the last 4 weeks	+1
Tenderness along the deep vein system	+1
Entire limb swollen	+1

Deep vein thrombosis (continued)

Wells DVT score	Score
Calf >3 cm in circumference (10 cm below the tibial tuberosity)	+1
Pitting oedema	+1
Dilated collateral superficial veins (non-varicose)	+1
Malignancy	+1
Alternative diagnosis more likely than DVT	–2
Clinical probability of DVT	High >3
	Moderate 1–2
	Low <1

Investigations
- Doppler ultrasound for medium- and high-risk patients.
- D-dimer (high sensitivity but low specificity) therefore should be used for low-probability patients to rule out DVT with negative result.
- FBC, U&Es, LFTs and clotting screen before starting anticoagulant therapy.
- ECG, CXR and ABG if PE is suspected.

Management
- Please refer to local guidance and protocols including the availability of thrombosis clinics for initial management.
- Unless contraindicated, patient should be treated with anticoagulation usually for 3 months.
- Patient should be treated with heparin until therapeutic INR is at target levels.
- High-risk patients where anticoagulation is contraindicated may need an IVC filter to prevent PE.

Heart failure
Definition
Heart failure refers to a syndrome when the heart's ability to maintain adequate circulation of blood is impaired. This results in a variety of symptoms including breathlessness and signs of fluid retention (pulmonary and/or peripheral oedema).

Epidemiology
The prevalence of heart failure increases with age. The British Heart Foundation estimates that around 1% of people aged between 45 and 64 years suffer from heart failure, increasing to 7% for people 75–84 years of age and 20% for people 85 years of age or older. Men are more likely than women to have heart failure.

History
Left-sided heart failure – Symptoms of pulmonary oedema (SOB, orthopnoea, wheeze, cough, pink frothy sputum)
Right-sided heart failure – Swollen ankles, fatigue, ↑ weight and ↓ exercise tolerance

Heart failure (continued)

Examination

- Peripheral oedema
- SOB
- Wheeze
- Cough
- Bilateral basal crackles and fine crackles throughout the lung field
- Heart murmur
- Raised JVP
- Hepatomegaly
- Ascites
- Tachycardia

Acute Left Ventricular Failure
Acutely unwell with SOB, acute respiratory distress, anxiety/agitation, cough with pink frothy sputum and signs of cardiogenic shock

Investigations

- Vital signs (RR, HR, BP, SpO_2 and temperature)
- Bloods – FBC, U&Es, LFTs, CRP, glucose, lipids, TFTs and troponin and ABG if acute LVF (to identify underlying causes and associated co-morbidities).
- CXR – for signs of cardiomegaly and pulmonary oedema
- ECG – to identify ischaemia, left ventricular hypertrophy and arrhythmias (may be normal)
- Echocardiogram – to assess the adequacy of ventricular contraction and ejection fractions

Management

- Acute LVF (pulmonary oedema) needs rapid intervention that includes the following:
  - Sit patient up.
  - Oxygen therapy if hypoxic.
  - Consider CPAP.
  - GTN (to reduce preload).
  - IV diuretic (if fluid overload).
- Chronic heart failure requires careful management with a clinician experienced in managing heart failure patients (GP, heart failure specialist nurse or cardiologist). Therefore patients who are not acutely unwell should be referred to an appropriate clinician.

  Management involves treating underlying causes and careful medicine management (ACE – inhibitors, β-blockers, diuretics, etc.).

Hypertension

Definition

Hypertension is defined as a systolic BP > 140 mmHg and/or a diastolic BP > 85 mmHg recorded on three separate occasions or following interpretation of ambulatory blood pressure monitoring (ABPM). Blood pressure can also be classified as malignant and typically involves a blood pressure as >200/130 mmHg.

Epidemiology

Hypertension is very common in the western world and affects around 10–20% of adults. The associated CVS risks of hypertension have been demonstrated with various epidemiological studies, with results suggesting that for every 20/10 mmHg rise in blood pressure above 115/70 mmHg, the risk of CVS events doubles.

Hypertension (continued)

History

- Commonly asymptomatic.
- Symptoms that do occur are often due to complications and causes of hypertension.
- Malignant hypertension symptoms include visual field loss, blurred vision, headache, nausea/vomiting and acute heart failure.

Examination

- Routine diagnosis of hypertension involves recoding the lowest reading on two or three different occasions.
- In severe, resistant and malignant hypertension, it is important to assess for signs of end-organ damage and includes the following:
  CVS – LVF, ischaemia, MI and aortic dissection
  Central nervous system – stroke, seizures, encephalopathy and subarachnoid haemorrhage
  Renal – acute kidney injury, proteinuria or haematuria

Investigations

- Bloods (FBC, U&Es, calcium, glucose, coagulation profile, glucose and lipids) to assess end-organ damage and general health
- Urinalysis to assess for renal disease
- CXR for signs of cardiomegaly and if presenting with CVS symptoms
- CT of the head if cerebral symptoms exist

Management

- For hypertension with no sign of end-organ damage or symptoms, then refer to GP or practice nurse for follow-up and ongoing management.
- If acutely unwell with symptoms or evidence of end-organ damage, then refer to the medical team or ED as appropriate and in line with local protocols.

Ischaemic lower limb

Definition

Acute limb ischaemia is often due to complete arterial occlusion from a thrombus or an embolus from a proximal distal site. Without surgical revascularisation or heparinisation, extensive tissue necrosis will occur within 6 hours.

History

There may be evidence of chronic vascular compromise (chronic ischaemic pain at rest, ulcers or gangrene in one or both legs) attributable to proven arterial occlusive disease. Explore the rapidity of onset of symptoms, features of pre-existing chronic arterial disease and potential source of embolus.

Examination

- 6 P's – pale, pulseless, painful, paralysed, paraesthetic and perishingly cold limb.
- Compare pulses in the contralateral limb.
- The onset of fixed mottling of the skin implies irreversible changes.
- Check for signs of compartment syndrome.

TOP TIP:
The ischaemic limb may appear red when dependent. This can lead to a misdiagnosis of inflammatory cause, for example, gout or cellulitis.

Ischaemic lower limb (continued)

Investigations

- Doppler ultrasound may help assess residual arterial flow.
- Arteriography.
- Bloods:
  FBC – to check for anaemia as it exacerbates ischaemia
  ESR – to check for inflammatory diseases
  Glucose – to check/assess diabetes
  Lipids
  Clotting screen

Management

- Urgent admission to vascular surgeons (refer to local protocols).
  (this is a limb-threatening emergency and often requires urgent surgery or angioplasty).
- Systemic anticoagulation (usually unfractionated heparin) is often required for patients with acute arterial emboli or thrombus.
- Appropriate analgesia will be required prehospital whilst awaiting transfer to the hospital.

Myocarditis

Definition

Myocarditis refers to acute or chronic inflammation and necrosis of the myocardium.

Aetiology

It is often idiopathic, commonly due to infection (viral, bacteria, fungal) but can be immune mediated, due to drug hypersensitivity or physical element (hyperpyrexia or electric shock).

Epidemiology

Not accurately known due to many cases not identified at stage of acute illness. In Europe and the United States, Coxsackie B virus is the most common cause.

History

Presentation can be variable in myocarditis but include:

- May have prodromal flu-like illness
- Fatigue
- Chest pain
- Fever
- Tachycardia
- Palpitations
- Signs of heart failure
- Shortness of breath

Examination

- Does the patient look well?
- ABCDE assessment if patient is unwell.
- Full CVS, respiratory and abdominal assessment as minimum.
- Are there signs of cardiac failure?

Investigations

- 12-Lead ECG (may show ST segment depression and elevation or T-wave inversion)
- Bloods (FBC, U&E, ESR, CRP and cardiac enzymes)

Myocarditis (continued)

- CXR (may show hear failure or pleural effusion)
- Echocardiogram (to assess for function, wall motion and the presence of a pericardial effusion)
- Pericardial fluid drainage or myocardial biopsy

Management

- Rest.
- Focus on treating complications (arrhythmia, heart failure).
- May require critical support (CCU or ITU).
- Corticosteroids are sometimes used but evidence for use is unproven.
- Patients in prehospital setting will require admission to the hospital ideally with referral to cardiology.

Pericarditis

Definition

Pericarditis refers to the inflammation of the pericardium, which can be acute or chronic.

Aetiology

Commonly idiopathic but can be due to infection (viral, bacteria or fungal), connective tissue disease (SLE), or post-myocardial infarction or secondary to malignancy.

Epidemiology

It is more common in males and accounts for <1 in 100 hospital admissions.

History

Patients often describe initial prodromal flu-like symptoms followed by other symptoms that vary depending on the severity of the inflammation as follows:

- Chest pain (often sharp and central)
- Chest pain pleuritic in nature
- Pain relieved by sitting forward
- Shortness of breath
- Fever
- Nausea

Examination

- Full CVS, respiratory and abdominal assessment as minimum.
- May hear pericardial friction rub at the lower left sternal edge
- Occasional cardiac tamponade – look for Beck's triad (↑ JVP, ↓ BP and muffled heart sounds)

Investigations

- Bloods (FBC, U&E, ESR, CRP and cardiac enzymes) to assess for infection markers and general health
- ECG (may have widespread saddle-shaped ST elevation)
- CXR (may be normal or show pericardial effusion)
- Echocardiogram (to assess for function, wall motion and the presence of a pericardial effusion)

Management

- Treat the underlying cause if known.
- High-dose NSAIDs.
- Patients with evidence of significant tamponade require urgent pericardiocentesis.

Shock

Definition

Shock is defined as acute circulatory failure resulting in inadequate tissue perfusion resulting in generalised cellular hypoxia.

Aetiology

Causes of shock include:

- *Hypovolaemic* due to loss of circulating volume (haemorrhage, burns, dehydration)
- *Cardiogenic* due to pump failure (MI, arrhythmia or drug induced)
- *Distributive* due to the movement of fluid from normal compartments caused by vasodilation (anaphylaxis, sepsis, spinal shock)
- *Obstructive*, which refers to a reduction in blood flow (PE, tension pneumothorax, cardiac tamponade)

Shock presents in two distinct phases depending on severity as follows:

1. Compensated where the body maintains oxygenation and perfusion by deliberate changes to the physiology examples, which include increased respiratory rate, vasoconstriction and tachycardia.
2. Decompensated where the body is failing to maintain perfusion and oxygenation through compensatory mechanisms with hypotension being the key sign

History

- The underlying cause of shock may be evident (haemorrhage, burns, anaphylaxis), but shock may also be the first presenting feature requiring the underlying cause to be investigated.
- Patients often present with anxiety, confusion or drowsiness.

Examination

- ABCDE assessment and interventions
- Secondary survey guided by suspected underlying cause (CVS, respiratory and abdominal assessment as minimum)
- Identification of early signs (↑ RR, capillary refill) during examination

Investigations

- 12-Lead ECG – to identify rhythm and acute pathology
- Bloods (standard FBC, U&Es, LFTs, glucose, calcium, clotting screen) – will require other bloods guided by suspected underlying cause (cardiac enzymes, group and save, cross-match, etc.)
- Blood gas – to assess overall systemic effects and lactate measurement
- CXR – as clinically indicated

Management

Treatment should be aimed at increasing perfusion along with identifying and managing the underlying cause of shock. Specific treatments include:

- Oxygen titrated to SpO_2 levels (refer to BTS guidance).
- Fluid challenge (refer to local guidelines, consider boluses of 250 ml or 20 ml/kg crystalloid, and be cautious in cardiogenic shock).

Shock (continued)

- Treat the underlying cause of shock.
- Blood.
- Urinary catheterisation and urine output monitoring.
- Critical care review and intervention may be required.

Patients with identified or suspected shock in the prehospital environment require emergency transfer by ambulance to the hospital either to an ED.

Tachycardia

Definition

Tachycardia refers to a heart rate of >100 bpm and is broadly categorised into narrow complex and broad complex tachycardias. Causes can be varied and include medication, disease, pain, fever, hyperthyroidism, exertion, hypoxia and stimulant drugs (e.g. caffeine, cocaine, amphetamines).

History

- Chest pain
- Palpitations
- SOB
- Fatigue and exercise tolerance
- Dizziness, syncope or collapse
- Medication including recreational drug use

Examination

- Does the patient look well?
- ABCDE assessment if patient is unwell.
- Full CVS, respiratory and abdominal assessment.
- Are there signs of poor cardiac output, for example, cold peripheries and cyanosis?
- Are there signs of cardiac failure?

Investigations

- ECG monitoring – to identify underlying rhythm and any arrhythmias
- Vital signs (HR, RR, BP, SpO_2, temperature) – to identify systemic effects
- 12-Lead ECG – to identify rhythm and other ECG abnormalities
- Bloods (FBC, U&Es, LFTs, glucose, calcium, cardiac enzyme and TFTs) to identify possible underlying causes
- CXR – as clinically indicated

Management

The focus for any patient with tachycardia is whether the rate is causing harmful adverse effects as follows:

- Systolic <90 mmHg
- Heart rate <40 bpm
- Ventricular arrhythmias
- Heart failure

Acutely unwell patient will need to be referred to an ED for ongoing care.

2010 Resuscitation Guidelines

Resuscitation Council (UK)

Adult tachycardia (with pulse) algorithm