

Acute pericarditis

This characteristically produces chest pain, low-grade fever \pm intermittent pericardial friction rub. Pericarditis and myocarditis often coexist.

Causes

- Viruses (Coxsackie virus A9, B1–4, echovirus 8, mumps, EBV, CMV, varicella, HIV, rubella, parvovirus B19)) are believed to be responsible for most cases affecting previously well young or middle-aged adults.
- MI (including Dressler's syndrome—see Pericarditis, management, p. 83).
- Bacterial infection (pneumococcus, meningococcus, *Chlamydia*, gonorrhoea, *Haemophilus*).
- Tuberculosis (TB) (especially in patients with HIV) (see Tuberculosis, p. 242).
- Locally invasive carcinoma (eg bronchus or breast).
- Rheumatic fever (see Rheumatic fever, p. 513).
- Uraemia.
- Collagen vascular disease: systemic lupus erythematosus (SLE), polyarteritis nodosa (PAN), rheumatoid.
- After cardiac surgery or radiotherapy.
- Drugs (hydralazine, procainamide, methyldopa, minoxidil).

Diagnosis

Classical features of acute pericarditis are pericardial pain, friction rub, and concordant ST elevation on ECG. The characteristic combination of clinical presentation and ECG changes often results in a definite diagnosis.

- Chest pain is typically sharp, central, retrosternal, and worse on deep inspiration, and change in position, on exercise, and on swallowing. A large pericardial effusion may cause dysphagia by compressing the oesophagus.
- A pericardial friction rub is often intermittent, positional, and elusive. It tends to be louder during inspiration and may be heard in both systole and diastole. Low-grade fever is common.
- Appropriate investigations include: ECG, CXR, FBC, CRP, U&E, and troponin. Note troponin may be ↑ in pericarditis—consider repeat/serial troponins \pm other investigations if there is doubt about the underlying cause. Obtain blood cultures if there is evidence of sepsis or suspicion of a bacterial cause (eg spread of intrathoracic infection). A pericardial effusion is most quickly and easily demonstrated by bedside echocardiography; clinical evidence of cardiac tamponade is rare.

ECG changes

In acute pericarditis, ECG changes result from associated epicardial inflammation (see Fig. 3.10). Sinus tachycardia is usual, but AF, atrial flutter, or atrial ectopics may occur. ST elevation is concave up (unlike MI—see Myocardial infarction: ECG changes 1, p. 76) and present in at least two limb leads and all chest leads (most marked in V_{3–6}). T waves are initially prominent, upright, and peaked, becoming flattened or inverted over several days. PR depression (reflecting atrial inflammation) may occur in the same leads as ST elevation (this PR-ST discordance is characteristic). Pathological Q waves are not present.

Pericardial effusion causes ↓ QRS amplitude in all leads. Electrical alternans is diagnostic, but rare.

Management

The appropriate treatment depends on the underlying cause.

Idiopathic pericarditis or viral pericarditis in young patients is usually benign and self-limiting. Admit patients with high-risk features: pyrexia $>38^{\circ}\text{C}$, ↑ WCC, large pericardial effusion/tamponade, acute trauma, immunosuppression, oral anticoagulation, failure of NSAID treatment (see <https://www.rcemlearning.co.uk>). Advise rest, with avoidance of exercise/sport until symptoms resolve. Occasionally, it follows a relapsing course before 'burning itself out'. If symptoms do not respond to NSAIDs, the GP may consider colchicine.

Admit patients with *other causes of pericarditis* for investigation and management. Dressler's syndrome (autoimmune pericarditis ± effusion 2–14 weeks after 3% of MIs) requires cardiology specialist care.

Pericardial effusion may occur with any type of pericarditis. It is relatively common in acute bacterial, tuberculous, and malignant pericarditis. Acute tamponade may occur following cardiac rupture with MI, aortic dissection, or after cardiac surgery. Summon senior help and arrange immediate bedside echocardiography for patients with signs of tamponade, with pericardiocentesis under USS guidance, and then a definitive drainage procedure.

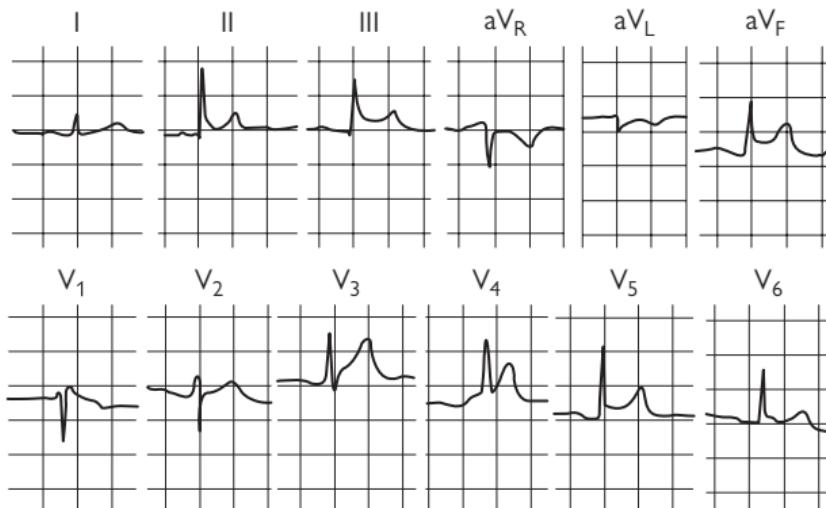


Fig. 3.10 ECG of pericarditis.

Bradyarrhythmias

Bradycardia is a ventricular rate of <60/min in the adult. It usually reflects influences on, or disease of, the SA node, or atrioventricular (AV) block. Intraventricular conduction disturbances may progress to AV block. Sinus bradycardia may be physiological (eg athletes), due to drugs (β -blockers), or pathological (hypothyroidism, hypothermia, hypoxia, ↑ ICP, sick sinus syndrome, MI (see Fig. 3.11), myocardial ischaemia). Bradycardia also occurs in up to one-third of patients with hypovolaemia (eg GI bleed, ectopic pregnancy).

Sick sinus syndrome ('sinus node disease')

Is usually the result of ischaemia or degeneration of the SA node. It is characterized by sinus pauses (>2s) or sinus arrest. Junctional or other escape beats may occur, and occasionally a tachyarrhythmia may emerge ('tachy-brady' syndrome). Patients may present with dizziness, collapse, loss of consciousness, or palpitations. A continuous 24-hr ECG tape may demonstrate arrhythmias.

AV block

Causes IHD, drugs (eg excess digoxin), or cardiac surgery.

First-degree AV block Conduction from atria to ventricles occurs every time but is delayed. The PR interval is >0.2s (five small squares on standard ECG) (see Fig. 3.12).

Second-degree AV block Only a proportion of P waves are conducted to the ventricles. There are two types:

- **Mobitz type I block (Wenckebach):** the PR interval becomes increasingly lengthened until a P wave fails to conduct (see Fig. 3.13).
- **Mobitz type II block:** failure to conduct P waves may occur regularly (eg 3:1) or irregularly, but the PR interval remains constant (see Fig. 3.14).

Third-degree (complete) heart block Atrial activity is not conducted to the ventricles. With a proximal block (eg at the AV node), a proximal escape pacemaker in the AV node or bundle of His may take over, producing narrow QRS complexes at a rate of ~50/min. With distal AV block, a more distal escape pacemaker results in broad, bizarre complexes at a rate of ~30/min. Ventricular asystole may occur if the escape pacemaker stops discharging, unless a subsidiary pacemaker takes over (see Fig. 3.15).

Intraventricular conduction disturbances

The intraventricular conducting system commences as the bundle of His and divides into right and left bundle branches—the latter subdivides further into antero-superior and postero-superior divisions. These two divisions and the right bundle branch are referred to as the 'fascicles'. Blockage of two out of three fascicles = *bifascicular block*.

- RBBB + left anterior hemiblock (blockage of the left antero-superior fascicle) causes LAD and an RBBB pattern on ECG.
- RBBB + left posterior hemiblock causes RAD and an RBBB pattern on ECG.

Bifascicular block accompanied by a prolonged PR interval is often referred to as *trifascicular block*. Note that true blockage of all three fascicles would cause complete heart block, so bifascicular block with prolonged PR interval represents impending progression to complete heart block.

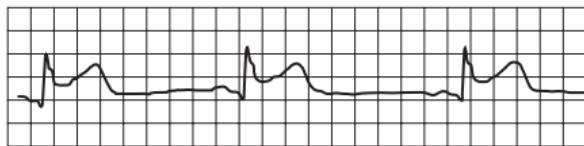


Fig. 3.11 ECG of sinus bradycardia with STEMI.



Fig. 3.12 ECG of first-degree heart block.

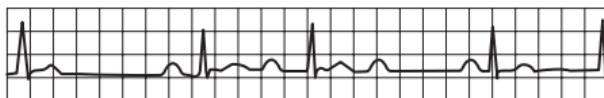


Fig. 3.13 ECG of Mobitz type I AV block.

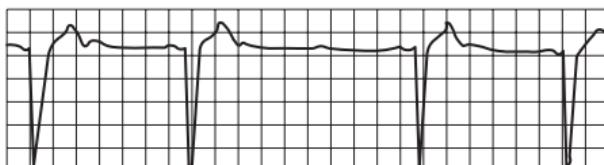


Fig. 3.14 ECG of Mobitz type II AV block.

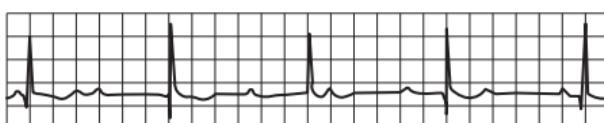


Fig. 3.15 ECG of complete AV block.

Treatment of bradyarrhythmias

The emergency treatment of bradycardia depends upon two important factors: the clinical condition of the patient and the risk of asystole. Give O₂ if hypoxic; insert an IV cannula, and follow the Resuscitation Council guidelines shown in Fig. 3.17 (🔗 <https://www.resus.org.uk>).

Atropine is the first-line drug. The standard dose is 500mcg IV, which may be repeated to a total of 3mg. Further doses are not effective and may result in toxic effects (eg psychosis, urinary retention).

Adrenaline (epinephrine) can be used as a temporizing measure prior to transvenous pacing if an external pacemaker is not available. Give by controlled infusion at 2–10mcg/min, titrating up according to response (adrenaline 6mg in 500mL of 0.9% saline infused at 10–50mL/hr). A (traditional) alternative to using an IVI of adrenaline is to use an isoprenaline IVI.

External transcutaneous pacing allows a pacing current to be passed between adhesive electrodes placed over the front of the chest and the back. Select external demand pacing mode at a rate of 70/min, then gradually ↑ the pacing current from zero until capture is shown on the monitor. Clinically, capture results in a palpable peripheral pulse at the paced rate and clinical improvement. Provide small doses of IV opioid ± sedation as needed.

Transvenous cardiac pacing is the treatment of choice for bradycardic patients who are at risk of asystole. The technique should only be performed by an experienced doctor. The preferred route of access is the internal jugular or subclavian vein. However, if thrombolysis has recently been given or is contemplated, or if the patient is taking anticoagulants, use the right femoral vein instead. Obtain a CXR to exclude complications. A correctly functioning ventricular pacemaker results in a pacing spike followed by a widened and bizarre QRS (see Fig. 3.16).

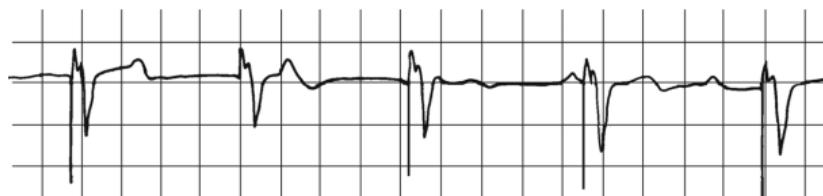


Fig. 3.16 Paced rhythm.

Algorithm for the management of bradycardia

(See Fig. 3.17; see also  <https://www.resus.org.uk>)

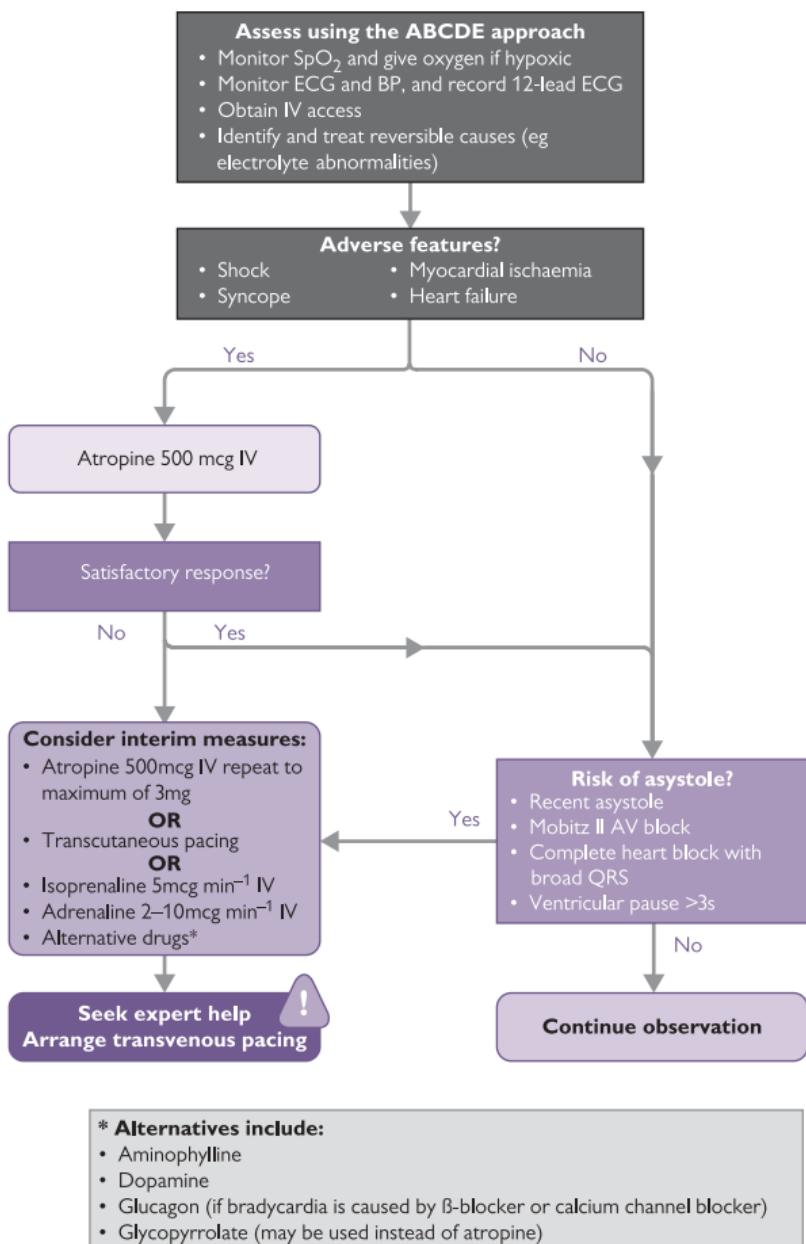
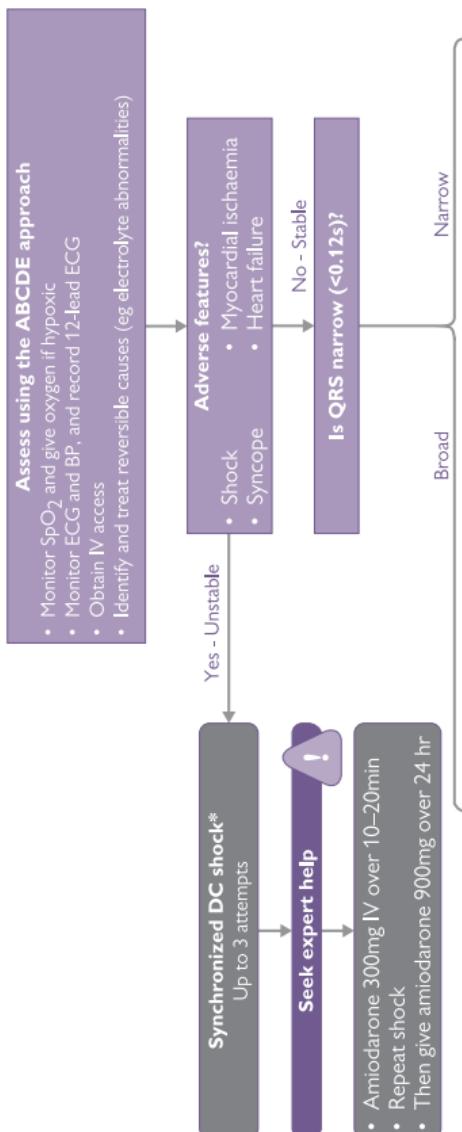


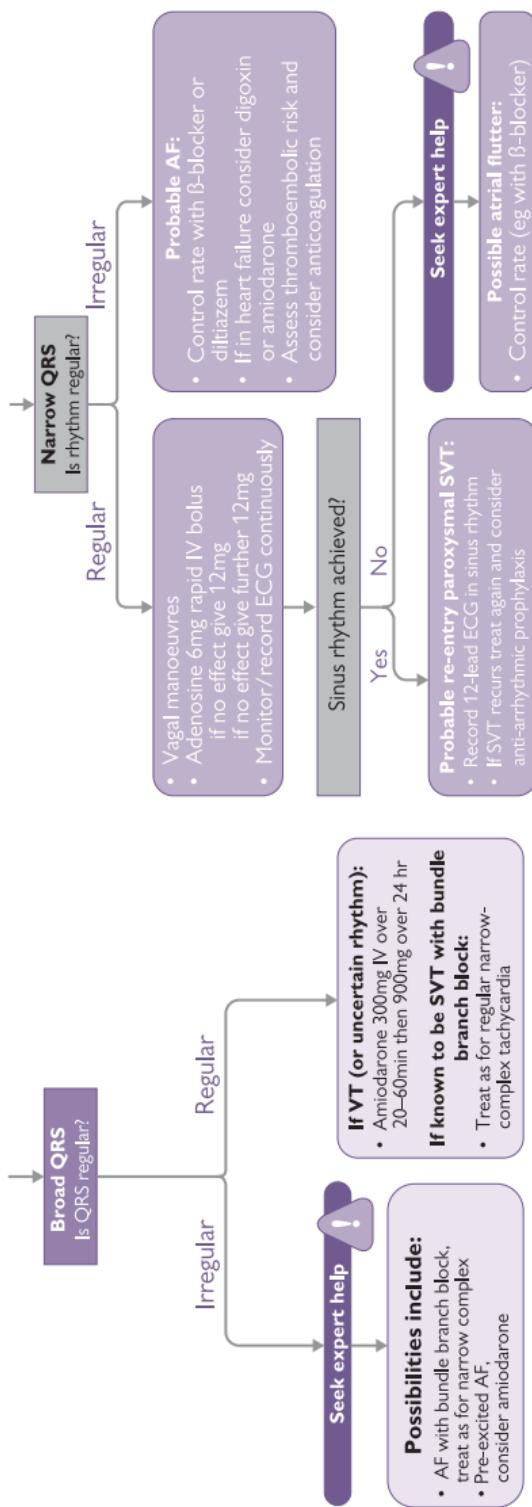
Fig. 3.17 Algorithm for the management of bradycardia, 2015.

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Tachycardia algorithm—with pulse

(See Fig. 3.18.)





*Conscious patients require sedation or general anaesthesia for cardioversion

Fig. 3.18 Tachycardia algorithm with pulse, 2015.

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Tachyarrhythmias

The single Resuscitation Council 2015 tachycardia algorithm (see Fig. 3.18) (see  <https://www.resus.org.uk>) is based on the fact that, irrespective of the exact underlying cardiac rhythm, many of the initial management principles in the peri-arrest setting are the same:

- Rapidly assess Airway, Breathing, and Circulation.
- Monitor cardiac rhythm and record a 12-lead ECG.
- Provide O₂ if hypoxic.
- Identify and treat reversible causes.
- Assess for adverse features (signs of shock, syncope, signs of heart failure, or myocardial ischaemia)—these indicate the need for urgent intervention, initially in the form of synchronized cardioversion.

The unstable patient with tachyarrhythmia

Synchronized cardioversion

This requires two doctors—one to perform cardioversion, the other (experienced in anaesthesia) to provide sedation/anaesthesia and manage the airway. The patient will not be fasted and is therefore at particular risk of aspiration. The arrhythmia will almost certainly ↓ cardiac output and ↑ circulation times, so IV drugs take much longer to work than usual. If the ‘sedation doctor’ does not appreciate this and gives additional doses of anaesthetic drugs, hypotension and prolonged anaesthesia may result.

Synchronize electrical cardioversion so that it occurs with the R wave to minimize the risk of inducing VF. Synchronized cardioversion is effective in treating patients who exhibit evidence of instability with underlying rhythms of supraventricular tachycardia (SVT), atrial flutter, AF, and VT—choose an initial level of energy according to the rhythm and defibrillator:

- For broad complex tachycardia or AF, start with 120–150J (biphasic). If unsuccessful, ↑ in increments.
- Start with a lower energy level for atrial flutter and paroxysmal SVT—use 70–120J (biphasic). If this is unsuccessful, ↑ in increments to 150J.

Amiodarone

If cardioversion is unsuccessful after three synchronized shocks, give amiodarone IV 300mg over 10–20min and repeat the shock. Give amiodarone by central vein, when possible, as it causes thrombophlebitis when given peripherally. However, in an emergency, it is acceptable to use a large peripheral vein.

Clinically stable patient with tachyarrhythmia

Tailor treatment according to the likely underlying rhythm. Establish if the QRS is broad or narrow (<0.12s) and if the rhythm is regular or not, then treat as outlined in Fig. 3.18.

Broad complex tachyarrhythmias

May be caused by VT (see Fig. 3.19) or, rarely, by SVT with aberrant conduction. The default position should be that broad complex tachycardia is VT. Provide O₂ as appropriate; insert an IV cannula, and follow the Resuscitation Council guidelines (see Fig. 3.18 and <https://www.resus.org.uk>).

The priorities in broad complex arrhythmias associated with tricyclic antidepressant overdose are airway management, oxygenation, ventilation, and correction of metabolic disorders—give IV bicarbonate, but avoid anti-arrhythmic drugs (see  Tricyclic antidepressant poisoning, pp. 202–3).

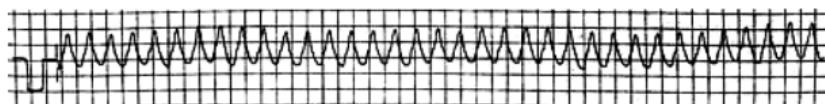


Fig. 3.19 ECG of VT.

Evaluating ECGs: VT or SVT with aberrant conduction?

VT is much more likely as a cause of broad complex tachycardia if:

- The patient is >60y.
- The patient has a history of IHD or cardiomyopathy.
- There is clinical evidence of AV dissociation (intermittent cannon 'a' waves seen on JVP, first heart sound of variable intensity).
- Inverted P waves in lead II.
- The frontal plane axis is bizarre (−90° to −180°).
- The QRS is >0.13s.
- There are 'capture' or 'fusion' beats.
- The QRS is bizarre, not resembling a bundle branch block pattern.
- All chest leads (V_{1–6}) concordant (QRS complexes point the same way).
- R > R' (or r') in V₁.
- There is a deep S wave (QS, rS, or RS) in V₆.

Torsades de pointes

Rare form of polymorphic VT, associated with hypomagnesaemia, hypokalaemia, long QT interval (congenital or drug-related, eg sotalol, antipsychotics, antihistamines, antidepressants). A constantly changing electrical axis results in QRS complexes of undulating amplitude (see Fig. 3.20). Usually paroxysmal, it may degenerate to VF. Stop drugs that might prolong QT and avoid amiodarone. Correct electrolyte abnormalities. Get expert help and treat with IV magnesium sulfate (2g over 10min = 8mmol or 4mL of 50% magnesium). Refractory cases may require overdrive pacing. Arrange synchronized cardioversion if adverse features.

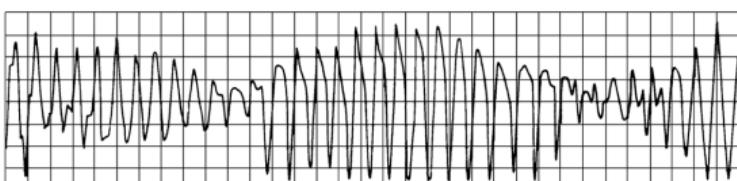


Fig. 3.20 ECG of torsades de pointes.

Regular narrow complex tachyarrhythmias

Most patients presenting with regular narrow complex tachycardia (see Fig. 3.21) that is not sinus tachycardia have paroxysmal SVT which responds to vagal manoeuvres or adenosine. If the ventricular rate is exactly 150/min, atrial flutter with 2:1 block is likely. Note: vagal manoeuvres/adenosine may temporarily slow the heart rate and reveal the rhythm.

Give O₂ if hypoxic; insert an IV cannula, and follow the algorithm in Fig. 3.18 (see also <https://www.resus.org.uk>).

Unstable patients

Treat the compromised patient (shock, syncope, acute cardiac failure, or cardiac ischaemia) with emergency electrical cardioversion. Consider vagal stimulation and/or giving IV adenosine whilst arranging the cardioversion, as long as this does not delay the procedure.

Stable patients

- Try *vagal stimulation*. The most effective way is a modified Valsalva manoeuvre. Whilst semi-recumbent, instruct the patient to attempt to blow the plunger out of a 20mL syringe for 15s, then lie the patient supine and manually raise the legs for 15s.
- *Carotid sinus massage* of the carotid sinus for 15s (one side only), by gently rubbing in a circular action lateral to the upper border of the thyroid cartilage, is used less frequently now. It may be dangerous (especially if there is a carotid bruit or risk of stroke/TIA).
- Adenosine temporarily blocks conduction through the AV node. It has a very short half-life (10–15s) and can successfully terminate re-entrant tachycardias and may ‘unmask’ other conditions (eg atrial flutter) by temporarily producing a conduction block. It is contraindicated in second- or third-degree AV block, severe hypotension, and patients with asthma. The effects are blocked by theophylline and potentiated markedly (and dangerously) in the presence of dipyridamole or carbamazepine or in a denervated heart—seek advice. Warn the patient about transient flushing and chest discomfort. Give adenosine by fast IV bolus 6mg into an IV cannula in the antecubital fossa and flush with 0.9% saline (see Fig. 3.18) whilst recording a rhythm strip. If unsuccessful, repeat with 12mg (then use 12mg again, if needed).
- If adenosine is contraindicated, consider IV *verapamil* 2.5–5mg over 2min. Avoid verapamil in patients with cardiac failure, hypotension, concomitant β-blocker therapy, or WPW.



Fig. 3.21 Narrow complex tachycardia.

Atrial fibrillation

Most patients with a fast, irregular pulse are in AF. This is characterized by rapid, irregular, unco-ordinated atrial activity, associated with an irregular ventricular response. It is a common condition with numerous causes.

Causes

Acute AF may be associated with: IHD (33%), heart failure (24%), hypertension (26%), and valvular heart disease (7%). Other cardiac causes are sick sinus syndrome, pericarditis, infiltrative heart disease, cardiomyopathy, myocarditis, congenital heart disease, and post-cardiac surgery.

Non-cardiac causes include: sepsis, PE, thyrotoxicosis, electrocution, lung or pleural disease, chest trauma, hypokalaemia, hypovolaemia, hypothermia, drug abuse (eg cocaine), and 'holiday heart syndrome'. Paroxysmal AF sometimes occurs in fit athletes.

Clinical features

Some patients may present to the ED with palpitations and other symptoms as a result of suddenly developing AF; some may develop AF as part of a serious acute illness (eg sepsis), whilst in others it may be an apparently incidental finding. AF ↓ cardiac output by 10–20%, irrespective of the underlying ventricular rate.

Clinical presentation varies according to the cause and effect of AF. Some patients are asymptomatic; others suffer life-threatening complications (heart failure, angina). Patients with underlying IHD may develop ischaemia during periods of rapid ventricular rate.

Treatment

Patients in AF can be treated with rate or rhythm control. Rhythm control can be achieved by either chemical or electrical cardioversion.

If signs of shock, syncope, acute cardiac failure, or ischaemia, consider electrical cardioversion under sedation (as shown in Fig. 3.18). Patients may be chemically cardioverted with flecainide 50–150mg IV or 300mg PO (contraindicated in patients with cardiac disease) or amiodarone 300mg IV (safer in patients with cardiac disease). Both drugs may cause hypotension.

If the patient has had symptoms for longer than 48hr, he/she is at risk of cardiac thromboembolism and stroke when cardioverted, so instead, give rate control medications and commence oral anticoagulant or LMWH. Rate control drugs include metoprolol 5mg IV and diltiazem (IV form not available in the UK). Digoxin 500mcg IV is the drug of choice in patients with congestive cardiac failure. (See NICE guidelines at <https://www.nice.org.uk>)

AF ↑ the risk of stroke. This risk can be quantified by applying the CHADS₂VA_{SC} score (see <https://www.mdcalc.com>)—any patient scoring >1 should be considered for anticoagulation (eg apixaban, rivaroxaban, edoxaban, or dabigatran). The decision on whether and what treatment to commence will be informed by current renal function, previous bleeding events, and the need to minimize co-prescription of antiplatelet drugs. Local policy will determine if this will occur in the ED or in early GP follow-up.

Hypertensive problems

- Most patients with hypertension are asymptomatic.
- Hypertension is an important risk factor for cardiovascular disease and stroke.
- Most patients found to be hypertensive in the ED do not require any immediate intervention or treatment, but do require careful follow-up—usually by their GP.
- Never intervene on the basis of a single raised BP measurement in the absence of any associated symptoms and signs.

Hypertensive emergency

↑ BP with rapid-onset neurological signs, retinopathy, myocardial ischaemia, or renal failure. BP is often $>230/130\text{mmHg}$. Search for evidence of hypertensive encephalopathy: headache, nausea, vomiting, confusion, retinal changes (haemorrhages, exudates, papilloedema), fits, focal neurological signs, ↓ conscious level. Check for symptoms of aortic dissection. Consider recent drug ingestion (eg ecstasy or cocaine).

Investigations

Insert an IV cannula and send blood for U&E, creatinine, and glucose. Obtain a CXR and an ECG, and perform urinalysis. If there is ↓ conscious level, focal signs, or other clinical suspicion that the hypertension may be secondary to stroke or intracranial haemorrhage, arrange an emergency CT scan. If there is concern for aortic dissection, request a CT angiogram.

Management

In a true hypertensive emergency (eg encephalopathy, aortic dissection, or intracranial haemorrhage), aim to reduce BP by no more than 25% in the first hour. If treatment is appropriate, commence an IV of sodium nitroprusside, labetalol, or GTN, with continuous BP monitoring via an arterial line and admit to HDU or ICU. Sodium nitroprusside has a very short half-life (~1–2min) and acts as a vasodilator of both arterioles and veins. IV labetalol may be preferred if aortic dissection (see  Aortic dissection, p. 97) or phaeochromocytoma are suspected.

β -blockers are contraindicated in hypertension caused by cocaine, amphetamine, or related sympathomimetic drugs (see  Recreational drugs, pp. 222–3), since β -blockade may cause unopposed α -adrenergic activity with paradoxical hypertension and ↓ coronary blood flow.

Hypertension in pregnancy

Hypertension may be part of pre-eclampsia or eclampsia (see  Medical complications of pregnancy, p. 606). Pre-eclampsia is diagnosed with two or more of: hypertension ($>140/90\text{mmHg}$), proteinuria, and oedema. This can be associated with haemolysis, elevated LFTs, low platelets (HELLP syndrome). Check urine for protein, and check blood for FBC, LFTs, platelets, uric acid level, and coagulation screen. Call for senior obstetric help. Eclampsia is diagnosed with the onset of grand mal seizures after 20 weeks' gestation and carries a significant mortality rate.

Implantable cardiac devices

Pacemaker letter codes

Enable pacemaker identification:

- First letter: chamber paced (A = atria; V = ventricles; D = dual chamber).
- Second letter: chamber sensed (A = atria; V = ventricles; D = dual; O = none).
- Third: pacemaker response (T = triggered; I = inhibited; D = dual; R = reverse).
- Fourth (P = programmable; M = multi-programmable).
- Fifth (P = the pacemaker will pace in tachycardia; S = the pacemaker shocks in tachycardia; D = dual ability to pace and shock; O = none of these).

Types of pacemaker malfunction

Failure to capture Pacing spikes, but no QRS (battery or lead problem, exit block caused by infarcted muscle or electrolyte abnormality).

Undersensing Inappropriate pacing spikes (lead displacement, low-voltage native QRS complexes).

Oversensing Too few pacing spikes (picking up T waves or extracardiac potentials, mobile phones).

Abnormal rates Low battery, conduction from ventricles to atria.

Types of defibrillator problems

Increased shocks Oversensing of T waves causing ↑ shocks. Sensing of extracardiac potentials causing ↑ shocks. External transcutaneous pacing will provide temporary support whilst the problem is resolved. A special magnet may be needed to inactivate an implantable defibrillator which fires repeatedly. Admit patients whose implantable defibrillator has fired.

VT/VF Sustained or recurrent VT/VF cardiac arrest from lead displacement, low battery, failure to respond to shock.

Infection and pocket haematoma Common early after insertion of both pacemaker and defibrillator.

Left ventricular assist devices

Placed either as a 'bridge' to cardiac transplant or as 'destination therapy' to prolong life in end-stage cardiac failure. They pump blood from LV into aorta; battery-dependent. Patient carries backup battery and charger (as a backpack or belt). Future LVADs may have internal implantable batteries. Battery may last 4–6hr and will alarm when low. LVAD patients are anticoagulated with warfarin. They may not have a palpable pulse. Doppler or arterial line will measure BP. Do not give chest compressions in cardiac arrest because of risk of displacement. The device may have a hand pump for use in cardiac arrest.

Problems with these devices are most common soon after insertion:

- Battery depletion: check that the light is on. Ensure the device is plugged in and backup battery is to hand.
- Bleeding: intracranial, pulmonary, GI, and other. Do not reverse anticoagulation without seeking expert advice, as it carries a high risk of LVAD thrombosis (and patient demise).
- Clotting: embolic stroke, ischaemic limb or gut, LVAD thrombosis causing device failure. Machine may feel hot to touch in LVAD thrombosis.
- Infection: treat for severe sepsis.

Aortic dissection

► Remember: patients (especially those with hypertension) with sudden severe chest and/or back pain may have acute aortic dissection.

Pathology

Aortic dissection is longitudinal splitting of the muscular aortic media by a column of blood. The dissection may spread proximally (possibly resulting in aortic incompetence, coronary artery blockage, cardiac tamponade) or distally (possibly involving the origin of various arteries), or rupture internally back into the aortic lumen or externally (eg into the mediastinum, resulting in rapid exsanguination).

More than 70% of patients have a history of hypertension. It occurs more frequently in those with a bicuspid aortic valve, Marfan's syndrome, or Ehlers–Danlos syndrome. Up to 20% follow recent cardiac surgery or recent angiography/angioplasty.

Dissection may be classified as Stanford type 'A' or 'B', according to whether the ascending aorta is involved or not, respectively (see Fig. 3.22).

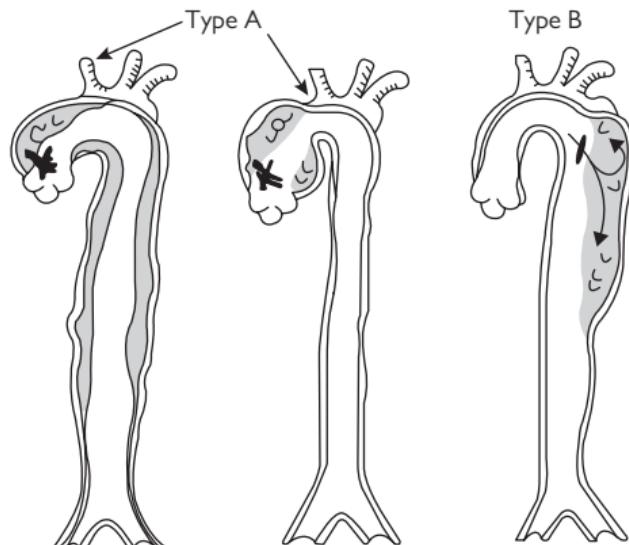


Fig. 3.22 Stanford classification of aortic dissection.

History

Aortic dissection may mimic an MI, so adopt a high index of suspicion. It typically presents with abrupt-onset sharp, tearing, or ripping pain (maximal at onset) in the anterior or posterior chest. The pain can resolve, then recur in the epigastrium or elsewhere. Pain migration may reflect dissection extension. Sometimes the patient is pain-free after the initial insult. Syncope occurs in ~10%, sometimes without any pain. Occasionally, patients present with an acute stroke, with neurological deficit plus chest pain. Involvement of the coeliac artery can cause bowel ischaemia. Likewise, involvement of the renal arteries can cause acute kidney injury (AKI).

Examination

The patient is usually apprehensive and distressed, with pain which is difficult to alleviate, even with using IV opioid. Clues to the diagnosis include:

- An aortic regurgitation murmur (30%).
- Asymmetry or absence of peripheral pulses or a pulse deficit.
- Hypertension.
- Hypotension with features of tamponade or neurological signs in association with pain (eg secondary to spinal/carotid artery involvement).

Investigations

Send blood for U&E, glucose, FBC, coagulation, and cross-matching. Obtain an ECG and a CXR. Thoracic aortic dissection usually results in an abnormal CXR. One or more of the following changes may be seen:

- A widened or abnormal mediastinum (present in ~75%).
- Left pleural effusion (~20%).
- Deviation of the trachea or nasogastric (NG) tube to the right.
- A 'double-knuckle' aorta and/or separation of the two parts of the wall of a calcified aorta by >5mm (the 'calcium sign').

The ECG may show MI, LVH, or ischaemia. Note: ~12% of patients with aortic dissection have a normal CXR and ~30% have a normal ECG. CT angiography will provide the definitive diagnosis (see Fig. 3.23).

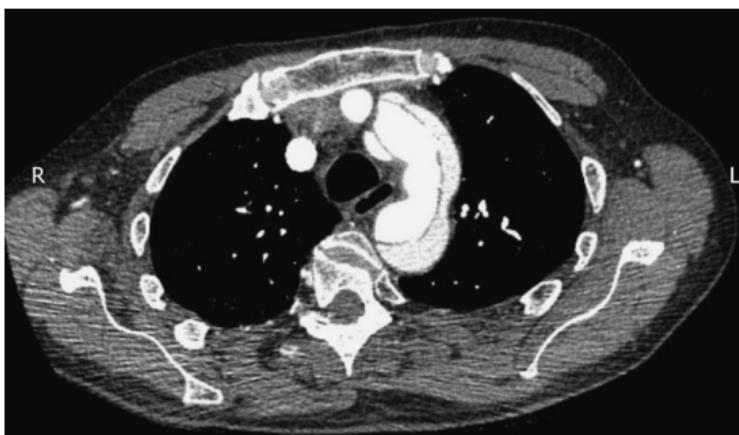


Fig. 3.23 CT scan showing dissection of the aortic arch.

Management

On suspicion of an aortic dissection:

- Provide O₂ by face mask as appropriate (see Oxygen, p. 99).
- Insert two large-bore (14G) IV cannulae and cross-match for 6U.
- Give IV morphine and titrate according to response (\pm antiemetic).
- Call the cardiothoracic team and the cardiologist at an early stage.
- Insert an arterial line (preferably right radial artery), and discuss with specialist teams how to control the BP (eg labetalol infusion).
- Arrange further investigation based upon specialist advice and available resources (eg aortography, echocardiography, CT scan, MRI).

Type A dissections are usually treated surgically; type B lesions are usually treated medically.

Haemoptysis

Haemoptysis may be the chief or sole complaint of patients presenting to the ED. It always warrants investigation. For causes of haemoptysis, see Table 3.5.

Table 3.5 Causes of haemoptysis

Respiratory	<ul style="list-style-type: none"> ● Carcinoma (bronchial or laryngeal) ● Infection (upper respiratory tract infection, pneumonia, TB, lung abscess) ● Bronchiectasis
Cardiovascular	<ul style="list-style-type: none"> ● Pulmonary oedema ● PE ● Ruptured aortic aneurysm (aorto-bronchial fistula)
Coagulation disorder	<ul style="list-style-type: none"> ● Drugs (eg warfarin, rivaroxaban, apixaban, dabigatran) ● Inherited (eg haemophilia)
Trauma	<ul style="list-style-type: none"> ● Penetrating or blunt
Other	<ul style="list-style-type: none"> ● Goodpasture's, granulomatosis with polyangiitis

Presentation

Ascertain the exact nature and volume (eg 'bright red streaks' or 'dark brown granules'). Patients sometimes have surprising difficulty distinguishing vomited blood from that coughed up. Enquire about weight loss, and take a drug history and a smoking history.

Investigations

- Send blood for FBC, coagulation screen, U&E, and LFTs.
- Request group and save if evidence of significant haemorrhage.
- If $\text{SpO}_2 < 90\%$ on air or the patient has COPD, check ABG.
- Obtain CXR and ECG. Request CT if lung cancer suspected.
- Perform urinalysis—if shocked, insert a catheter and monitor output.
- Collect sputum samples. Send for microscopy, culture, and sensitivity.
- Initiate further investigations according to the likely diagnosis.

Treatment

- Airway: clear and secure (coughing/suction). Put on a face mask and shield if maintaining the airway or intubating. Ensure nearby high-flow suction. Massive haemorrhage may require tracheal intubation. Whilst preparing for this, tilt the trolley so that the patient is head down.
- Breathing: provide O_2 to maintain SpO_2 at 90–94%. If ventilation is inadequate, assist with bag and mask or tracheal tube.
- Circulation: insert a large-bore (14G) IV cannula (use two if hypovolaemic). Give IV fluids/blood/clotting factors, as clinically indicated (see  Blood transfusion overview, pp. 180–1).

Further treatment Commence specific treatment measures aimed at the life-threatening underlying cause (eg LVF, PE, infection, coagulopathy). In cases of large haemoptysis, admit for further investigation and treatment. If the patient is stable and has only had a small amount of bloodstained sputum, urgent outpatient investigation may be appropriate.

Oxygen

O_2 is the most commonly administered hospital therapy. O_2 therapy is controlled and targeted, as it has been recognized that giving too much O_2 can ↑ mortality for medical patients in hospital.

Oxygen requirements

Patients with high oxygen requirements

A small number of patients with certain specific conditions may benefit from the provision of high-flow O_2 , with the target of SpO_2 approaching 100%, including:

- Carbon monoxide (CO) poisoning (see ↗ Carbon monoxide poisoning, p. 216).
- Cluster headaches (see ↗ Cluster headache, p. 138).
- Sickle-cell crisis (see ↗ Sickle-cell disease, pp. 184–5).
- Pneumothorax (see ↗ Spontaneous pneumothorax, pp. 118–20).

Most patients with acute medical illness

The aim of O_2 therapy is to optimize tissue O_2 delivery. Use pulse oximetry to guide whether the patient requires supplemental O_2 . In most previously healthy patients with acute medical illness who require O_2 , aim for SpO_2 of ≤96%, with a target range of SpO_2 of 90–94%.

Patients with COPD

In patients with known COPD or type II respiratory failure, aim for SpO_2 of 88–92%. Take an ABG in patients with chronic lung disease, to assess their optimal O_2 treatment (see ↗ Arterial blood gases, p. 102 on ABG interpretation). Repeat the ABG within 30min after changing the inspired O_2 concentration (FiO_2).

Prescribing oxygen

Include the target SpO_2 , the O_2 mask type, and the O_2 flow rate in the O_2 prescription. In an emergency, it is appropriate to administer O_2 prior to prescribing, but do not forget to prescribe the O_2 after resuscitation. (See ↗ <https://www.brit-thoracic.org.uk>)

Oxygen cylinders

When administering O_2 in the ED, always use piped O_2 from the wall outlet. Only use an O_2 cylinder when transporting the patient to the radiology department or ward. O_2 is highly flammable. Do not take a cylinder out of its support cage. In the UK, O_2 cylinders are colour-coded white. The most common small cylinder is B (or M-6), which holds 170L of O_2 . The most common large cylinder is E (or M-24), holding 680L of O_2 . Before a patient leaves the ED, check that the cylinder is full. If the patient is being transferred to another hospital, ensure there is enough O_2 for the journey. The formula is:

$$\text{Volume of cylinder in L} / \text{flow rate} = \text{how long cylinder will last in minutes}$$

The dyspnoeic patient

The normal adult RR is 11–18/min, with a tidal volume of 400–800mL. Acute dyspnoea is a common presenting symptom.

Common causes of acute dyspnoea

Cardiac

- Cardiogenic pulmonary oedema (see Cardiogenic pulmonary oedema, pp. 104–5).
- MI (see Acute coronary syndromes, pp. 72–3).
- PE (see Pulmonary embolism, pp. 124–5).
- Arrhythmias (see Bradyarrhythmias, p. 84).

Respiratory

- Asthma (see Acute asthma: assessment, pp. 108–9) or exacerbation of COPD (see Chronic obstructive pulmonary disease, pp. 112–13).
- Pneumonia (see Pneumonia, pp. 114–15).
- Pleural effusion (see Pleural effusion, p. 107).
- Pneumothorax (see Spontaneous pneumothorax, pp. 118–20).

Trauma

- Aspiration of FB or vomit (see Pulmonary aspiration, pp. 116–17).
- Pneumothorax/haemothorax (see Traumatic pneumothorax, pp. 344).
- Flail chest (see Flail segment, p. 342).
- Drowning incident (see Drowning or near drowning, pp. 268–9).

Other

- Hypovolaemia or fever from any cause.
- Hyperventilation syndrome (see Hyperventilation, p. 101).
- Respiratory compensation for metabolic acidosis (diabetic ketoacidosis (DKA), salicylate overdose).

Approach

Follow the ABC approach and resuscitate as necessary. The main aim of treatment is to correct life-threatening hypoxia. Enquire about the speed of onset of dyspnoea, past medical history, and associated symptoms (cough, haemoptysis, fever, wheezing, chest pain). Examine carefully, paying attention to the RR, depth, and pattern. Apply a pulse oximeter.

Pulse oximetry

Simple, rapid, safe, and non-invasive, but it does *not* provide information about ventilation or arterial partial pressure of CO₂ (pCO₂). A normal SpO₂ does not exclude significant lung pathology (eg PE). Pulse oximetry may be inaccurate or misleading in:

- Poor peripheral perfusion/shock and hypothermia.
- Methaemoglobinæmia.
- CO poisoning (see Carbon monoxide poisoning, p. 216). SpO₂ values may be falsely high as COHb reads as oxyhaemoglobin. COHb can be measured on VBG testing or with a COHb pulse oximeter.
- Nail varnish/synthetic fingernails (if a finger probe is used).
- Excessive movement.

Correlate readings with clinical findings—a non-pulsatile trace (or a heart rate different from that on the cardiac monitor) suggests the saturation reading is probably inaccurate.

Hyperventilation

Hyperventilation is breathing which occurs more deeply and/or more rapidly than normal. CO_2 is 'blown off', so that $\text{pCO}_2 \downarrow$. Hyperventilation may be primary ('psychogenic') or secondary. A classical secondary cause is DKA—Kussmaul's respiration represents respiratory compensation for metabolic acidosis.

Secondary causes of hyperventilation

- Metabolic acidosis (eg DKA, uraemia, sepsis, hepatic failure).
- Poisoning (eg aspirin, methanol, CO, cyanide, ethylene glycol).
- Pain/hypoxia.
- Hypovolaemia.
- Respiratory disorders (eg PE, asthma, pneumothorax).

Primary (psychogenic or inappropriate) hyperventilation

Typically, the patient is agitated and distressed, with a past history of panic attacks or episodes of hyperventilation. They may complain of dizziness, circumoral paraesthesiae, carpopedal spasm, and occasionally sharp or stabbing chest pain. Initial examination reveals tachypnoea, with equal air entry over both lung fields, and no wheeze or evidence of airway obstruction. It is important to consider secondary causes (such as PE or DKA). Therefore, perform the following investigations:

- SpO_2 .
- ECG.
- ABG if $\text{SpO}_2 \downarrow$ or if symptoms do not completely settle in a few minutes.
- BMG.

If symptoms do not completely settle in a few minutes, obtain:

- CXR.
- U&E, blood glucose, FBC.

Treatment

Do not sedate a patient who is hyperventilating. Once serious diagnoses have been excluded, use this information to help reassure the patient with primary hyperventilation. Often this is all that is required, but it may be helpful to try simple breathing exercises (breathe in through the nose—count of 8, out through the mouth—count of 8, hold for count of 4, and repeat). Discharge the patient with arrangements for GP follow-up. If these simple measures fail, reconsider the diagnosis and refer the patient to the medical team for subsequent observation and treatment.

Arterial blood gases

Assessing respiratory function

Arterial sampling helps in the assessment of a patient with low SpO_2 or patients with known lung disease (especially if they are receiving supplemental O_2). Document the FiO_2 . Look specifically for:

- Hypoxia ($\text{pO}_2 < 10.6\text{kPa}$ on air).
- Hypercarbia ($\text{pCO}_2 > 6.0\text{kPa}$).
- Bicarbonate retention ($\text{HCO}_3^- > 28\text{mmol/L}$).
- Acidosis ($\text{pH} < 7.35$).

Differentiating between type I and type II respiratory failure

In type I failure, there is hypoxia with normal or $\downarrow \text{pCO}_2$. In type II failure, there is hypoxia with $\uparrow \text{pCO}_2$ and frequently $\uparrow \text{HCO}_3^-$. In type II failure, the patient may develop life-threatening respiratory failure if administered high concentrations of O_2 . Aim to maintain SpO_2 at 88–92% in COPD, and recheck ABGs in 30min.

Differentiating between acute and chronic type II respiratory failure

Patients who normally have a slightly $\uparrow \text{pCO}_2$ will also show $\uparrow \text{HCO}_3^-$ on ABG. The kidneys adapt over a period of days to retain HCO_3^- , in an attempt to buffer the respiratory acidosis (see  nomogram inside front cover). Respiratory acidosis in a patient with chronic type II respiratory failure ($\uparrow \text{pCO}_2$, $\uparrow \text{HCO}_3^-$, and $\text{pH} < 7.35$) indicates life-threatening impairment of lung function.

In acute respiratory failure, the lungs are unable to eliminate CO_2 (caused by $\downarrow \text{GCS}$ or hypoventilation from any cause), which results in $\uparrow \text{pCO}_2$ and respiratory acidosis. Patients may require ventilatory support.

Metabolic acidosis

The usual pattern of results in metabolic acidosis is $\text{pH} < 7.35$, $\text{HCO}_3^- < 24\text{mmol/L}$, and base excess (BE) $< -2\text{mmol/L}$. There may be compensatory hypocarbia ($\text{pCO}_2 < 4.5\text{kPa}$). Metabolic acidosis has many possible causes:

- \uparrow acid load (lactic acidosis, ketoacidosis, or ingestion of salicylates, methanol, ethylene glycol, or metformin).
- \downarrow removal of acid (renal failure or renal tubular acidosis types 1 and 4).
- Loss of HCO_3^- from the body (diarrhoea, pancreatic or intestinal fistulae, acetazolamide, or renal tubular acidosis type 2).

The anion gap

The anion gap is the quantity of anions not balanced out by cations (a measurement of negatively charged plasma proteins). The normal value is 12–16mmol/L. It is measured by (all measured in mmol/L):

$$(\text{Na}^+ + \text{K}^+) - (\text{Cl}^- + \text{HCO}_3^-)$$

Measuring the anion gap helps distinguish the cause of metabolic acidosis. A high anion gap indicates that there is excess H^+ in the body. The most common cause of a high anion gap metabolic acidosis is lactic acidosis. Most blood gas analysers measure lactate (normal $< 2.0\text{mmol/L}$).

Causes of lactic acidosis

- Tissue hypoperfusion (trauma with major haemorrhage, sepsis).
- Tissue hypoxia (hypoxaemia, CO or cyanide poisoning).
- Hepatic failure.
- Renal failure.
- Ethylene glycol or methanol poisoning (see \Rightarrow Ethylene glycol poisoning, p. 212 and \Rightarrow Methanol poisoning, p. 211).
- Cocaine or amphetamines (see \Rightarrow Recreational drugs, pp. 222–3).
- Salicylate poisoning (see \Rightarrow Salicylate poisoning, p. 197) or iron poisoning (see \Rightarrow Iron poisoning, p. 209).
- Biguanides (metformin).
- Isoniazid.
- Strenuous exercise.

The other causes of a high anion gap metabolic acidosis are *ketoacidosis* (diabetic or alcohol-induced) and renal failure.

Causes of a normal anion gap metabolic acidosis are chronic diarrhoea, pancreatic or intestinal fistulae, acetazolamide, and renal tubular acidosis.

The osmolal gap

This is the difference between the calculated serum osmolarity and the laboratory-measured serum osmolality. Serum osmolarity can be calculated by (all measured in mmol/L):

$$(2 \times \text{Na}^+) + \text{urea} + \text{glucose}$$

Subtract the calculated result from the laboratory-measured osmolality to give the osmolal gap. Normally this is <10mOsm/kg.

An elevated osmolal gap can be caused by alcohol, methanol, ethylene glycol or acetone ingestion, mannitol, or sorbitol.

Venous blood gases

Whilst not providing quite as much information as arterial samples, VBGs are incredibly useful in the initial work-up of many ‘trolley’ cases presenting to the ED. The availability and ease with which samples can be processed by modern analysers has meant that VBG analysis has become routine. The biggest advantage for the patient is that VBG analysis does not require a painful arterial puncture—it can be performed on a sample that was going to be taken for lab analysis, rather than done as an additional test.

A venous blood sample will give accurate readings for K^+ , lactate, glucose, HCO_3^- , haemoglobin (Hb), and COHb. In addition, a normal venous pCO_2 will exclude hypercarbia.

Venous lactate levels are useful in helping with the early identification of patients who are sicker than they initially appear to be, particularly those with sepsis.

Use serial VBG analyses to establish the response to treatment, especially in terms of lactate and K^+ .

Cardiogenic pulmonary oedema

Left heart failure results in ↑ LV end-diastolic pressure, causing ↑ pulmonary capillary hydrostatic pressure. Fluid collects in extravascular pulmonary tissues faster than the lymphatics clear it.

Causes of cardiogenic pulmonary oedema

Often an acute complication of MI and IHD, or an exacerbation of pre-existing cardiac disease (eg hypertension, aortic/mitral valve disease). Other causes are:

- Arrhythmias.
- Failure of prosthetic heart valve.
- Ventricular septal defect.
- Cardiomyopathy.
- Negatively inotropic drugs (eg β-blockers).
- Acute myocarditis.
- Left atrial myxoma (may cause syncope, fever, ↑ ESR)—very rare.
- Pericardial disease.

History Frequently dramatic. Dyspnoea and distress may prevent a full history from being taken. Find out the length of the history and whether there is any chest pain. Check current drug therapy/allergies, and establish what emergency prehospital treatment has been administered.

Examination Usually reveals a tachypnoeic, tachycardic, and anxious patient. If pulmonary oedema is severe, the patient may be cyanosed, coughing up frothy pink sputum and unable to talk. Check pulse and BP; auscultate the heart for murmurs and third/fourth heart sounds of gallop rhythm. Look for ↑ JVP (also a feature of PE and cardiac tamponade). Listen to the lung fields—fine inspiratory crepitations (crackles) may be limited to the bases or be widespread. Wheeze may be more prominent than crepitations. Cardiogenic pulmonary oedema is associated with evidence of ↓ cardiac output (sweaty, peripherally cool, and pale). Consider other diagnoses (eg sepsis) in patients with warm, flushed extremities.

Investigations

Commence treatment before completing investigations:

- Attach a cardiac monitor and check SpO₂ with a pulse oximeter.
- Obtain an ECG. Check for arrhythmias, LAD, LVH, LBBB, and recent or evolving MI.
- Send blood for U&E, glucose, FBC, troponin, and B-type natriuretic peptide (BNP).
- If severely ill or SpO₂ <90% on air, obtain an ABG.
- Obtain a CXR and look for features of cardiogenic pulmonary oedema:
 - Upper lobe diversion (distension of upper pulmonary veins).
 - Cardiomegaly (LV and/or left atrial dilatation).
 - Kerley A, B, or C septal lines (see Fig. 3.24).
 - Fluid in interlobar fissures.
 - Peribronchial/perivascular cuffing and micronodules.
 - Pleural effusions.
 - Bat's wing hilar shadows.
- Request old hospital notes/ECGs. In newly diagnosed heart failure, an urgent transthoracic echo will identify the presence or absence of cardiac abnormalities.

Treatment

- Check that the airway is clear.
- Raise the trolley to sit the patient up (support with pillows, if needed).
- Provide high-flow O₂, as required, by a tight-fitting face mask.
- Give furosemide IV 40mg. Note that larger doses may be needed in patients already taking oral furosemide.
- Nitrates and opioids are no longer recommended routinely (↗ <https://www.nice.org.uk>). Reserve the use of IV nitrates for specific circumstances (eg concomitant myocardial ischaemia, severe hypertension, or regurgitant aortic or mitral valve disease), starting IVI slowly (eg GTN IVI, starting at 10mcg/min), ↑ every few minutes according to clinical response; monitor BP closely—take special care to avoid hypotension. If the patient has chest pain, consider giving very small titrated increments of IV opioid (with antiemetic). Do not give opioids to patients who are drowsy, confused, or exhausted, as this may precipitate respiratory arrest.
- Consider NIV (continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP)) if the patient is very breathless with acidaemia and there is no immediate improvement.
- Monitor urine output (inserting a urinary catheter if necessary).
- Treat the underlying cause and associated problems (arrhythmias, MI, cardiogenic shock, acute prosthetic valve failure).

Monitor the SpO₂ and clinical response to initial treatment. Rapid improvement may occur, due to venodilatation and reduction of preload. If the patient does not improve, recheck the ABG and consider the following:

- If hypotensive, refer to ICU for treatment of cardiogenic shock (see ↗ STEMI treatment, pp. 80–1). An intra-arterial line, a Swan–Ganz catheter, and inotropic support (dobutamine) are likely to be required.
- Echocardiography may help to exclude valve or septal rupture and guide treatment.
- Rapid sequence intubation (RSI) in the presence of cardiogenic pulmonary oedema may be associated with cardiovascular collapse. Stop nitrates prior to administering anaesthesia and be ready to give pressors ± fluids immediately post-induction.

Prosthetic valve failure

Always consider valve failure in patients with prosthetic valves—a large variety are in common use. All are associated with some risks (eg embolism, failure, obstruction, infection, haemorrhage from associated anticoagulation), which vary according to the design. Acute failure of a prosthetic aortic or mitral valve results in dramatic acute-onset pulmonary oedema with loud murmurs. The patient may deteriorate rapidly and not respond to standard drug treatment. Resuscitate as described earlier. A CXR will show a prosthetic heart valve ± pulmonary oedema. Call urgently for expert help (ICU team, cardiologist, and cardiothoracic surgeon). Emergency transthoracic or transoesophageal echocardiography will confirm the diagnosis. Immediate valve replacement is required.

Non-cardiogenic pulmonary oedema

Pulmonary oedema may occur in the absence of ↑ pulmonary venous pressure. The following mechanisms may be responsible:

- ↑ capillary permeability.
- ↓ plasma oncotic pressure.
- ↑ lymphatic pressure.

Changes in capillary permeability, secondary to a variety of triggers, is the mechanism most frequently implicated in non-cardiogenic pulmonary oedema, when it occurs as adult respiratory distress syndrome (ARDS). Since the mechanisms producing cardiogenic and non-cardiogenic pulmonary oedema differ, so does the approach to treatment.

Causes of non-cardiogenic pulmonary oedema

- ARDS (sequel to sepsis, trauma, pancreatitis, COVID-19).
- Intracranial (especially subarachnoid) haemorrhage.
- IV fluid overload.
- Hypoalbuminaemia (liver failure, nephrotic syndrome).
- Drugs/poisons/chemical inhalation.
- Lymphangitis carcinomatosis.
- Smoke inhalation.
- Near drowning incidents.
- High altitude mountain sickness.

Approach

Distinguishing non-cardiogenic from cardiogenic pulmonary oedema is usually apparent from the history. Evaluate the patient and resuscitate according to ABCs. Direct treatment towards the underlying cause and according to the physiological disturbance. Use NIV early and consider urinary, intra-arterial, and central venous lines. Involve ICU early and provide appropriate IV fluids and inotropes—deterioration may require intubation, whilst being mindful of the risk of hypotension afterwards.

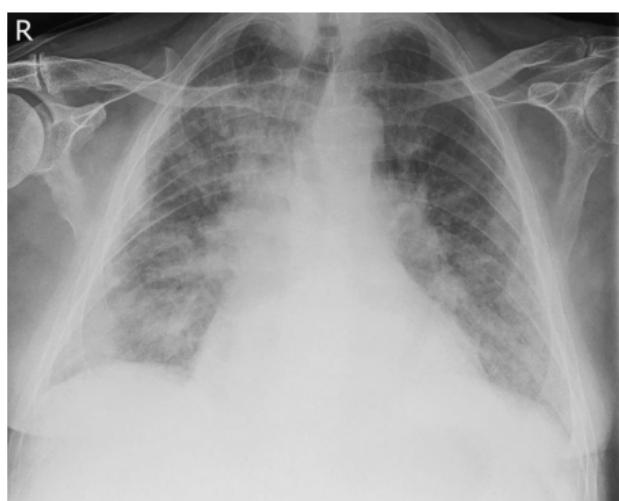


Fig. 3.24 CXR showing pulmonary oedema.

Pleural effusion

Under normal circumstances, each pleural cavity contains <20mL of fluid.

An exudate is diagnosed if the pleural fluid:serum protein is >0.5, fluid:serum lactate dehydrogenase (LDH) >0.6, or fluid LDH more than two-thirds the upper limits of the laboratory normal value for serum LDH.

Table 3.6 Causes of pleural effusion

Exudates	Transudates
Pneumonia	Cardiac failure
Malignancy	Nephrotic syndrome
TB	Hepatic failure
PE with pulmonary infarction	Ovarian hyperstimulation
Collagen vascular disease	Peritoneal dialysis
Abscess (subphrenic and amoebic liver)	Ovarian fibroma (Meig's syndrome)
Pancreatitis	
Chylothorax (thoracic duct injury)	

Clinical presentation

Symptoms are usually due to the underlying disease process. A mild dull ache and dyspnoea (initially on exercise, later at rest) may occur if the effusion is large. A history of vomiting, followed by chest pain, points to a ruptured oesophagus—a surgical emergency.

Signs are not apparent until >500mL are present. Dyspnoea and stony dullness to percussion, with absent breath sounds over the effusion, are characteristic. Bronchial breathing may be heard just above the effusion. Very large unilateral effusions may produce evidence of mediastinal shift.

Investigation

CXR can demonstrate pleural effusions as small as 250mL as blunting of the costophrenic angle (see Fig. 3.25). Other investigations depend on the likely cause. For causes of pleural effusion, see Table 3.6.

Treatment

Provide O₂ and resuscitate as necessary, according to the underlying pathology. Emergency therapeutic pleural aspiration is rarely required in the ED, except where haemothorax is suspected. Refer to the medical team for further investigation (including USS-guided diagnostic pleural aspiration).

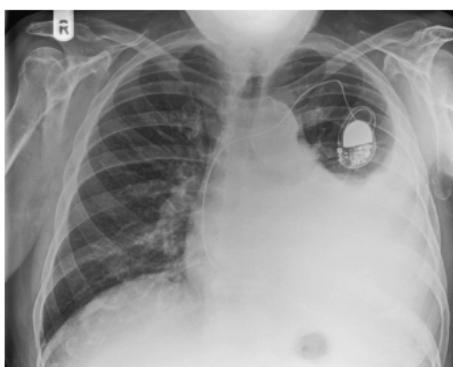


Fig. 3.25 CXR showing left pleural effusion and pacemaker.

Acute asthma: assessment

Follow the British Thoracic Society (BTS)/Scottish Intercollegiate Guidelines Network (SIGN) guidelines (<https://www.brit-thoracic.org.uk>) to assess and manage adults presenting with asthma (see  Acute asthma: management, pp. 110–11). The guidelines reflect continuing concern over asthma deaths. Patients with severe asthma and one or more adverse psychosocial factors (psychiatric illness, alcohol or drug abuse, unemployment) have ↑ mortality. Measure the peak expiratory flow rate (PEFR) and compare it against that expected (see Fig. 3.26). The peak flow acts as an immediate triage tool—remember that patients with life-threatening asthma may be too dyspnoeic to do this.

Make an initial assessment of the severity of acute asthma based upon a combination of clinical features, peak flow measurement, and pulse oximetry, as outlined below.

Moderate exacerbation of asthma

- ↑ symptoms.
- Peak flow 50–75% best or predicted.
- No features of acute severe asthma (see below).

Acute severe asthma

Any one of:

- Peak flow 33–50% best or predicted.
- RR ≥25/min.
- Heart rate ≥110/min.
- Inability to complete sentences in one breath.

Life-threatening asthma

A patient with severe asthma with any one of:

- Peak flow <33% best or predicted.
- SpO₂ <92%.
- pO₂ <8kPa.
- Normal pCO₂ (4.6–6.0kPa).
- Silent chest.
- Cyanosis.
- Poor respiratory effort.
- Arrhythmia.
- Exhaustion.
- Altered conscious level.
- Hypotension.

Near-fatal asthma

- ↑ pCO₂ and/or requiring mechanical ventilation with ↑ inflation pressures.

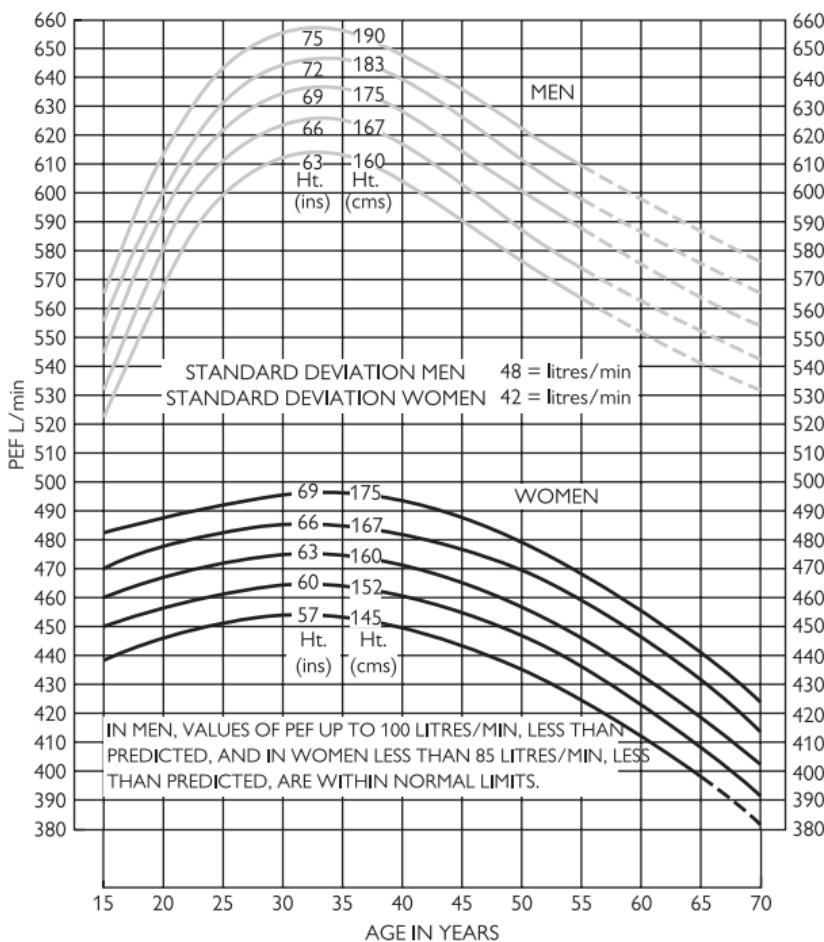


Fig. 3.26 Peak expiratory flow rates in normal adults.

Source: data from Nunn AJ, Gregg I. (1989). New regression equations for predicting peak expiratory flow in adults. *Br Med J* 298: 1068–70.

Investigations in asthma

Peak flow is most useful when expressed as a percentage of that patient's previous best, but the percentage of predicted is a rough guide.

Pulse oximetry (SpO_2) determines the adequacy of O_2 therapy and the need for ABG measurement. Use O_2 to aim for SpO_2 of 94–98%.

Obtain ABG if $\text{SpO}_2 < 92\%$ or if there are other features of life-threatening asthma.

Obtain a CXR (without delaying treatment) if there is:

- Suspected pneumomediastinum or pneumothorax.
- Suspected consolidation.
- Life-threatening asthma.
- Failure to respond to treatment satisfactorily.
- Requirement for ventilation.

Acute asthma: management

Initial treatment

Follow BTS/SIGN guidelines (<https://www.brit-thoracic.org.uk>), summarized as follows:

- Provide high-flow O₂.
- Put the trolley back and side rails up, so the patient is sitting up and holding on to the side rails (to use the pectoral muscles as accessory muscles of respiration).
- If the patient cannot talk, start treatment, but get senior ED and ICU help in case intubation and ventilation are required.
- Check the trachea and chest signs for pneumothorax.
- Ask about previous admissions to ICU.
- Administer high-dose (O₂-driven) nebulized β₂-agonist (eg salbutamol 5mg or terbutaline 10mg), or ten puffs of salbutamol into a spacer device and face mask. For severe asthma or asthma that responds poorly to the initial nebulizer, consider continuous nebulization. Reserve the use of IV salbutamol for those patients in whom inhaled therapy cannot be used reliably (in which case, draw up salbutamol 5mg into 500mL of 5% glucose and run at a rate of 30–60mL/hr).
- Give a corticosteroid to all patients with acute asthma—either prednisolone 40–50mg PO or hydrocortisone (preferably as sodium succinate) 100mg IV.
- Add nebulized ipratropium bromide (500mcg 4- to 6-hourly) to β₂-agonist treatment for patients with acute severe or life-threatening asthma or those with a poor initial response to β₂-agonist therapy.
- Consider a single dose of IV magnesium sulfate (1.2–2g IVI over 20min), after consultation with senior medical staff, for patients with acute severe asthma without a good initial response to inhaled bronchodilator therapy or for those with life-threatening or near-fatal asthma.
- The use of IV aminophylline remains controversial and is not likely to result in any additional bronchodilation, compared to standard care. Use IV aminophylline only after consultation with senior medical staff. Some individual patients with near-fatal or life-threatening asthma with a poor response to initial therapy may gain additional benefit. The loading dose of IV aminophylline is 5mg/kg over 20min, unless on maintenance therapy, in which case check blood theophylline level and start IVI of aminophylline at 0.5–0.7mg/kg/hr.
- A patient who cannot talk will be unable to drink fluids and may be dehydrated.
- Avoid 'routine' antibiotics.
- Repeat ABG within an hour if: initial pO₂ is <8kPa (unless subsequent SpO₂ is >92%), or pCO₂ is normal or ↑, or if the patient deteriorates.
- Hypokalaemia may be caused or exacerbated by β₂-agonist and/or steroid therapy. Correct electrolyte abnormalities.

Criteria for admission

Admit patients with any features of:

- A life-threatening or near-fatal attack.
- Severe attack persisting after initial treatment.

Management of discharge

Consider for discharge those patients whose peak flow is >75% best or predicted 1hr after initial treatment. Prescribe a short course of oral prednisolone (eg 40–50mg for 5 days) if initial PEFR is <50%, and ensure an adequate supply of inhalers. If possible, arrange for review by an asthma liaison nurse before discharge. At a minimum, the inhaler technique and peak expiratory flow monitoring should be reviewed. Arrange/advise GP/asthma liaison nurse follow-up within 2 days. Email/send the discharge summary to the GP. Advise to return to hospital if symptoms worsen/recur.

Referral to intensive care unit

Refer any patient requiring ventilatory support or with acute severe or life-threatening asthma failing to respond to therapy, as evidenced by:

- Deteriorating peak flow.
- Persisting or worsening hypoxia.
- Hypercapnia.
- ABG showing ↓ pH.
- Exhaustion, feeble respiration.
- Drowsiness, confusion, altered conscious state, or respiratory arrest.

Post-intubation care

Mechanical ventilation is associated with a high risk of barotrauma and dynamic hyperinflation. Breath stacking can result in critically reduced venous return and hypotension. High inspiratory pressures and volumes can cause pneumothorax. Typically, set the ventilator to provide low tidal volumes (6–8mL/kg) at a low RR (7–8/min) and inspiratory:expiratory ratio of 1:4.

Cardiac arrest in acute asthma

The underlying rhythm is usually PEA. This may reflect one or more of the following: prolonged severe hypoxia (secondary to severe bronchospasm and mucus plugging), hypoxia-related arrhythmias, or tension pneumothorax (may be bilateral). Give ALS according to the guidelines in  Cardiac arrest, p. 48, and treat tension pneumothorax if present (see  Tension pneumothorax, pp. 338–9). Aim to achieve tracheal intubation early in view of the higher than normal required lung inflation pressures and the attendant risk of gastric inflation in the absence of a tracheal tube.

Chronic obstructive pulmonary disease

COPD is characterized by chronic airflow limitation due to impedance to expiratory airflow, mucosal oedema, infection, bronchospasm, and bronchoconstriction due to ↓ lung elasticity. Smoking is the main cause, but other causes are chronic asthma, α -1 antitrypsin deficiency, and chronic infection (eg bronchiectasis).

History

Exertional dyspnoea, cough, and sputum are usual complaints. Ask about:

- **Present treatment:** including inhalers, steroids, antibiotics, theophyllines, nebulizers, opiate analgesia, and home O_2 treatment.
- **Past history:** enquire about previous admissions and comorbidity.
- **Exercise tolerance:** how far can they walk on the flat without stopping? How many stairs can they climb? Do they get out of the house?
- **Recent history:** ask about wheeze and dyspnoea, and sputum volume and colour. Chest injuries, abdominal problems, and other infections may cause respiratory decompensation.
- **Read the hospital notes:** have there been prior ICU assessments? Has the respiratory consultant advised whether ICU would be appropriate?

Examination

Examine for dyspnoea, tachypnoea, accessory muscle use, and lip-pursing. Look for hyperinflation ('barrel chest'), and listen for wheeze or coarse crackles (large airway secretions). Cyanosis, plethora (due to secondary polycythaemia), and right heart failure (cor pulmonale) suggest advanced disease. Look for evidence of hypercarbia: tremor, bounding pulses, peripheral vasodilatation, drowsiness, or confusion.

Check for evidence of other causes of acute dyspnoea, particularly: asthma (see ↗ Acute asthma: assessment, pp. 108–9), pulmonary oedema (see ↗ Cardiogenic pulmonary oedema, pp. 104–5), pneumothorax (see ↗ Spontaneous pneumothorax, pp. 118–20), and PE (see ↗ Pulmonary embolism, pp. 124–5). Remember that these conditions may coexist with COPD.

Investigation

- SpO_2 , RR, pulse rate, BP, T° , and peak flow (if possible).
- CXR (look for pneumothorax, hyperinflation, bullae, and pneumonia).
- ECG.
- ABG (or VBG), documenting the FiO_2 . Use pCO_2 to guide O_2 therapy.
- FBC, U&E, glucose, theophylline levels, and, if pneumonia is suspected and/or pyrexial, blood cultures, CRP, and pneumococcal antigen.
- Send sputum for microscopy and culture if purulent.

Treatment

Give oxygen Remember that hypercapnia with O_2 is multifactorial. The aim is to maintain SpO_2 of 88–92% without precipitating respiratory acidosis or worsening hypercapnia (see ↗ Oxygen, p. 99 and ↗ Arterial blood gases, pp. 102–3). If the patient is known to have COPD and is drowsy or has a documented history of previous hypercapnic respiratory failure, give an FiO_2 of 28% via a Venturi mask and obtain an ABG. Titrate up the FiO_2 with serial ABG sampling until the minimum FiO_2 that achieves SpO_2 of 88–92%. Reduce inhaled O_2 concentration if SpO_2 is >92%.

Give bronchodilators and steroids

- Give nebulized salbutamol 5mg or terbutaline 5–10mg.
- Consider adding nebulized ipratropium 0.5mg.
- Use O₂-driven nebulizers unless the patient has hypercapnic, acidotic COPD, in which case use nebulizers driven by compressed air, supplemented by O₂ via nasal prongs at 1–4L/min.
- Give steroids (eg prednisolone 30mg PO stat, then continued once daily for 7 days). Use hydrocortisone 100mg IV if the patient cannot take prednisolone PO.

Other drug treatments

- Give antibiotics (eg amoxicillin, doxycycline, or clarithromycin) if the patient reports ↑ purulent sputum or there is clinical evidence of pneumonia and/or consolidation on CXR.
- Only consider IV aminophylline if there is an inadequate response to nebulized bronchodilators. Beware interactions with other drugs and the potential for toxicity if the patient is already taking oral theophylline.
- Consider naloxone if the patient is taking an opioid analgesic that may cause respiratory depression.

(See NICE guideline on COPD, 2018 at <https://www.nice.org.uk>)

Non-invasive ventilation

NIV is standard early therapy for hypercapnic ventilatory failure during exacerbations of COPD. NIV improves blood gas measurements in the ED and ↓ intubation rates, mortality, and length of hospital stay. Ensure patients started on NIV have a plan in the event of deterioration (agreed ceiling of care).

NIV takes two forms—CPAP and BiPAP (which may be more suitable for treating type II respiratory failure in COPD). Both CPAP and BiPAP have been used to treat acute cardiogenic pulmonary oedema. Patients with sleep apnoea use CPAP at night. The positive airway pressure is delivered by a tightly adhered face mask, which is sized to fit the patient. The patient is awake and must be compliant with wearing the mask.

Unlike tracheal intubation, NIV does not protect the airway, so coma and vomiting are contraindications. Absolute contraindications include apnoea and cardiac arrest. Check CXR before starting—a pneumothorax will be converted into a tension pneumothorax with NIV. Severe agitation may make effective NIV impossible.

The patient should always be cared for by staff who are familiar with the ventilator and mask, in the resuscitation room.

Start BiPAP at 10cmH₂O inspiratory positive airway pressure (IPAP)/5cmH₂O expiratory positive airway pressure (EPAP), and titrate upwards:

- To treat persistent hypercapnia, ↑ IPAP by 2cmH₂O at a time.
- To treat persistent hypoxia, ↑ IPAP and EPAP by 2cmH₂O at a time.
- The maximum IPAP/EPAP is 25/15cmH₂O.
- For CPAP, commence treatment at 5–8cmH₂O.

Pneumonia

Pneumonia involves symptoms and signs of lower respiratory tract infection (breathlessness, productive cough, and fever) usually associated with CXR abnormalities. *Pneumocystis* pneumonia may occur with minimal or no CXR changes. Consider pneumonia in patients with septicaemia or acute confusional states.

Causes

Bacterial (80–90%) *Streptococcus pneumoniae* is the most common cause of community-acquired pneumonia. Others include *Mycoplasma pneumoniae*, *Haemophilus influenzae*, *Legionella*, *Chlamydia psittaci*, and *Staphylococcus aureus* (can cause fulminant pneumonia in patients with influenza). Gram –ve and anaerobic infections are rare. Always consider TB, particularly in chronic alcoholism, poor social circumstances, immigrants and those travelling to developing countries, or individuals not BCG-vaccinated. Immunosuppressed patients (eg HIV, steroid therapy) are at ↑ risk of TB and *Pneumocystis jirovecii* pneumonia.

Viral (10–20%) Predominantly COVID-19, influenza A and B, RSV, rarely varicella and SARS.

Rickettsial (1%) Rarely *Coxiella burnetii*.

Signs and symptoms

Fever, cough, and production of sputum are common complaints. Breathlessness, pleuritic chest pain, myalgia, rigors, or haemoptysis may occur. Pneumonia can present without obvious chest signs. *Mycoplasma* pneumonia may present in children and young adults with sore throat, headache, nausea, abdominal pain, and diarrhoea. *Legionella* can present with constitutional upset, diarrhoea, or confusion, particularly in the elderly. *Pneumocystis* pneumonia in immunosuppressed patients may present with cough, dyspnoea, and marked hypoxia, with relatively few other findings.

Examination and investigation

- If there is suspicion of COVID-19 infection, place the patient in isolation, restrict staff interaction, and ensure staff don PPE prior to entering the room.
- Assess for signs of severe sepsis.
- Check RR, pulse, and BP.
- Auscultation may reveal a patch of inspiratory crackles; signs of consolidation are present in <25%.
- Check BMG and SpO₂ (obtain ABG if <90% or known to have COPD).
- Take blood for U&E, FBC, and CRP, and blood cultures before giving IV antibiotics.
- Obtain a CXR. Look for patchy or lobar opacification (see Fig 3.27), mass lesions, or an air bronchogram. In early pneumonia, the CXR may be normal.
- Obtain blood cultures and sputum cultures, and consider urinary pneumococcal and *Legionella* antigen testing. If suspicion of COVID-19 infection, take a nasopharyngeal swab for viral testing.

Assessment: admit or discharge

Some patients with 'mild' illness, good social circumstances, and no significant comorbidity may be safely discharged with appropriate antibiotics (eg amoxicillin 0.5–1g PO tds), simple analgesia for pleuritic pain to aid deep breathing/coughing, and GP follow-up.

Patients with a CURB-65 score of ≥ 3 (see Table 3.7) have severe pneumonia with a high risk of death; those who score 2 are at ↑ risk of death and should be considered for inpatient treatment or hospital-supervised outpatient care; patients with a CURB-65 score of 0 or 1 are at low risk of death and may be suitable for home treatment (<https://www.brit-thoracic.org.uk>).

Table 3.7 CURB-65 score for pneumonia

	Score
Confusion	1
Urea $>7\text{ mmol/L}$	1
RR $\geq 30/\text{min}$	1
Low BP (systolic $<90\text{ mmHg}$ or diastolic $\leq 60\text{ mmHg}$)	1
Age $\geq 65\text{ years}$	1

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Treatment

(See NICE guideline 2014 at <https://www.nice.org.uk>)

Patients deemed suitable for discharge

Give analgesia, oral antibiotics for 5 days, and GP follow-up (including a decision about the need to repeat CXR in 6 weeks to confirm changes have cleared). Explain to the patient what rate of recovery to expect.

Patients admitted, but not severely unwell

Start either PO or IV antibiotics, as follows:

- Either amoxicillin 0.5–1g PO tds + erythromycin 500mg PO qds (or clarithromycin 500mg bd).
- Or if IV therapy is needed: ampicillin 500mg IV qds + erythromycin 500mg IV qds (or clarithromycin 500mg bd). Local guidelines will apply.
- Monitor SpO₂ and provide O₂ accordingly.
- Provide simple analgesia.

Patients with sepsis

(See  Sepsis, pp. 62–3.)

Commence IV crystalloid fluids; take blood cultures and administer IV antibiotics (eg co-amoxiclav 1.2g IV tds + clarithromycin 500mg IV bd) immediately. Contact ICU and insert a urinary catheter. Aim for mean arterial pressure (MAP) of $>65\text{ mmHg}$ and urine output of $>0.5\text{ mg/kg/hr}$. (See sepsis guidelines at <http://www.survivingsepsis.org>)

Differential diagnosis

Pneumonia-like presentations can occur with pulmonary oedema, pulmonary infarction, pulmonary vasculitis (eg SLE, PAN, Churg–Strauss syndrome, and granulomatosis with polyangiitis), aspergillosis, allergic alveolitis, bronchial or alveolar cell carcinoma, acute pancreatitis, and subphrenic abscess.

Pulmonary aspiration

Aspiration of solid or liquid material into the upper and lower airways is likely when one or more of the following features are present:

- ↓ GCS: head injury, stroke, overdose, seizures, sedation, anaesthesia.
- ↓ cough and/or gag reflexes: related to above factors and/or bulbar dysfunction, intubation/extubation, Guillain–Barré syndrome, multiple sclerosis (MS), myasthenia gravis.
- Tendency to regurgitate/vomit: alcohol, full stomach, upper GI tract pathology (including hiatus hernia, oesophageal obstruction, pregnancy).
- May occur in infirm or elderly fed via NG tube.

Clinical features

Large food particles sufficient to cause complete airway obstruction cause choking, inability to speak, ↑ respiratory effort, cyanosis, loss of consciousness, and death. Smaller particles may pass through the vocal cords, causing coughing, stridor, tachypnoea, and wheeze. 80% of patients are aged <4y, with peanuts being the classic inhaled objects. Delayed presentation with cough, wheeze, haemoptysis, unresolved pneumonia, abscess formation, or empyema occurs in ~30%, often days or weeks later.

Vomiting/regurgitation is often witnessed, and pulmonary aspiration confirmed by seeing gastric contents in the oropharynx or trachea during intubation or following suction. Gastric content is a mixture of semi-solid and liquid material—aspiration leads to a sudden onset of severe dyspnoea, wheeze, and cyanosis. Its acid nature causes severe damage to the alveolar–capillary membrane, with denaturation of pulmonary surfactant and ↑ pulmonary permeability, with oedema and atelectasis.

Hydrocarbons (eg petrol, paraffin) cause severe pulmonary toxicity if aspiration occurs during ingestion or following regurgitation/vomiting.

Investigation

ABG

These show hypoxaemia within minutes of acid aspiration. Initially, patients may hyperventilate, with ↓ pCO₂, until pulmonary compliance ↑ work of breathing, sufficient to result in hypoventilation.

CXR

Abnormalities develop in >90% of patients but may take hours/days. Appearances depend on the nature of the aspirated material and the patient's position at the time of the episode (the right lower lobe is most frequently and severely affected, followed by the left lower lobe and the right middle lobe). In severe aspiration, diffuse bilateral infiltrates and pulmonary oedema similar to ARDS appearances are present. Less severe episodes produce atelectasis, followed by alveolar infiltration.

Intrapulmonary foreign body (including peanuts)

Rarely radio-opaque. Resulting collapse, hyperinflation, or consolidation are usually obvious and depend on whether obstruction is complete or partial and if supervening infection is present. If the history strongly suggests an inhaled FB, but CXR is normal, consider bronchoscopy or CT.

Prevention

Prevention is everything. Pay meticulous attention to airway protection. This may involve positioning (tilt head down on the right-hand side), suction to the oropharynx (Yankauer catheter avoiding stimulation of the gag reflex), and, if necessary, tracheal intubation. Tracheal intubation does not completely protect against aspiration of fluid into the lungs, but it is the best preventative measure. In at-risk patients, pass an NG tube to empty the stomach. However, NG tubes can also predispose to aspiration by preventing closure of the oesophageal sphincters and interfering with coughing and clearing the pharynx.

Treatment

Correct hypoxia and give nebulized salbutamol for associated bronchospasm. If particulate aspiration is present, refer for urgent bronchoscopy. Although secondary infection is common, the use of antibiotics or steroids is not routinely indicated.



Fig. 3.27 CXR showing right upper lobe pneumonia.

Spontaneous pneumothorax

Primary spontaneous pneumothorax (PSP) may occur in previously healthy individuals. Secondary spontaneous pneumothorax (SSP) occurs in older patients with pre-existing chronic lung disease (like COPD or TB) and may also occur with asthma, bronchial carcinoma, Marfan's syndrome, infection, cystic fibrosis, and oesophageal rupture.

Presentation

Most patients present with unilateral pleuritic chest pain and dyspnoea. Classical physical signs may not be present (depending upon the size of the pneumothorax): tachypnoea, tachycardia, normal/hyper-resonant percussion note with ↓ air entry on the affected side. Rarely, there may be a clicking sound at the cardiac apex.

Severe symptoms (inability to speak, gasping, low SpO₂) should prompt rapid assessment for tension pneumothorax: tracheal deviation, tachypnoea, tachycardia, and hypotension. Treat tension pneumothorax with immediate decompression using a needle in the second intercostal space (just above the third rib) in the mid-clavicular line (see ↗ Tension pneumothorax, pp. 338–9). Severe symptoms are also found in patients with SSP (disproportionate to the pneumothorax size). In the absence of signs of tension pneumothorax, obtain an emergency portable CXR and involve an experienced doctor.

Initial assessment and management

- Monitor pulse, SpO₂, and BP. Ensure IV access.
- Administer high-flow O₂ (in patients with COPD, aim for an SpO₂ of 90–92%).
- When there are no signs of tension, an ABG will help assess patients with chronic lung disease and guide O₂ therapy.
- Erect CXR is the principal way of making the diagnosis (see Fig. 3.28), but beware pitfalls (see ↗ Pitfalls in CXR analysis for possible pneumothorax, p. 120).
- CT scan is not the primary diagnostic modality but can identify small pneumothoraces not apparent on the CXR. CT is also of use in the subacute setting for assessing bullous lung disease in a stable patient.

Intervention

- Follow the algorithm shown in Fig. 3.29.
- Be guided primarily by the patient's symptoms. If the patient is breathless, they should undergo an intervention.
- The size of the pneumothorax can be estimated on CXR by measuring from the chest wall to the lung edge at the level of the hilum. This is only an estimate and assumes symmetrical lung collapse. The cut-off of 2cm is used to determine treatment.
- Intervention for PSP is needle aspiration. If unsuccessful, do not repeat aspiration. Instead insert a Seldinger chest drain.
- Treatment for symptomatic SSP is chest drain insertion and admission.
- Treatment for SSP without breathlessness is admission. Aspiration should be performed by an experienced doctor and may require CT.
- Adopt a very low threshold for inserting chest drains for bilateral pneumothoraces.

- Always insert a chest drain immediately following emergency needle decompression.
- Pleural aspiration and drain insertion should be performed by a doctor who has prior training and experience.
- Ensure the patient has IV access. Perform in a monitored environment with an assistant and appropriate supervision. Use aseptic technique.
- Always discuss the procedure with the patient, and document that they have given their consent.
- If the patient is on anticoagulation or has a known coagulopathy disorder, discuss with a haematologist first.

Aspiration technique

Confirm the side of the pneumothorax. Sit the patient upright. Infiltrate 1% lidocaine, then insert a 16G IV cannula just above the third rib (in the second intercostal space) in the mid-clavicular line. Alternatively, lay the patient on their side, with the pneumothorax side upwards. Insert a cannula in the fifth intercostal space in the anterior axillary line. Remove the needle; attach a three-way tap, then aspirate air with a 50mL syringe. Continue aspiration until the patient coughs excessively or until 2.5L of air is removed.

Seldinger chest drain insertion

Confirm the side of the pneumothorax. Keep the patient comfortable; ensure adequate analgesia (this may require 1mg increments of morphine IV), but avoid sedation. Sit the patient upright, and rest their hand behind their head. Infiltrate 10mL of 1% lidocaine at the anterior axillary line in the fifth intercostal space. Aspirate a small amount of pleural air during infiltration and note the depth of the pleural space. Locate the pleural space with the introducer needle (aspirate whilst advancing through the chest wall), then advance the guidewire through the needle. Remove the introducer needle; make a small skin incision, and gently pass the dilator over the guidewire using a twisting action. Do not push the dilator >1cm past the depth of the pleural space. Pass the chest drain over the guidewire to a depth of 10–12cm. Remove the guidewire; connect to an underwater seal drain and suture in place. Check the drain is bubbling and swinging, and organize a CXR.

Discharge

Patients without breathlessness with a small PSP may be considered for discharge. Give the patient verbal and written instructions to return if their symptoms worsen and warn them not to fly or go diving. Ensure they have a plan for follow-up (and repeat CXR) with a respiratory physician or GP in 2–4 weeks (see  <https://www.brit-thoracic.org.uk>).



Fig. 3.28 CXR showing spontaneous right pneumothorax.

Pitfalls in CXR analysis for possible pneumothorax

- When using digital images, always use a picture archiving and communication system (PACS) workstation. The signs of pneumothorax may be subtle and difficult to spot. Compare with previous CXRs, if available.
- Look for a displacement of the pleural line.
- Do not mistake the scapular edge for the lung edge. Similarly, clothing, O₂ tubing, and overlying sheets can cause artefacts which mimic the edge of the lung.
- Some patients with COPD have emphysematous bullae, which can mimic pneumothorax. If in doubt, ask for senior review prior to treating for pneumothorax.
- An air–fluid level at the costophrenic angle may be present.

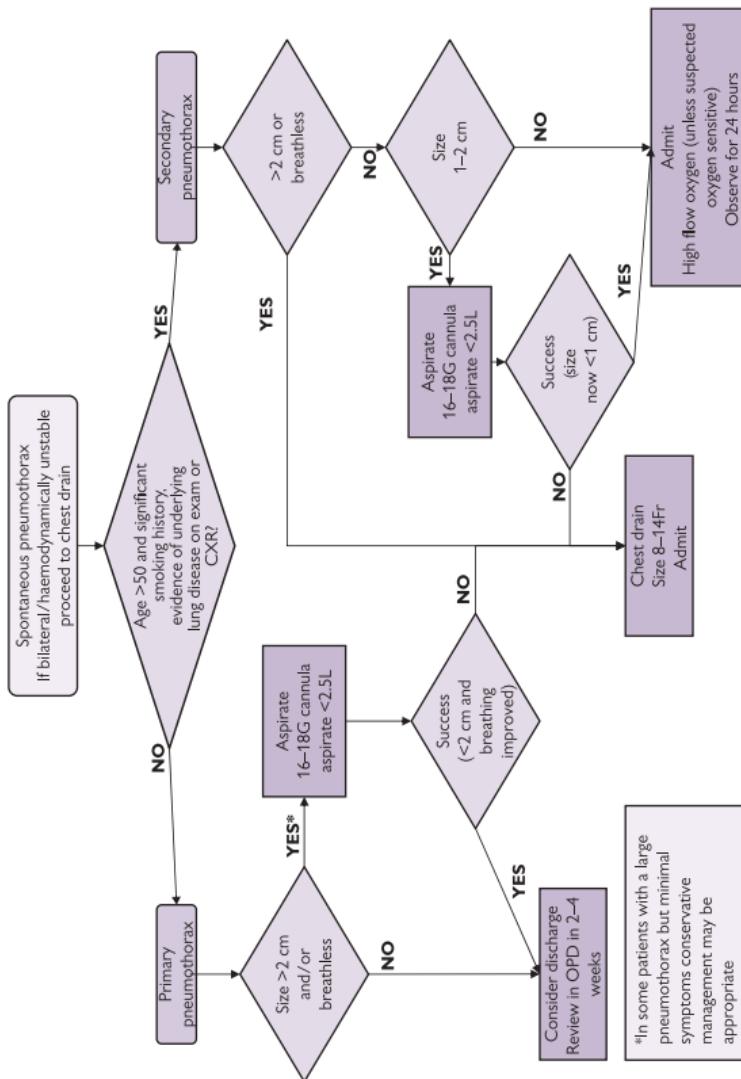


Fig. 3.29 Management of pneumothorax.
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Deep vein thrombosis

DVT and PE are manifestations of the same disease process whereby abnormal clotting occurs in the veins of the legs or pelvis. The clots may break from the vein wall and embolize to the lungs. Untreated DVTs are associated with 1% mortality from PE. Around half of those with DVT will go on to develop post-thrombotic syndrome, with lifelong pain and swelling of the leg.

Risk factors

- Recent surgery (where a general anaesthetic was administered, especially orthopaedic, abdominal, spinal, and obstetric).
- Recent admission to hospital.
- Current malignancy.
- Being bedbound.
- Sepsis.
- IV drug use (where the patient injects in the femoral vein).
- Pregnancy/pelvic masses.
- Limb immobility such as recent fracture with crutches and plaster cast.
- Previous DVT/PE.
- Thrombophilia or family history of venous thromboembolism.

Clinical features

DVT classically produces leg pain with swelling, warmth, tenderness, and dilated superficial veins in the affected leg. These signs are non-specific and often not present. A small or partially occluding thrombus may be asymptomatic. History and clinical examination alone cannot safely exclude DVT—if a DVT is suspected, investigate further. Investigate for PE instead if the patient has tachycardia, hypoxia, ↑ RR, or breathlessness (see ↗ Pulmonary embolism, pp. 124–5).

Differential diagnosis

- Muscular tear: typically acute onset.
- Rupture of a Baker's cyst: again, typically acute onset.
- Cellulitis or other infection.

Investigation and management

- Record pulse rate, RR, BP, SpO₂, and T° in all patients.
- Take a full history, including concurrent illness, past history, recent operations, travel, and family history.
- Examine the affected leg for signs of plethora, deep vein tenderness, swelling (measure both legs, 10cm distal to the tibial tuberosity), oedema, and dilatation of the skin veins.
- Perform a full examination, checking for signs of PE or occult carcinoma.
- Calculate the clinical probability assessment score. The Wells score (see Table 3.8) is the most widely used clinical prediction score.
- Take FBC, U&E, CRP, and glucose.
- Take D-dimer if the Wells score indicates DVT is 'unlikely' (<2 points).
- If D-dimer normal *and* DVT 'unlikely', DVT has been ruled out.

Table 3.8 Wells clinical probability assessment score for DVT

Clinical feature	Score
Active cancer (treatment ongoing or within 6 months or palliative)	1
Paralysis, paresis, or recent POP immobilization of a leg	1
Recently bedridden for >3 days or major surgery <12 weeks	1
Localized tenderness along the distribution of the deep venous system	1
Entire leg swelling	1
Calf swelling >3cm, compared with asymptomatic leg	1
Pitting oedema (greater in the symptomatic leg)	1
Dilated superficial veins (non-varicose)	1
Previously documented DVT	1
Another diagnosis more likely than DVT	-2

Total score ≤1 means DVT is 'unlikely'. Score of ≥2 signifies DVT is 'likely'.

All patients investigated for DVT with a 'likely' Wells score (≥2) or an elevated D-dimer level require USS. A normal whole leg USS (femoral, popliteal, and calf vein scan) will exclude DVT. If a thigh scan is performed (femoral and popliteal veins), DVT can only be excluded by two normal thigh USS, 1 week apart.

Anticoagulate with LMWH all patients with a 'likely' score whilst awaiting an outpatient USS (see Table 3.9).

Treat patients diagnosed with calf or thigh DVT with LMWH, rivaroxaban, or apixaban, and discharge with a sufficient supply and medical outpatient follow-up. Advise them to return immediately if they become breathless or have chest pain. Note: if prescribing apixaban, be sure to include the dose reduction at 7 days (see Table 3.10).

Upper limb DVT

Often seen with chemotherapy central or long lines. May be associated with plethora and swelling of the arm or face. If suspected, request an USS of the axillary, subclavian, and jugular veins, or a CT scan of the central veins. Treat as for lower limb DVT.

Superficial thrombophlebitis

Patients present with a painful, tender area of the skin. The diagnosis is made clinically with a firm, tender superficial vein and overlying erythema. This may coexist with DVT. If there is any doubt as to the presence of a DVT, investigate using the DVT protocol. Otherwise, treat with an NSAID or a 6 week course of an oral anticoagulant. Arrange follow-up either in the ED, in a medical clinic, or with the GP to check for resolution.

Pulmonary embolism

The mortality of diagnosed and treated PE is 7%. Pulmonary embolic disease can result in a variety of symptoms often misdiagnosed as asthma, anxiety, pneumonia, and ACS.

History

Most patients with PE experience dyspnoea, commonly without other symptoms. Syncope with cyanosis, cardiac arrest, or angina are signs of massive PE. A minority present with pleuritic chest pain, some with additional haemoptysis. Always consider PE in patients with unexplained hypoxia or breathlessness. Take a full history of concurrent illness, surgical procedures, recent hospital admission, past history, including DVT and PE, and travel and family history.

Examination

Examination may be normal.

- Tachycardia and tachypnoea are common.
- Pyrexia following lung infarction is common.
- 30% of all patients with PE have normal SpO₂.
- Always record BP. Hypotension indicates massive PE.
- Perform a full respiratory and cardiovascular examination.
- Always examine the legs for signs of DVT.

Table 3.9 Modified Wells clinical probability assessment score for PE

Clinical feature	Score
Signs of DVT (minimum of objective leg swelling and tenderness)	3.0
IV drug use	3.0
PE is the most likely diagnosis	3.0
Heart rate >100	1.5
Prior PE or DVT diagnosis	1.5
Bedridden for >3 days or surgery within the past 4 weeks	1.5
Cancer (treated actively or with palliation within last 6 months)	1.0
Haemoptysis	1.0

Total score ≤4.0 = PE unlikely; score ≥4.5 = PE likely.

Any patient scoring ≥4.5 on the Wells score OR who has an elevated D-dimer requires pulmonary imaging. Only a normal D-dimer AND a 'PE unlikely' Wells score will safely exclude PE.

Investigations for suspected PE

- If hypoxic, tachycardic, or hypotensive, insert an IV cannula.
- All patients should have blood taken for FBC and U&E.
- Take a D-dimer test on any patient who scores ≤4.0 on the Wells score (see Table 3.9). A normal D-dimer in a patient scoring <4.0 excludes PE.
- Arrange an ECG (to look for MI or pericarditis) and a CXR (to look for pneumothorax or pneumonia). ECG and CXR are often normal in PE.

Diagnostic imaging for pulmonary embolus

There are two forms of imaging for PE: CT pulmonary angiography (CTPA) and ventilation–perfusion (V/Q) scanning. CTPA uses a higher dose of radiation (not good for young patients) but will give a definitive answer, as well as diagnose other conditions (like aortic dissection).

Planar V/Q and V/Q single-photon emission CT (SPECT) use a lower dose of radiation but may not give a definitive answer. The V/Q scan result must concord with the clinical probability to diagnose or exclude PE (both PE unlikely or both PE likely). Other combinations are non-diagnostic and necessitate CTPA.

Treatment of DVT/PE

Aim to treat patients with PE as outpatients after senior review if they are ambulant and have normal SpO_2 , RR, and heart rate. Outpatient treatment is dependent on reliable follow-up. Admit those who are hypoxic, hypotensive, tachycardic, tachypnoeic, or unable to cope at home (see  <https://www.brit-thoracic.org.uk>).

As soon as venous thrombosis is confirmed or if there is a delay of >4hr to diagnose in a high-risk patient, give a dose of anticoagulation (see Table 3.10).

Table 3.10 Choice of anticoagulant drug for PE in the ED

Anticoagulant	Dose
Rivaroxaban	15mg PO bd for 21 days, then 20mg PO daily
Apixaban	10mg PO bd for 7 days, then 5mg PO bd
Enoxaparin	1mg/kg SC bd, max 100mg bd
Dalteparin	200U/kg SC daily, max 18,000U
Tinzaparin	175U/kg SC daily, max 18,000U
Unfractionated heparin	IVI—arrange admission for warfarinization Give if estimated glomerular filtration rate (eGFR) <20mL/min

Suspected massive PE

- In patients with cardiovascular compromise, call for urgent ICU help.
- Bedside echo will demonstrate a dilated RV.
- Bedside USS may demonstrate DVT.
- Do not take unstable patients for CT or V/Q scanning.
- If suspicion of PE is high and the patient is haemodynamically unstable, administer thrombolytic therapy. Do not delay. Administer alteplase (rtPA) 10mg slow IV over 1–2min, followed by 90mg IVI over 2hr (max dose 1.5mg/kg if patient is <65kg).
- If thrombolysis is contraindicated or does not work, liaise with experts to consider other alternatives, if available (eg surgical embolectomy, catheter-directed thrombolysis).
- After thrombolysis, start unfractionated heparin IVI, with the dose based on the patient's weight.

Upper gastrointestinal bleeding

Causes of upper gastrointestinal bleeding

Common

- Peptic ulceration.
- Mucosal inflammation (oesophagitis, gastritis, or duodenitis).
- Oesophageal varices.
- Mallory–Weiss tear.
- Gastric carcinoma.
- Coagulation disorders (thrombocytopenia, warfarin).

Rare

- Aorto-enteric fistula (especially after aortic surgery).
- Benign tumours (eg leiomyomas, carcinoid tumours, angiomas).
- Congenital (eg Ehlers–Danlos, Osler–Weber–Rendu, pseudoxanthoma elasticum).

History

Take a detailed history, whilst resuscitating as necessary. Upper GI bleeding usually presents with haematemesis and/or melaena, and bleeding involving the lower GI tract with fresh per rectum (PR) bleeding. However, major upper GI bleeding may present with fresh PR bleeding.

Ask about the amount and duration of bleeding, any past history of GI bleeding or liver problems, and associated symptoms (abdominal pain, weight loss, anorexia). Syncope usually infers a significant bleed. Take a full drug history (ask about aspirin, NSAIDs, anticoagulants, iron), and enquire about alcohol consumption.

Examination

Check ABCs. Rapidly assess for hypovolaemic shock (pulse and RR, BP, GCS, skin colour/T°, capillary refill time). Look at any available vomit or faeces. Check for abdominal masses, tenderness, or surgical scars (including aortic grafting). Look for stigmata of liver disease. Perform a PR examination and check for faecal occult blood (FOB).

Investigations and diagnosis

Review the patient's old hospital notes, and send blood for FBC, clotting screen, U&E, blood glucose, and group and save or cross-matching (according to clinical features). Urea may be ↑, but creatinine will be normal unless renal function is impaired. Check SpO₂ (obtain ABG if <94%), and consider CXR and ECG. Endoscopy is the investigation of choice to identify the source of the bleeding.

Risk of further bleeding and death

The risk of mortality and further complications ↑ with ↑ age, comorbidities (especially cancer and heart failure), liver disease, continued bleeding, ↑ urea, and passage of PR blood. Scoring systems can provide an indication of the chance of a rebleed and/or death. The Glasgow–Blatchford score (see Table 3.11) is more useful in the ED than the Rockall score to identify which patients do not need admission.

Table 3.11 Glasgow–Blatchford score for upper GI bleeding

Blood urea (mmol/dL)		Systolic BP (mmHg)
6.5–8 = 2pt		100–109 = 1pt
8–10 = 3pt		90–99 = 2pt
10–25 = 4pt		<90 = 3pt
>25 = 6pt		
Hb (g/L) for men	Hb for women	Other markers:
120–129 = 1pt	100–119 = 3pt	Pulse ≥100/min = 1pt
100–119 = 3pt	<100 = 6pt	Presentation with melaena = 1pt
<100 = 6pt		Presentation with syncope = 2pt
		Hepatic disease = 2pt
		Cardiac failure = 2pt

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Only consider patients scoring 0 on the initial Blatchford score (see Table 3.11), with no further evidence of bleeding, for discharge home from the ED with follow-up. Any patient scoring >0 requires urgent endoscopy.

Treatment of moderate/severe haemorrhage

- Check airway/breathing. Give O₂ as needed (see Oxygen, p. 99). Insert two large-bore (14G) IV cannulae, and send blood for FBC, U&E, clotting, and cross-matching.
- Start IV fluids, followed by blood, as necessary.
- Avoid omeprazole acutely, unless the patient has known peptic ulcer disease (give 40mg diluted in 100mL of saline as IVI over 30min).
- If the patient is anticoagulated or has altered clotting (eg liver disease), talk to haematology. Reverse anticoagulation giving vitamin K/prothrombin complex concentrate/idarucizumab, as needed.
- Insert a urinary catheter and monitor the urine output.
- Ensure that patients with severe uncontrolled variceal bleeding, severe encephalopathy, hypoxia, acute agitation, or evidence of aspiration have their airways secured, if necessary by tracheal intubation.
- Transfuse using the massive transfusion protocol to maintain Hb >70g/L.

Managing severe haemorrhage possibly due to varices

For unstable patients with a past history of varices or clinical features of hepatic failure, arrange emergency endoscopic treatment.

- Commence fluid resuscitation.
- Give terlipressin (2mg IV, repeated every 4–6hr).
- Check the international normalized ratio (INR), and give IV vitamin K if prolonged.
- Give prophylactic antibiotics, eg ciprofloxacin or second-/third-generation cephalosporin, which may ↓ mortality in severe haemorrhage.
- Consider balloon tamponade as a salvage procedure in a patient with massive haemorrhage at risk of death. If experienced in the technique, insert a 4-lumen Sengstaken/Minnesota tube. Inflate the gastric balloon, then the oesophageal balloon to a pressure of 30–40mmHg in order to tamponade the bleeding varices. Regularly aspirate both ports.

(See <https://www.nice.org.uk>)

Lower gastrointestinal bleeding

The most common cause of apparent lower GI bleeding is upper GI haemorrhage. ~20% of acute GI haemorrhage is from the colon or rectum. Angiodysplasia and bleeding from diverticula are the most frequent causes, but inflammatory bowel disease or, very rarely, aorto-enteric fistulae may be responsible. Lower GI haemorrhage often settles spontaneously—localization of the bleeding source may be difficult.

History

Nature of bleeding Melaena may occur following small bowel or proximal colon bleeding, as well as upper GI haemorrhage. Conversely, large volumes of fresh or 'plum-coloured' rectal bleeding may follow upper GI haemorrhage. Bloody diarrhoea suggests inflammatory bowel disease or infective colitis.

Associated symptoms Weight loss, anorexia, or a change in bowel habit raise suspicion of colonic carcinoma. Abdominal pain may be a feature of ischaemic colitis, inflammatory bowel disease, or carcinoma. Anal pain commonly occurs with anal fissure or as a complication of haemorrhoids.

Syncope or postural dizziness May indicate significant haemorrhage.

Past medical history Ask about inflammatory bowel disease, peptic ulceration, or other illnesses. Previous aortic surgery with graft insertion can rarely result in the formation of an aorto-enteric fistula (symptoms include sporadic or fulminant bleeding, often with syncope).

Drug history Ask about salicylates, NSAIDs, corticosteroids, and anticoagulants.

Family and social history Note any family history of peptic ulcers or inflammatory bowel disease. Enquire about alcohol consumption.

Examination

First assess for signs of hypovolaemia, and commence resuscitation if necessary. Document pulse, BP (comparing erect and supine, noting any postural drop), T°, and SpO₂. Examine the abdomen, and PR in all cases.

Investigation

Obtain blood for cross-matching (ask for 4–6U of type-specific if urgent), FBC, U&E, glucose, and coagulation studies. Perform an ECG on any patient >50y. Review old patient notes, especially in relation to previous colonoscopy or GI pathology.

Risk of further bleeding and death

The risk of mortality and further complications ↑ with ↑ age, comorbidities, haemodynamic disturbance, and the use of NSAIDs or aspirin. Only consider discharge if the patient is young and otherwise healthy, has passed only a small amount of blood PR, and does not take NSAIDs or anticoagulants. Always arrange follow-up for these patients.

Treatment

Patients with signs of hypovolaemia require immediate resuscitation:

- Give O₂ as required (see Oxygen, p. 99).
- Attach monitoring (cardiac monitor, SpO₂, BP monitoring).
- Insert two large-bore IV cannulae.
- Give 1L of 0.9% saline or Hartmann's solution IV stat, and give further fluids according to response.
- Insert a urinary catheter.
- If the patient is anticoagulated or has a clotting disorder (eg due to liver disease), reverse anticoagulation and discuss with a haematologist.
- Consider the need for a central venous line.
- Contact the surgical team and ICU.

(See <https://www.sign.ac.uk>)

Gastrostomy tube problems

An ↑ number of individuals are being managed in the community in a variety of care settings with indwelling percutaneous endoscopic gastrostomy (PEG) tubes, which are being used for enteral nutrition and administration of medications.

Tube misplacement

Gastrostomy tubes are not infrequently pulled out or 'fall out' (become inadvertently misplaced). The track can close off within hours, so triage ahead and contact gastroenterology—if not immediately available, gently attempt to pass a replacement tube. A lubricated Foley catheter (of size ≤ original PEG tube) can act as a temporary measure to keep the track open (do not feed through this).

Other problems

Tube blockage may respond to gentle flushing with warm water using a 20mL syringe. Contact the gastroenterology team for other problems (infection, bleeding, or gastric leakage).

Refeeding syndrome

This relatively rare, but potentially life-threatening, condition is not always recognized early. Any patients who have not eaten for ≥5 days are at risk. Patients with low body mass index (BMI)/anorexia and alcoholics are at particular risk. Hypophosphataemia, hypomagnesaemia, and hypokalaemia can occur and worsen as food is reintroduced, so check baseline blood glucose and U&E (including phosphate and magnesium) in patients at risk, before considering starting to feed. Give oral thiamine and oral or IV vitamin B, and get expert advice before feeding. Rehydrate and start to correct electrolyte abnormalities. Ensure that, when feeding is commenced, it starts very slowly and with regular monitoring of electrolytes.

Jaundice

Serum bilirubin levels are usually >51 micromoles/L before clinical jaundice occurs. Causation may be categorized as pre-hepatic, intrahepatic, and post-hepatic, although a mixed aetiology may be present (see Table 3.12).

Table 3.12 Causes of jaundice

Pre-hepatic	<ul style="list-style-type: none"> ● Haemolytic anaemia ● Malaria
Intrahepatic	<ul style="list-style-type: none"> ● Viral infection (eg hepatitis A–E, EBV, leptospirosis) ● Alcohol ● Gilbert's syndrome ● Paracetamol poisoning/drugs ● Autoimmune liver disease ● Non-alcoholic fatty liver disease ● Biliary malignancy
Post-hepatic	<ul style="list-style-type: none"> ● Gallstones ● Malignancy (eg pancreatic, biliary, hepatic) ● Pancreatitis

Excessive ingestion of β -carotene can lead to pseudo-jaundice (although sclerae remain normal in colour).

History and examination

Ask about the duration of symptoms and any other associated features such as itching and weight loss. Pale stools and dark urine may be seen in obstructive jaundice. Ascertain recent prescribed, over-the-counter, and illicit drug use (including herbal medicines), alcohol intake, foreign travel, tattoos, and piercings. Red flags include evidence of hepatic dysfunction/encephalopathy (see \ominus Acute confusional state, pp. 140–1), haematemesis or melaena, or signs of sepsis (see \ominus Sepsis, pp. 62–3). Check for hepatosplenomegaly, abdominal tenderness, and masses.

Investigations

- Check FBC, coagulation screen, U&E, LFTs, and amylase. Alkaline phosphatase is \uparrow more than alanine aminotransferase (ALT) in cholestatic aetiologies— \uparrow gamma glutamyl transpeptidase can confirm this. \uparrow ALT suggests a hepatic cause. Isolated \uparrow bilirubin most commonly reflects Gilbert's syndrome. Aspartate aminotransferase (AST):ALT ratio of >1 is seen in alcohol-induced liver disease.
- Consider a viral hepatitis screen.
- Urinalysis: bilirubin in the urine suggests conjugated hyperbilirubinaemia.
- USS can identify gallstones, biliary duct dilatation, and some tumours.
- CT can identify smaller liver and pancreatic lesions.

Management

Admit the patient if acutely unwell, appears septic, is encephalopathic, or has red flags. Generally, admit obstructive causes under surgeons, and others under a medical team—follow local policy. Refer patients who do not require admission to an appropriate outpatient clinic.

Ascites and liver failure

Background

Ascites is an abnormal accumulation of fluid within the peritoneal cavity (deriving from the Greek 'askos', meaning pot or bag). Up to 20mL of fluid may be present physiologically in women of child-bearing age. The most common cause of ascites is hepatic cirrhosis, but it can occur with heart failure, peritonitis, pancreatitis, TB, acute hepatitis, and intra-abdominal malignancy. Patients having peritoneal dialysis also have excess fluid within the peritoneal cavity and are at particular risk of bacterial peritonitis. Cirrhotic ascites is associated with a poor prognosis. If refractory, the survival at 1y is <50%.

Clinical features

Patients may attend the ED when the volume of ascitic fluid causes discomfort due to local pressure effects on the GI tract. Loss of appetite, nausea, and altered bowel habit are often features. Respiratory difficulty may occur due to diaphragmatic compression. Look for stigmata of liver disease. Gross ascites is often clearly visualized with a taut, distended abdomen out of proportion with the patient's body habitus. Examine for shifting dullness and fluid thrill. Check for other evidence of hepatic decompensation, i.e. jaundice, encephalopathy, and variceal haemorrhage. If clinical evidence of infection, consider spontaneous bacterial peritonitis.

Investigations

- FBC, U&E, LFTs, amylase, coagulation screen, group and save (cross-match if variceal bleed suspected).
- Ammonia level if hepatic encephalopathy suspected.
- Hepatitis screen.
- Bedside USS may confirm ascites.
- USS or CT may help identify the cause if it is not apparent.
- Perform an ascitic tap under USS guidance and send fluid for microscopy, culture and sensitivity, neutrophil count (EDTA tube), protein, amylase, and cytology if infection is suspected.

Management

- Resuscitate as appropriate.
- If there is evidence of an upper GI bleed, consider treatment as for oesophageal varices, even if no previous endoscopy (see Upper gastrointestinal bleeding, pp. 126–7).
- If evidence of spontaneous bacterial peritonitis or peritoneal dialysis infection, commence empirical IV antibiotics (e.g. ciprofloxacin PO 500mg bd, or in severe infection/unable to swallow piperacillin–tazobactam IV 4.5g tds).
- Treat hyponatraemia (see Sodium derangements, p. 162).
- If encephalopathic, prescribe lactulose.
- Stop NSAIDs and ACE inhibitors.
- Refer to gastroenterology for ascitic drainage and albumin replacement. If the patient is not overtly unwell, they may be able to have drainage the next day in an ambulatory setting.

Headache

Headaches of non-traumatic origin account for ~0.5% of ED attendances—10–15% of these have serious underlying pathology. Patients typically present in one of three ways:

- Severe headache, unlike any previous one ('first severe' or 'worst ever').
- Headache with associated worrying features (altered mental status, fever, focal neurology).
- Chronic severe headache unresponsive to treatment.

Causes

Primary headaches

- Migraine.
- Tension headaches.
- Cluster headaches.
- Miscellaneous (benign cough headache, benign exertional headache, headache associated with sexual activity).

Secondary headaches

- Head injury.
- Vascular (stroke, intracranial haematoma, subarachnoid haemorrhage, unruptured arteriovenous malformation, venous thrombosis, hypertension).
- Non-vascular intracranial disorder (\uparrow CSF pressure, post-LP, intracranial tumour).
- Substance misuse or withdrawal (including analgesia withdrawal or rebound).
- Infection (encephalitis or meningitis).
- Metabolic (hypoxia, hypercapnia, hypoglycaemia, CO poisoning, dialysis).
- Craniofacial disorder (pathology of skull, neck, eyes, nose, ears, sinuses, teeth, mouth, temporomandibular joint dysfunction).
- Neuralgias (trigeminal, occipital, and other cranial nerves).

Approach

Use a detailed history and examination (including vital signs and neurological examination) to search for potentially serious causes. Look particularly for the following (some typical features in brackets):

- Subarachnoid haemorrhage (sudden, severe onset, syncope)—see Subarachnoid haemorrhage, pp. 134–5.
- Meningitis or encephalitis (fever, neck rigidity, \pm indwelling ventriculoperitoneal shunt)—see Meningitis, pp. 232–3.
- Head injury (history or signs of trauma)—see Head injury: introduction, pp. 362–3.
- \uparrow ICP (papilloedema, loss of retinal vein pulsation).
- Stroke (focal neurological signs)—see Stroke, pp. 150–1.
- Acute glaucoma (painful red eye, \downarrow VA, irregular semi-dilated pupil)—see The red eye, p. 558.
- Cranial arteritis (jaw pain, temporal artery tenderness)—see Giant cell arteritis, p. 137.

History

Features suggesting possible serious pathology are:

- Sudden-onset headache.
- Worst headache ever.
- Dramatic change in pattern of headache.
- Known HIV or malignancy.
- Presence of a ventriculoperitoneal shunt.
- Headache coming on during exertion.
- New-onset headache in those aged >50y.

Ask about drugs and the possibility of toxins (eg CO).

Examination

- Check GCS, pulse rate, RR, BP, T°, and SpO₂.
- Feel the head for muscular tenderness, arterial tenderness, and trigger points for neuralgia, and look for evidence of head injury.
- Examine the eyes for VA, pupil reactions, and eye movements. Look at the fundi for papilloedema.
- Palpate the sinuses for tenderness.
- Look in the ears for haemotympanum or infection.
- Check the oral cavity for infection.
- Look for evidence of purpura/rash of meningococcal infection.
- Complete a full neurological examination (include cranial nerves, limb tone, power, sensation, co-ordination, and reflexes).
- Check for Kernig's sign: straightening the knee, whilst the hip is flexed, produces discomfort in the presence of meningeal irritation.

Management

Tailor investigation and emergency treatment according to the presentation, based upon the likely diagnosis.

- Check FBC, ESR, CRP, U&E, and blood glucose.
- If pyrexial and no other obvious source of infection found, take blood cultures and consider cefotaxime 2g IV ± aciclovir. Start IV fluids and refer—a CT head scan may be required (and LP if no sign of ↑ ICP).
- Give paracetamol PO (or IV if vomiting) and an NSAID.
- Consider metoclopramide 10mg IV with IV fluid (eg 1L of 0.9% saline), which can be an effective treatment for some headaches.
- Arrange an emergency CT brain scan for any patient with an acute severe headache or with a history of seizure or an abnormal neurological exam. Adopt a low threshold for CT scan for any patient with HIV.
- Use the Ottawa rule to rule out subarachnoid haemorrhage in patients aged 15–39y (see  Ottawa rule to exclude subarachnoid haemorrhage, p. 134).

It may be safe to discharge home a patient with slow-onset headache that has resolved following treatment, and with a normal examination and blood tests. Advise GP follow-up and to re-attend if symptoms worsen.

Subarachnoid haemorrhage

► Consider subarachnoid haemorrhage in any 'worst ever' or sudden-onset headache.

Atraumatic subarachnoid haemorrhage is an important cause of sudden collapse and death at any age. Most bleeds follow the rupture of saccular ('berry') aneurysms in the circle of Willis (see Fig. 3.30). Other bleeds may be due to arteriovenous malformations, tumours, or connective tissue disorders.

History

Up to 70% of patients with subarachnoid haemorrhage report rapid-onset or 'worst ever' headache. This is classically described as 'like a blow to the back of the head', accompanied by neck pain, photophobia, and vomiting. In 25%, exertional activities precede the event. The patient may present after syncope or fits. Drowsiness and confusion are common. 'Warning headaches' may precede subarachnoid haemorrhage. Unilateral eye pain may occur.

Examination

Document pulse rate, BP, T°, and GCS. An unconscious patient with signs of Cushing's response signifies ↑ ICP. Perform a full cranial and peripheral nerve examination. There may be focal motor and sensory signs due to intracerebral extension of the haemorrhage or vasospasm, subhyaloid haemorrhages (blotchy haemorrhages seen in the fundi), or cranial nerve palsies. Oculomotor nerve palsy is characteristic of a berry aneurysm involving the posterior communicating artery. Neck stiffness is often absent in ED presentations, either because meningeal irritation has not yet occurred or because the patient is deeply unconscious.

Ottawa rule to exclude subarachnoid haemorrhage

This excludes subarachnoid haemorrhage in alert patients aged 15–39y, with new severe atraumatic headache, with maximum intensity within 1hr, who have no neck pain or stiffness, no witnessed loss of consciousness, no onset during exertion, no thunderclap headache (defined as peak within 1s), and no limited neck flexion on examination. The rule does not apply to patients with new neurological deficits, previous aneurysm/bleed, known brain tumour, or chronic recurrent headaches.

Investigations

This may need to proceed alongside resuscitation in seriously ill patients:

- Assess ABC. If the patient is unconscious, open the airway and contact ICU. Consider urgent RSI, tracheal intubation, and IPPV.
- Obtain venous access, and check BMG, FBC, clotting screen, and U&E.
- CXR may show changes of neurogenic pulmonary oedema.
- ECG may demonstrate ischaemic changes.
- Modern high-resolution CT scanning within 6hr will identify >98% of subarachnoid haemorrhages. If the scan does not show a bleed, but the patient is deemed to be at high risk, admit for LP and CSF analysis (to be done >12hr after headache onset).

Once diagnosed on plain CT, involve the neurosurgical team and consider the need and timing for a CT angiogram. It may be useful to use the Hunt and Hess score (see Table 3.13) when communicating the severity by phone.

Table 3.13 Hunt and Hess scale for subarachnoid haemorrhage

Grade	
1	Asymptomatic, mild headache, slight nuchal rigidity
2	Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy
3	Drowsiness/confusion, mild focal neurological deficit
4	Stupor, moderate to severe hemiparesis
5	Coma, decerebrate posturing

Treatment

Tailor this according to the presentation and the need for resuscitation:

- Give O₂ as required.
- Provide adequate analgesia and antiemetic. Codeine (30–60mg PO), paracetamol (1g PO), and/or NSAID may suffice. Some patients require more potent analgesics (eg morphine titrated in 1mg increments IV, according to response)—proceed slowly to avoid drowsiness.
- If unconscious (GCS <8), severely agitated, or combative, tracheal intubation (with GA) will allow IPPV and control of pCO₂ to within normal levels. Insert a urinary catheter and an arterial line.

Contact the neurosurgical team—further treatment options include:

- Nimodipine (60mg PO every 4hr or 1mg/hr IV) to prevent and treat ischaemic neurological deficits secondary to vasospasm.
- Mannitol IV (eg 200mL of 10%) if there is evidence of ↑ ICP.

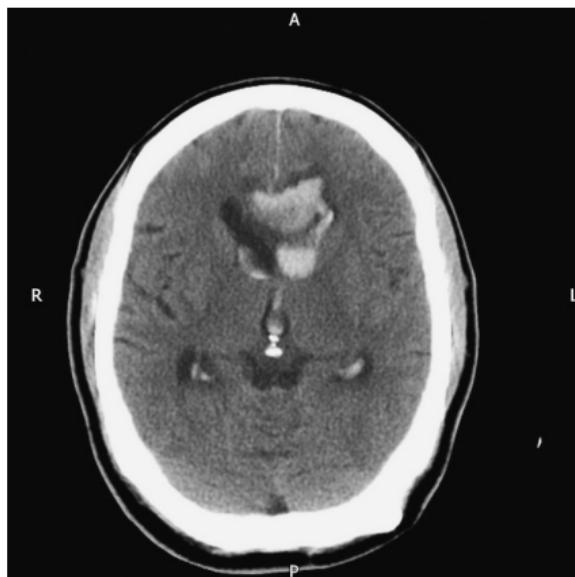


Fig. 3.30 CT showing acute subarachnoid haemorrhage following rupture of berry aneurysm.

Migraine

Patients with recurrent migraine rarely attend the ED unless symptoms are different from usual—take care to avoid missing more serious conditions. The pathogenesis of migraine is not entirely clear, but there is initial vasoconstriction and subsequent vasodilatation of both intracranial and extracranial blood vessels.

Presentation

Precipitants include fatigue, alcohol, menstruation, oral contraceptive pill (OCP), hunger, chocolate, cheese, shellfish, and red wine.

A *prodrome* lasting 5–30min occurs in a third of patients, with blurred vision, photophobia, or scintillating scotomata (an area of blurred or absent vision surrounded by moving zigzag lines), malaise, anorexia, and vomiting. A few experience hemiparaesthesiae, mild unilateral weakness, ataxia, or dysphasia. The following headache may last 4–72hr and is usually ‘throbbing’ and unilateral, but may be generalized. Photophobia, nausea, or phonophobia are common.

Rare forms of migraine

Hemiplegic migraine Profound hemiplegia precedes the development of the headache by 30–60min. Weakness and other focal deficits usually resolve quickly. Occasionally, they may be slow or fail to resolve.

Basilar migraine Brainstem disturbances, with impaired consciousness, vertigo, dysarthria, diplopia, and limb weakness.

Ophthalmoplegic migraine Transient unilateral ophthalmoplegia and ptosis, which may last several days.

Acephalic migraine Very occasionally, neurological defects may be present without headache.

Examination

Look for evidence of other serious diagnoses.

Treatment of acute attacks

- Give simple analgesia (eg paracetamol 1g PO PRN qds or soluble aspirin 600–900mg PO or an NSAID), in combination with a 5HT₁ agonist (eg sumatriptan 50–100mg PO or 6mg SC). Oral triptans are not licensed in patients under 18y—consider nasal sumatriptan instead for patients aged 12–17y. Triptans cause vasoconstriction and are contraindicated in IHD, uncontrolled hypertension, and basilar and hemiplegic migraine. Rebound headache may occur in up to 45%—offer a second dose of triptan if there is initial improvement but then relapse within 2–4hr. Advise GP follow-up for all patients treated with a triptan.
- Consider an antiemetic (eg metoclopramide 10mg PO or IV).
- Refer for admission patients who have neurological signs or altered mental status or where there is diagnostic uncertainty (including a change in severe headache pattern).
- Avoid ergotamine and opioids (see  <https://www.nice.org.uk>).

Giant cell arteritis

Also known as 'temporal arteritis' or 'cranial arteritis' (see  Giant cell (temporal) arteritis, p. 557).

Consider this in all patients >50y with a recent onset of headache or a change in headache pattern. There may be weight loss, night sweats, low-grade fever, jaw claudication, and ↓ vision (up to 10% present with acute visual loss), shoulder girdle stiffness, and muscular aches (polymyalgia). Involvement of carotid or vertebral arteries may lead to TIAs or stroke.

Examination The temporal arteries may be tender, reddened, pulseless, or thickened. Fundoscopy is usually normal, but papilloedema can occur later in the disease.

Investigation ↑ CRP and/or ↑ ESR >> 40mm/hr, often with low-grade anaemia and leucocytosis. A normal CRP/ESR does not exclude temporal arteritis—a minority (10%) with the condition will have normal markers at presentation.

Treatment In view of the serious risk of rapidly progressive visual loss, if suspected, give hydrocortisone 200mg IV (or prednisolone 40mg PO) immediately. Refer to the neurologist or ophthalmologist as an emergency—the diagnosis may be confirmed by temporal artery biopsy.

Space-occupying lesions

If the headache is always located on the same side, consider space-occupying lesions and arteriovenous malformations. Headaches that are dull, aching, and made worse by lying down or straining are typical of space-occupying lesions. Space-occupying lesions include primary tumours, metastases (see Fig. 3.31), aneurysms, haematomas, and abscesses. They may present with personality change, seizures, focal neurological signs ± ↑ ICP.

Diagnosis is apparent on CT scan. Provide analgesia (eg paracetamol and codeine), as required. Liaise with the neurosurgeon—if there is associated cerebral oedema, give dexamethasone 4mg PO and discuss the need for mannitol (eg 0.5g/kg IVI over 20min).

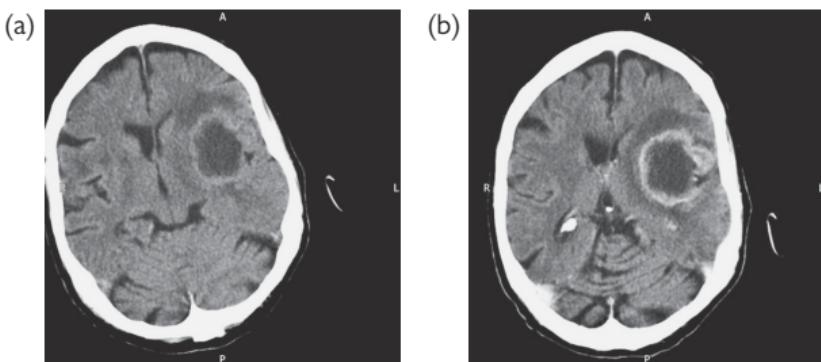


Fig. 3.31 CT showing cerebral metastasis from a lung primary (a), 'ring-enhancing' with contrast (b).

Other causes of headache

Cluster headache

These are more common in men. Often there is a family history. Headache usually occurs at night, waking the patient. Sometimes alcohol may act as a precipitant. Headaches are typically 'clustered' into up to eight attacks per day, each lasting between 15 and 180min. Pain is usually severe, centred upon the eye. Associated symptoms, often unilateral, include conjunctival injection, lacrimation, nasal congestion, rhinorrhoea, forehead and facial sweating, miosis, and ptosis.

Treatment High-flow O₂ (12L/min via reservoir mask) for 15min sometimes provides relief. Otherwise, use paracetamol/NSAID. Consult before contemplating starting ergotamine or sumatriptan.

Trigeminal neuralgia

Characterized by stabbing unilateral pain within the distribution of the trigeminal nerves. Stimulation of the 'trigger area' (eg by touching, hairbrushing, or even chewing) induces very severe pain. Treat with carbamazepine and oral analgesia. Admit if the pain is severe and unrelieved.

Malignant hypertension

Hypertension is an unusual cause of headaches but is seen in patients with malignant hypertension and diastolic BP >130mmHg (see  Hypertensive problems, p. 94).

Ventricular shunts

Assume that any patient who presents with headaches associated with a ventricular shunt has infection/blockage and refer as an emergency. Associated drowsiness is a particular pointer to blockage.

Send bloods (including WCC and CRP) and request a CT scan.

CSF spinal leak headache

Headache may occur in the first few days after a spinal or epidural anaesthetic as a result of a CSF leak. Provide simple analgesia for mild symptoms. Patients with more severe headache may need treatment in the form of an injected 'blood patch' (contact the anaesthetic team).

Analgesic headache

Chronic use of simple analgesics, sympathomimetics, ergotamine, or cocaine is associated with headaches. Stopping or starting certain medications (eg OCP) can also cause headache, as can withdrawal from caffeine. Exclude serious causes and advise GP follow-up with advice on medication use.

Cerebral venous thrombosis

More common than previously realized. It presents in a similar fashion to subarachnoid or subdural haemorrhage—sudden-onset headache with nausea and vomiting. It may be associated with sinus infections, pregnancy, and the post-partum period. Presenting features partly reflect the sinus involved (most commonly the sagittal sinus or transverse sinus). Cerebral venous thrombosis may not be apparent on initial plain CT but can be diagnosed on contrast CT or MRI. Treat with heparinization and refer for admission.

Idiopathic intracranial hypertension

Previously known as 'benign intracranial hypertension'. More common in women with a high BMI. ↑ ICP can cause permanent damage to the optic nerve and result in blindness if not treated. Presenting symptoms and signs reflect ↑ ICP: headache (worse on coughing/sneezing), vomiting, cranial nerve palsies, and papilloedema. The diagnosis is made by a combination of a normal CT scan with ↑ opening pressure on LP.

Meningitis See  Meningitis, pp. 192–3.

Encephalitis See  Acute encephalitis, p. 234.

Miscellaneous causes

Headaches may also result from:

- Hypoxia and hypercapnia.
- Poisons, eg CO and solvents (see  Carbon monoxide poisoning, p. 216).
- Drugs, eg nitrates, sildenafil, and after alcohol ('hangover').
- Post-traumatic (see  Post-concussion symptoms, p. 376).
- Glaucoma (see  The red eye, p. 558).
- Sinusitis (see  Paranasal sinusitis, p. 571).

Tension headache

The diagnosis is only made after exclusion of more serious pathology.

The history may be described in a dramatic manner. The headache is usually continuous, pressing, or tight ('band-like') in nature. It is usually bitemporal or occipital. Usual features of migraine are absent and the headache does not worsen with exertion.

Examination often reveals pericranial muscle tenderness but is otherwise normal.

Treat with simple analgesia (eg paracetamol 1g PO qds PRN) and advise GP follow-up. Reassure the patient that a thorough history and examination have not revealed any worrying features.

Acute confusional state (delirium)

Definition of delirium

Delirium is a form of organic brain syndrome characterized by:

- Disturbed conscious level and mood (overactivity, excitement, drowsiness, or stupor).
- Global disturbance of cognition (memory, orientation, attention, speech, motor function).
- Rapid onset with fluctuating course (often worse at night, with reversal of usual sleep–wake cycle) and brief duration.
- Perceptual distortions and hallucinations (especially visual).

Causes of acute confusion

One or more of the following may be the underlying cause of an acute confusional state (several causes frequently coexist):

- *Prescribed medication*: digoxin, cimetidine, steroids, analgesics, diuretics, anticholinergics, antiparkinsonian drugs.
- *Drugs of abuse*: opioids, benzodiazepines, ecstasy, amphetamines, hallucinogens.
- *Withdrawal*: from alcohol, opioids, hypnotics, or anxiolytics.
- *Infection*: pneumonia, urinary tract infection (UTI), septicaemia, meningitis, encephalitis.
- *Metabolic*: hypoxia, hypercapnia, hypoglycaemia, acidosis, hyponatraemia, hypercalcaemia.
- *Cardiac*: acute MI, cardiac failure, endocarditis.
- *Neurological*: head injury, chronic subdural haematoma, meningitis, post-ictal state.
- *Organ failure*: respiratory, renal, and hepatic failure.
- *Endocrine*: myxoedema, thyrotoxicosis, diabetes, Addison's disease.

Differential diagnosis

Delirium can occur at any age but is much more common in the elderly. It is often misdiagnosed as schizophrenia, depression, or dementia (see  Dementia, p. 141). Differentiation can be difficult, but the following are more suggestive of physical illness:

- Non-auditory hallucinations.
- Dysarthria.
- Ataxia.
- Gait disturbance.
- Incontinence.
- Focal neurological signs.

Approach

Search systematically for (and exclude) the physical causes of acute confusion outlined above.

Investigation of acute confusion

Perform a thorough, careful physical and mental state examination (see  Mental state examination, pp. 622–3) on acutely confused patients. It may be impossible to obtain an accurate history from the patient, so actively seek other sources of information: relatives, carers, GP, and previous medical records.

Look for evidence of alcohol/drug intoxication or withdrawal states. Examine for focal neurological signs and acute cardiac, respiratory, or abdominal abnormalities (including acute urinary retention). Document basic vital signs (GCS, pulse, BP, RR, and T°) in all cases.

Mandatory basic investigations

- BMG.
- U&E, FBC, and blood glucose.
- Urinalysis.
- SpO₂ and ABG.
- ECG.
- CXR.

Adopt a low threshold for additional tests based on clinical suspicion—blood cultures, thyroid function tests (TFTs), serum digoxin, paracetamol and salicylate, CT brain scan, and even LP may be indicated.

Be *careful not to miss*: hypoglycaemia, head injury, Wernicke's encephalopathy, opioid intoxication, acute alcohol withdrawal, and CO poisoning.

Dementia

Dementia is an acquired, progressive decline in intellect, behaviour, and personality. It is irreversible and typically occurs with a normal level of consciousness. Note that patients with dementia are at risk of delirium resulting from an acute infective or metabolic origin—a clue to this may be an acute deterioration in mental state.

The *most common causes of dementia* are Alzheimer's disease, vascular dementia, and Lewy body dementia.

Transient global amnesia

This curious and poorly understood condition is probably more common than generally appreciated. It is characterized by sudden unexplained memory loss in a middle-aged/elderly patient, not accompanied by any other neurological or other abnormalities (such as weakness). The patient is able to follow simple instructions but may appear bewildered and have poor short-term memory. Family members may also be understandably distressed. Investigations do not reveal any abnormality. Often mistaken for delirium or stroke/TIA, transient global amnesia is characterized by the way that memory is so dramatically affected, but without other signs being present. Spontaneous recovery within 24hr is the norm. Transient global amnesia is not a particular predictor of future stroke or other vascular event—advise GP follow-up.

The unconscious patient: 1

Common causes

- Hypoglycaemia.
- Drug overdose.
- Head injury.
- Stroke.
- Subarachnoid haemorrhage.
- Convulsions.
- Alcohol intoxication.

Uncommon causes

- Type II respiratory failure.
- Cardiac failure.
- Arrhythmias.
- Hypovolaemic shock.
- Anaphylaxis.
- Hepatic/renal failure.
- Hypo-/hyperthermia.
- Meningitis/encephalitis.
- Malaria.
- DKA/hyperosmolar hyperglycaemic state (HHS).
- Non-convulsive status epilepticus.
- Wernicke's encephalopathy.

Treatment may be needed before any diagnosis is made. Remember:

- Airway.
- Breathing.
- Circulation.

Initial resuscitation

Airway and cervical spine Whatever the cause of coma, a patient may die due to airway obstruction, respiratory depression, or circulatory failure. Clear and protect the airway immediately, and immobilize the cervical spine if trauma is suspected. Arrange intubation if no improvement.

Breathing If breathing is inadequate, ventilate with O₂ using a self-inflating bag with an O₂ reservoir. An uninjured patient who is breathing adequately can be examined supine, but nurse in the recovery position to ↓ the risk of airway obstruction. Record the RR.

Circulation Measure pulse and BP. Observe and feel the skin for colour, sweating, and T°. Obtain reliable venous access. Monitor ECG. Replace IV fluid if indicated.

Conscious level Assess the level of consciousness using GCS (see  Head injury: examination, pp. 368–9). Check blood glucose (initially by BMG) and treat hypoglycaemia immediately (see  Hypoglycaemia, pp. 158–9). Record pupil size. Give slow IV thiamine (i.e. two pairs of Pabrinex® ampoules in 100mL of 5% glucose over 30min—see the (BNF) to patients with a history of alcoholism or who appear malnourished.

History

Obtain a history from the ambulance crew and the patient's relatives and friends. Ask:

- How was the patient found?
- When was he/she last seen?
- Is there any suggestion of trauma?
- Is there any history of fits?
- Has there been recent foreign travel?
- Previous symptoms and medical history (including depression).
- Note any drugs available.

Check previous ED records and hospital notes.

Examination

Examine thoroughly for illness and injury. Check clothes and possessions for tablets and cards/bracelets warning of pre-existing disease.

- ↑ RR may reflect obstructed airway, aspiration, pneumonia, DKA, liver/renal failure, salicylate poisoning, methanol, or ethylene glycol.
- Respiratory depression may be due to poisoning (eg barbiturates, opioids, tricyclics) or ↑ ICP. Brainstem compression or damage by stroke may cause rapid, irregular, or intermittent (Cheyne–Stokes) breathing.
- If bradycardic, consider: hypoxia, complete heart block, ↑ ICP, digoxin or β-blocker poisoning (see ↗ Beta-blocker poisoning, p. 206).
- If tachycardic, consider: airway obstruction, hypoxia, hypovolaemia, SVT, VT, or anticholinergic overdose.
- AF may be associated with cerebral emboli.
- Hypotension suggests hypoxia, shock (hypovolaemic, anaphylactic, septic), or poisoning.
- Hypertension may be due to ↑ ICP.
- Skin: look for pallor, cyanosis, jaundice, spider naevi, skin crease/scar pigmentation (Addison's disease), rashes (eg purpura in meningococcal infection or (DIC), injection marks (drug addiction or medical treatment), and signs of trauma. Erythema or blistering over pressure points indicate the patient has been unconscious for some hours.
- Measure rectal T° with a low-reading thermometer if the skin feels cold. Coma is common at $<30^\circ\text{C}$ (see ↗ Hypothermia: presentation, pp. 264–5).

The unconscious patient: 2

Neurological examination includes GCS, limb strength, muscle tone and reflexes, optic fundi, eardrums, neck stiffness (except in neck injury), and palpation of the fontanelle in babies. Lateralizing signs, such as facial or limb weakness, may be caused by a stroke, intracranial bleeding, or pre-existing problems (eg previous stroke or Bell's palsy). Ocular nerve palsy or divergent squint with coma can indicate Wernicke's encephalopathy, requiring IV thiamine, or tricyclic poisoning. Look for subtle signs of seizure activity (eg twitching of ocular muscles or eyelids, unusual limb movements), which may indicate non-convulsive status epilepticus. Look at the fundi—spontaneous central retinal venous pulsations are rare with ↑ ICP. Subhyaloid haemorrhages (blotchy fundal haemorrhages) suggest subarachnoid haemorrhage.

Hypoglycaemia can cause localized weakness/coma and mimic stroke.

Coma without lateralizing signs is usually due to poisoning, a post-ictal state, brainstem stroke, or hepatic failure—extensor plantar reflexes are common in these conditions.

Tricyclic antidepressants often cause coma with dilated pupils, a divergent squint, ↑ muscle tone, jerky limb movements, and extensor plantars. In severe poisoning, there may be muscle flaccidity with respiratory depression and ↓ reflexes (see ↗ Tricyclic antidepressant poisoning, pp. 202–3).

Coma with small pupils and respiratory depression suggests opioid poisoning (see ↗ Opioid poisoning, p. 196). In unexplained coma, give a therapeutic trial of naloxone (0.4–0.8mg IV), observing for changes in conscious level, RR, and pupil size.

Investigations

- BMG and blood glucose. If BMG is low, do not wait for the laboratory result to confirm this before starting treatment.
- VBG/ABG (record FiO_2 and whether breathing spontaneously or with IPPV).
- FBC, prothrombin time, U&E.
- Check paracetamol and salicylate levels if poisoning is suspected—paracetamol alone does not cause coma (except in late cases with liver failure), but a mixture of drugs may have been taken. Do not routinely send blood for drug screening for sedatives/hypnotics, but in unexplained coma, keep blood for later analysis if necessary.
- ECG may show arrhythmias (see ↗ Tricyclic antidepressant poisoning, pp. 202–3).
- CXR may show pneumonia, aspiration, trauma, or tumour.
- CT scan will identify subarachnoid haemorrhage, stroke, or head injury.

Psychogenic coma

Patients sometimes pretend to be unconscious. It can be difficult to be certain of this—exclude other causes first. Suspect psychogenic coma if serious pathology has been excluded, and when the eyes are opened, only the sclerae show as the eyes deviate upwards (Bell's phenomenon).

Falls in the elderly

With an increasingly elderly population living in more isolated, and often precarious, situations, it is not surprising that many elderly present to the ED following a fall. In addition to the standard approach, pay particular attention to the following questions.

What caused the fall?

Trying to distinguish between a trip/stumble, a dizzy spell, or medical collapse can be very difficult. Basic observations, lying/standing BPs, ECG, and BMG act as basic screening, but be aware that a medical problem responsible for the fall may not be immediately apparent.

What injuries resulted?

The classic injury resulting from a fall in an elderly individual who is then unable to get up after is a hip fracture—adopt a low threshold for requesting an X-ray of the pelvis. Fractures of the pubic rami (see Pelvic fractures, pp. 480–1) are also common as a result of a fall (see Fig. 3.32) and can tip the balance in terms of whether a patient is safe to discharge home. Falls, especially down steps or stairs, can result in significant head and neck injuries which are not always obvious at first.

Did the patient lie for a long period?

Be aware that as a result of being unable to get up after a fall, the patient may have experienced a 'long lie', with attendant risks of hypothermia, dehydration, pressure sores, and muscle damage (with rhabdomyolysis, hyperkalaemia, and AKI).

Is it safe to consider discharge home?

Having established that the patient does not have medical problems requiring treatment and hospital admission, determining whether a patient is safe to discharge is very often a complex issue. It requires input from a number of sources, including the patient, relatives, GP, and other specialists (see Discharging the elderly patient, p. 23).



Fig. 3.32 Right-sided pubic ramus fractures in an elderly patient with previous hemiarthroplasty.

Collapse and syncope

Syncope is a sudden, transient loss of consciousness, with spontaneous recovery. If a patient suddenly loses consciousness in the ED, assess responsiveness and check for a pulse. Keep the airway clear; give O₂, and monitor pulse and ECG. Note any neurological signs during the episode, and obtain BP, SpO₂, and BMG.

Priorities

- Identify serious or life-threatening problems and institute treatment.
- Decide which patients require admission.
- Decide which patients require follow-up.

History of syncopal episode

Was it a simple faint? Vasovagal or neurally mediated syncope is a common response to an overwarm environment or prolonged standing, and can be precipitated by sudden fright or visual stimuli (eg the sight of blood). Other contributors are large meals, prolonged starvation, or alcohol. There are usually premonitory symptoms of feeling unwell, nauseated, dizzy, or tired, with yawning, blurred or 'tunnel' vision, or altered hearing. If the fainter cannot get supine (eg bystanders keeping them upright), seizure-like twitching may occur (*convulsive syncope*). Vomiting and incontinence may occur and do not reliably discriminate seizures from faints.

Was it a seizure? Look at the ambulance records. An eyewitness account is crucial. Ask what the witnesses *actually saw* (do not assume they know what a 'fit' looks like). There should typically be no prodrome, and there is often a cry followed by tonic/clonic movements. Cyanosis, saliva frothing from the mouth, heavy breathing, tongue biting, or incontinence suggest a generalized seizure. Post-ictal drowsiness or confusion is normal—very rapid recovery questions the diagnosis.

Was it a cardiac event? Cardiac syncopal events are also abrupt in onset (eg collapse due to HCM) and may be accompanied by pallor and sweating. Recovery may be rapid, with flushing and deep/sighing respiration in some cases (eg Stokes–Adams attacks). Nausea and vomiting are not usually associated with syncope from arrhythmias. Ask about previous episodes and chest pain, palpitations, history of cardiac disease, and family history of sudden death. Syncope associated with exertion is a worrying feature—possible causes include aortic or mitral stenosis, pulmonary hypertension, cardiomyopathy, or coronary artery disease.

Other causes Carotid sinus syncope is neurally mediated and often occurs with shaving or turning the head. Syncope may be secondary to the effects of medication (eg GTN, β-blockers, antihypertensive drugs). Syncope may also be the presenting feature of subarachnoid haemorrhage, ruptured ectopic pregnancy, aortic or carotid dissection, PE, or GI bleed. Syncope is rarely caused by a TIA.

Assessment and treatment

Obtain a detailed account from the patient and witnesses. Look for signs of tongue biting, incontinence, or other injuries, and examine the cardiovascular system (CVS) for murmurs, arrhythmias, or abnormalities. Perform a neurological examination and look for focal signs. Do postural tests (supine and standing or sitting pulse and BP). A degree of postural hypotension is common, but postural symptoms (eg dizziness, weakness) are always significant (look for causes of hypovolaemia, eg GI bleed, ectopic pregnancy). Check BMG to exclude hypoglycaemia, and an ECG looking for arrhythmias, LVH, ischaemia, previous or acute MI, and QT prolongation. An abnormal ECG may be the only clue to an underlying HCM or Brugada syndrome (various ECG patterns, including ST elevation in V_{1-3} and RBBB).

Disposal

Admit patients for cardiology review within 24hr if they present with:

- An ECG abnormality.
- Heart failure.
- Loss of consciousness on exertion.
- Family history of sudden death <40y or an inherited cardiac condition.
- New or unexplained breathlessness.
- A heart murmur.

Treat patients as if they have had a 'first fit' (see  Seizures and status epilepticus, pp. 156–7) if they present with one or more of:

- A bitten tongue.
- Amnesia, unresponsiveness, unusual posturing or prolonged limb jerking, head turning to one side.
- History of an aura.
- Post-ictal confusion.

Aim to discharge patients who have made a full recovery and have an appropriate history for vasovagal syncope and a normal examination. Consider suggesting to the GP to arrange outpatient ECG monitoring/investigation if discharging a patient >65y with unexplained syncope.

(See NICE guideline CG109 at  <https://www.nice.org.uk>)

Diagnoses not to be missed

- *GI bleed*: syncope (\pm postural symptoms) indicate significant blood loss and hypovolaemia. Perform PR examination to check for blood/melaena.
- *Ectopic pregnancy*: suspect this in women with syncope and abdominal pain or gynaecological symptoms. Do a pregnancy test.
- *Ruptured abdominal aortic aneurysm*.
- *PE* (see  Pulmonary embolism, pp. 124–5). A witness may give a history of cyanosis. Indicative of massive thrombus.

Acute generalized weakness

Weakness may be a feature of common neurological problems (eg TIA/stroke) or accompany many of the causes of collapse (see Collapse and syncope, pp. 146–7). Less commonly, generalized muscle weakness may be the presentation of a number of other diseases.

Clinical features which may help to distinguish between upper and lower motor neurone lesions are shown in Table 3.14.

Table 3.14 Distinguishing between upper and lower motor neurone lesions

Feature	Upper motor neurone	Lower motor neurone
Wasting	No	Yes
Fasciculation	No	Yes
Tone	↑	↓
Power	↓	↓
Reflexes	↑	↓
Plantars	Upgoing	Downgoing

Guillain–Barré syndrome

Guillain–Barré syndrome follows a respiratory or GI infection and is characterized by progressive symmetrical weakness, spreading from distal muscles to involve proximal muscles. Symptoms and signs include muscle tenderness, back pain, loss of muscle reflexes, sensory symptoms (paraesthesiae of fingers and toes), and disturbance of the autonomic nervous system (hyper- or hypotension, tachy- or bradycardia, bladder atony). Beware respiratory failure, which can rapidly progress to respiratory arrest. Serial vital capacity measurements are advised. Refer to the medical team/ICU.

Multiple sclerosis

This demyelinating disease of the central nervous system (CNS) is more common in ♀ and usually presents at 20–50y. It follows a relapsing and remitting course with sensory loss, stiffness, weakness of legs, ataxia, autonomic impairment (bladder dysfunction), and diplopia. Patients may present with these symptoms during their first exacerbation or with optic neuritis (pain in one eye, with visual blurring and ↓ VA). Admit under neurology. If there are eye symptoms, arrange urgent ophthalmology review.

Polymyositis

Polymyositis is an inflammatory myopathy that presents with symmetrical proximal muscle weakness, arthritis, and sometimes muscular tenderness. Patients report difficulty climbing stairs, standing from a low chair, or lifting arms to brush hair. Creatine kinase (CK) levels are raised. Refer to a rheumatologist for treatment.

Myasthenia gravis

This is a rare autoimmune disease with antibodies to the nicotinic acetylcholine receptors. Crises can be precipitated by infection, with painless weakness in which the muscles are fatigable, but tendon reflexes and pupil responses are normal. Usually, cranial nerves are involved to a greater extent than limb muscles and the distribution is asymmetrical. Ptosis, diplopia, and blurred vision are the most common presentations, which can be treated with pyridostigmine. Crises may present with severe muscle weakness when the major concern relates to respiratory compromise. The patient may require emergency RSI using rocuronium for paralysis. ICU treatment includes plasmapheresis.

As a diagnostic adjunct in the ED, placing an ice pack over the eyelids improves ptosis.

Patients with known myasthenia gravis may present with weakness due to under-treatment or over-treatment (cholinergic crisis) or as an adverse reaction to an unrelated drug. Refer to the medical team for investigation.

Periodic paralysis

This encompasses a family of hereditary diseases associated with defects in muscle ion channels. Episodes of weakness can be associated with fluctuations in serum K⁺ levels, lasting a few hours to a week. On occasions, this is associated with eating a large meal. Patients may develop myotonia between attacks and fixed proximal muscle weakness. Treatment tends only to be required for hypokalaemic periodic paralysis with oral K⁺ supplementation.

Wound botulism

Botulism has made a comeback in the IV drug-injecting community. Botulinum toxin inhibits the release of acetylcholine at neuromuscular junctions and sympathetic and parasympathetic synapses. Wound infection with *Clostridium botulinum* presents with diplopia, blurred vision, ptosis, and neck weakness, which can progress to respiratory failure. Treatment is with anti-toxin, benzylpenicillin, and metronidazole, along with respiratory support.

Note that generalized weakness may also be caused by:

- Spinal cord compression.
- Tetanus.
- Alcoholic myopathy.
- Diphtheria.
- Lead poisoning.

Stroke

A stroke is an acute onset of a focal neurological deficit of vascular origin which lasts >24hr. The blood supply to the brain has two sources—the internal carotid and the basilar arteries. The internal carotids supply the anterior and middle cerebral arteries, known as the anterior circulation. The basilar artery supplies the posterior cerebral artery in 70% of people (the posterior circulation). Anterior and posterior communicating arteries in the circle of Willis provide collateral circulation in cases of carotid artery stenosis.

Pathogenesis

70% of strokes occur in those aged >70y, but they can occur at any age. Cerebral infarction (80%) results from:

- Thrombosis secondary to atherosclerosis, hypertension, and arteritis.
- Cerebral embolism from AF, valve disease/replacement, post-MI, ventricular aneurysm, myxoma, endocarditis, or cardiomyopathy.
- An episode of hypoperfusion.

Cerebral haemorrhage (20%) is associated with:

- Hypertension (rupture of small arteries in the brain).
- Subarachnoid haemorrhage (see  Subarachnoid haemorrhage, pp. 134–5).
- Arteriovenous malformations.
- Intracranial tumours
- Bleeding disorders (including anticoagulants) and intracranial tumours.

Presentation

Stroke preceded by neck pain may indicate carotid/vertebral artery dissection or subarachnoid haemorrhage. Headache is an unusual presentation of ischaemic stroke and may indicate cerebral haemorrhage. Be alert to the possibility of different pathology requiring urgent treatment (eg hypoglycaemia, Todd's paresis, hemiplegic migraine, meningitis, encephalitis, brain abscess, head injury, Bell's palsy, 'Saturday night palsy', tumours).

Undertake a thorough examination, including:

- Assessment of mental status/GCS and signs of meningeal irritation.
- Examination of pupils, fundi, and cranial nerves.
- Assessment of motor function (tone, power, and reflexes).
- Assessment of sensory function (including speech and comprehension).
- Examination for cerebellar signs (co-ordination, speech).
- Record initial NIHSS (National Institutes of Health Stroke Scale).
- Check for sources of embolism (AF, murmurs, carotid bruits).

Localization on clinical grounds alone can be difficult, and differentiation between infarction and haemorrhage requires CT/MRI. NICE recommends use of the ROSIER score to identify patients presenting with acute stroke (see Table 3.15). The ROSIER score will pick up the majority of patients who are having a stroke but may not identify patients with posterior circulation infarcts.

Table 3.15 ROSIER score for stroke recognition

Criteria	Points
Facial weakness (asymmetrical)	1
Arm weakness (asymmetrical)	1
Leg weakness (asymmetrical)	1
Speech disturbance	1
Visual field defect	1
Loss of consciousness or syncope	-1
Seizure	-1

Stroke is unlikely if score is 0 or lower.

Investigations

Examine and investigate first to exclude other conditions, and second to confirm the diagnosis of stroke. As a minimum requirement: BMG, FBC, ESR, U&E, blood glucose, ECG, and CXR. Apply a pulse oximeter (if $\text{SpO}_2 < 94\%$, consider ABG) and a cardiac monitor.

Arrange emergency non-enhanced CT scan where:

- Stroke thrombolysis or thrombectomy may be indicated. If considering thrombectomy, also perform CT contrast angiography.
- The patient is on oral anticoagulant and/or has a bleeding tendency.
- The GCS is <13.
- There are unexplained progressive or fluctuating symptoms.
- There is papilloedema, neck stiffness, or fever.
- There was a severe headache at the onset of symptoms.

Initial management

Quickly decide if emergency thrombolysis or thrombectomy is indicated—if it is, involve the stroke service and follow protocols (see Stroke thrombolysis, p. 152).

If the CT scan reveals a bleed, treat accordingly (see Intracerebral haemorrhage, p. 154).

If emergency thrombolysis is not indicated and the CT shows no bleed:

- Immediately correct hypoglycaemia if present (see Hypoglycaemia, pp. 158–9).
- If hypoxic, give O_2 —aim for SpO_2 of 90–94% (see Oxygen, p. 99).
- Screen the patient's ability to swallow (try a teaspoon of water). If unable to safely swallow, prescribe maintenance IV fluids.
- Give aspirin 300mg as soon as possible—PO, or if unable to swallow, PR. Give a proton pump inhibitor (PPI) (eg omeprazole) if previous dyspepsia. If allergic to aspirin, give an alternative antiplatelet drug (eg clopidogrel).
- Do not routinely give anticoagulants or start statins in the ED.
- Hypertension and labile BP are common in the early post-stroke period. Do not attempt to reduce the BP at presentation, unless there is: aortic dissection, pre-eclampsia/eclampsia, hypertensive encephalopathy/nephropathy/cardiac failure/MI, or in some patients with intracerebral bleed.
- Get specialist advice if vertebral artery dissection is suspected.
- Admit directly to a stroke unit.

Stroke thrombolysis

A treatment which has generated a lot of debate but is in widespread use, stroke thrombolysis benefits some patients but carries a risk of life-threatening haemorrhage. Many departments have protocols which offer thrombolysis to patients aged >18y within 3hr of onset (and up to 4.5hr after onset for those aged 18–80y) for significant stroke symptoms which are not improving, provided CT shows no bleed (see Fig. 3.33) and there are no contraindications.

Deciding whether to thrombolyse can be difficult—defer to senior staff, with involvement of the patient. It is particularly difficult to plan treatment for patients who have relatively minor symptoms or present slightly later or whose symptoms are improving but not resolved.

Contraindications/clinical exclusions to stroke thrombolysis

- Awoke with symptoms/time of onset unknown.
- Seizure at onset.
- Clinical presentation suggestive of subarachnoid haemorrhage.
- Known bleeding diathesis or low platelets ($<100 \times 10^9/L$).
- Arterial puncture at non-compressible site or LP within past 7 days.
- GI or urinary tract haemorrhage in the past 3 weeks.
- Head injury, intracranial surgery, or stroke in the past 3 months.
- Any previous intracranial haemorrhage, brain tumour, arteriovenous malformation, or aneurysm.
- Diastolic BP $>140\text{mmHg}$ (Note: if systolic BP is $>180\text{mmHg}$ and/or diastolic BP is 105–140mmHg, consider thrombolysis if BP reduces to $<180/105\text{mmHg}$ after intervention, eg IV labetalol 10mg IV over 2min, repeated once after 15min if not responding enough).

Procedure for stroke thrombolysis

- Having decided to thrombolyse, time is of the essence.
- Ensure patient comfort; insert two venous cannulae and monitoring.
- Confirm there is no bleeding on the CT brain scan.
- Do not give aspirin.
- Give alteplase (0.9mg/kg, up to a maximum of 90mg) as an IVI over 1hr, with the first 10% as a slow bolus over 1–2min.
- Monitor closely with regular observations. Stop the IVI of alteplase if there is any suspicion of intracranial haemorrhage (new headache, ↓ GCS, acute hypertension, seizure, vomiting) and get a new CT scan. If this shows haemorrhage, liaise with the stroke team/neurosurgeon.
- After administration of thrombolysis, admit under the care of the stroke team to an acute stroke unit. Do not give any anticoagulant or antiplatelet therapy—this will be considered at 24hr if a repeat CT scan at that time shows no bleeding.

Stroke thrombectomy

Evidence is growing to support the use of intra-arterial clot extraction. It is becoming increasingly available in specialist centres—follow local protocols. Consider mechanical thrombectomy if there is proximal intracranial large vessel occlusion causing a disabling neurological deficit (NIHSS ≥ 6) in individuals who have a pre-stroke modified Rankin score of ≤ 3 (in other words, they are not moderately to severely disabled or severely disabled). NICE (2019) outlines situations where thrombectomy may be offered as soon as possible for certain situations:

- Patients within 6hr of symptom onset (together with IV thrombolysis, if not contraindicated and delivered within the licensed time window) with an acute ischaemic stroke, with confirmed occlusion of the proximal anterior circulation demonstrated by CT angiography or magnetic resonance angiography.
- Patients who were last known to be well between 6 and 24hr previously (including those who woke up with symptoms) who have an acute ischaemic stroke and confirmed occlusion of the proximal anterior circulation demonstrated by CT/magnetic resonance angiography, and imaging confirms there is potential to salvage brain tissue.
- Patients who were last known to be well up to 24hr previously (together with IV thrombolysis, if not contraindicated and delivered within the licensed time window), including those who woke up with symptoms, who have an acute ischaemic stroke and confirmed occlusion of the proximal posterior circulation on CT/magnetic resonance angiography, and imaging confirms there is potential to salvage brain tissue.

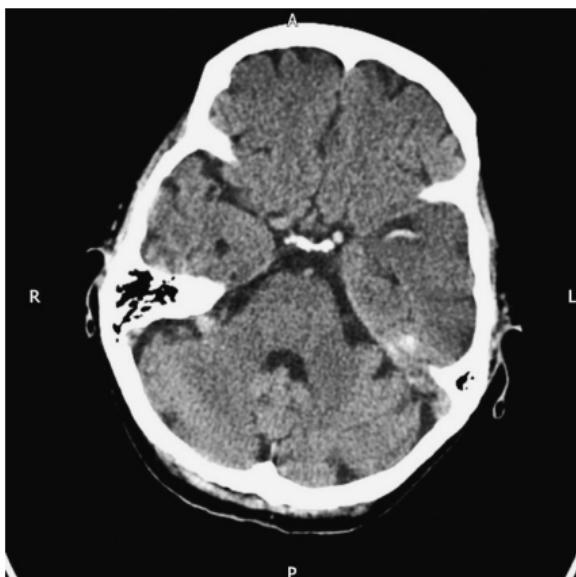


Fig. 3.33 Hyperdense left middle cerebral artery seen on CT scan as an early sign of acute stroke.

Intracerebral haemorrhage

Primary acute intracerebral haemorrhage (haemorrhagic stroke) is responsible for a significant proportion (~10%) of acute strokes and ranges from minor dot haemorrhages to an overwhelming bleed (see Fig. 3.34). Treat supportively and medically in the first instance, and seek advice from the relevant specialist team regarding definitive management. For some patients with a large bleed ± significant comorbidities, palliative measures may be appropriate. For others:

- Aim to urgently reverse any anticoagulation (eg give prothrombin complex concentrate and vitamin K for patients taking warfarin, idarucizumab for those on dabigatran, andexanet for rivaroxaban or apixaban).
- Treat significant hypertension—aim to lower systolic BP to <140mmHg within 1hr. Options to achieve this include IVI of isosorbide dinitrate (eg 2mg/hr, titrating up to 12mg/hr) or IV labetalol (initially 10mg slow IV over 2min, repeated every 2min up to 200mg). Do not rapidly lower BP in patients who have an underlying tumour, aneurysm, arteriovenous malformation, or GCS <6/15, or who are going to have surgical evacuation, or who have a massive haematoma with a poor prognosis.
- Liaise with the neurosurgical team. Surgical intervention is not often indicated.



Fig. 3.34 Large acute left intracerebral haemorrhage, with associated swelling.

Transient ischaemic attacks

A TIA is an episode of transient focal neurological deficit of vascular origin lasting <24hr. A TIA gives major warning for the development of stroke (5% within 48hr, up to 50% in 5y). Even in patients with resolution of symptoms/signs, most have evidence of infarction on CT/MRI.

Presentation

- *Carotid territory involvement* produces unilateral weakness or sensory changes, dysphasia, homonymous hemianopia, or amaurosis fugax.
- *Vertebobasilar territory involvement* produces blackouts, bilateral motor or sensory changes, vertigo, and ataxia.

Causes

Most result from thromboembolic disease involving the heart (AF, mitral stenosis, artificial valves, post-MI) or extracranial vessels (carotid artery stenosis). Other causes are: hypertension, polycythaemia/anaemia, vasculitis (temporal arteritis, PAN, SLE), sickle-cell disease, hypoglycaemia, hypoperfusion (eg arrhythmia, hypovolaemia), and syphilis.

Assessment

To diagnose a TIA, the symptoms must have resolved within 24hr. Document vital signs, and perform a thorough neurological examination. Look for possible sources of emboli, eg arrhythmias (especially AF), heart murmurs, carotid bruits, and MI (mural thrombus).

Investigation

- Check BMG and send blood for FBC, ESR, U&E, glucose, lipids, and INR.
- Record an ECG to search for MI and arrhythmia.
- Many patients who end up being diagnosed as having a TIA are symptomatic when they present to the ED, so they will have a CT brain. For patients whose symptoms have resolved by the time of arrival at the ED, do not request a CT scan unless there is a clinical suspicion of a different diagnosis which CT could identify.

Management

- Give aspirin 300mg PO immediately and continue aspirin until TIA clinic specialist review (which should be in <24hr). The ABCD² score is no longer recommended to risk-stratify patients (<https://www.nice.org.uk>).
- Unless there is a contraindication, start treatment with a statin (eg atorvastatin 20–80mg od at night).
- Liaise with the medical team (and follow local guidelines) for patients with newly diagnosed AF to start anticoagulation, providing CT has excluded haemorrhage and there is no uncontrolled hypertension.

Advice for discharged patients

Provide patients who are discharged after a TIA with verbal and written advice, including not to drive until seen in a TIA clinic and to call for an ambulance if any symptoms of TIA or stroke occur.

Seizures and status epilepticus

First fit

► A first fit has enormous consequences—do not diagnose without good evidence.

A detailed history from both the patient and any witnesses is crucial to the diagnosis. The presence of jerking movements or incontinence does not necessarily reflect epilepsy. Carefully document what was seen, in order to avoid confusion with vasovagal syncope or other types of collapse. Full rapid recovery suggests a syncopal event. Always consider alcohol/drug use, withdrawal states, hypoglycaemia, arrhythmia, head injury, subarachnoid haemorrhage, stroke/TIA, infection (including meningitis), or metabolic disturbance.

As part of the general examination, carefully examine the CNS, documenting: GCS, confusion, focal abnormalities, and findings on fundoscopy. Examine the CVS and check for signs of aspiration.

Todd's paresis May follow seizures—focal deficit or hemiparesis may persist for up to 24hr and indicates a high chance of structural lesion.

Investigations BMG, glucose, FBC, U&E, blood cultures if pyrexial, ECG, and, if there are chest signs, a CXR. Check urine pregnancy test if of child-bearing age. All patients with new-onset seizures need brain imaging at some stage—a significant number have structural CNS abnormalities.

Arrange an emergency CT scan for patients with focal signs, head injury, known HIV, suspected intracranial infection, bleeding disorder (including anticoagulants), or where conscious level fails to improve as expected.

Disposal A patient presenting with a first seizure may be discharged home, accompanied by an adult, if they have normal neurological and cardiovascular examinations, the ECG and electrolytes are normal, and there is an appointment with an epilepsy specialist in the coming week. Admit any patient with more than one seizure that day or who does not fit the above criteria. Ensure clear documentation of follow-up arrangements, including booked clinic appointment. Meanwhile, advise the patient not to drive or use machinery and to take sensible precautions, with supervision when performing activities such as swimming/bathing until reviewed. Document this advice in the notes.

Seizures in known epileptics

Ask about any change from the patient's normal seizure pattern. Possible causes of poor seizure control include: poor compliance with medication, intercurrent illness/infection, alcohol, or drug ingestion. Examine to exclude any injury occurring from the fit, especially to the head. Occult dislocations (eg shoulder) may occur. Check vital signs, BMG, and anticonvulsant levels if toxicity or poor compliance is suspected. Refer patients with a significant change in seizure pattern to the medical team. Discharge to the care of a responsible adult those patients who are fully recovered with no injuries, symptoms, or other concerns.

Status epilepticus

This is continuous generalized seizures lasting >30min or without intervening recovery. Cerebral damage ↑ with duration. Precipitants include cerebral infection, trauma, cerebrovascular disease, toxic/metabolic disturbances, and childhood febrile seizures. Mortality is ~10% (due to underlying pathology). Although seizures typically start as generalized tonic/clonic, these features may gradually diminish, making diagnosis difficult (coma with virtually no motor evidence of seizure, eg minimal twitching of ocular muscles only). Complications include hypoglycaemia, pulmonary hypertension, and pulmonary oedema, and precipitous ↑ ICP can also occur.

Treatment of status epilepticus

- Establish a clear airway (a nasopharyngeal airway may help).
- Give O₂ as needed.
- Monitor ECG, SpO₂, T°, pulse rate, and BP.
- Obtain IV access; check BMG and correct hypoglycaemia if present (50mL of 20% glucose IV).
- Give lorazepam 4mg IV slowly into a large vein (diazepam 10mg is an alternative). Repeat IV lorazepam 4mg slowly after 10min if seizures continue.
- Buccal midazolam 10mg (can be repeated once) or rectal diazepam solution 10–20mg (can be repeated up to a total of 30mg) are alternatives if there is no venous access.
- If alcohol abuse or malnutrition is suspected, give slow IV thiamine in the form of Pabrinex® two pairs of ampoules in 100mL of 0.9% saline (this occasionally causes anaphylaxis; be prepared to treat—see BNF).
- Consider the possibility of pregnancy-related fits (eclampsia) in women of child-bearing age and treat accordingly (with IV magnesium sulfate—as outlined in  Eclampsia, p. 611).
- Check ABG and save blood for cultures, FBC, U&E, glucose, Ca²⁺, Mg²⁺, LFTs, clotting, and drug levels (and toxicology screen if poisoning/overdose is suspected).
- Search for features of injury (especially head injury) and infection (look for a rash).
- If seizures continue despite above therapy, call ICU and consider the use of phenytoin (20mg/kg IV, up to a max of 2g, at a rate of 50mg/min), with ECG monitoring, or fosphenytoin (20mg/kg phenytoin equivalent IV, <150mg/min). A 70kg patient would require 1400mg phenytoin equivalent of fosphenytoin (28mL Pro-Epanutin®) diluted in 100mL of 0.9% saline or 5% glucose, given over 10–15min.
- After 30min, contact ICU and proceed without delay to rapid sequence induction (RSI) (ideally with thiopental) and tracheal intubation, and continue anticonvulsant medication.

Hypoglycaemia

Hypoglycaemia can mimic any neurological presentation, including coma, seizures, acute confusion, or isolated hemiparesis.

► Always exclude hypoglycaemia in any patient with coma, altered behaviour, and neurological symptoms or signs.

Plasma glucose is normally maintained at 3.6–5.8 mmol/L. Cognitive function deteriorates at levels of <3.0 mmol/L, but symptoms are uncommon at levels of >2.5 mmol/L. In diabetics, however, the threshold for symptoms can be very variable. Hypoglycaemia is potentially fatal and accounts for 2.4% of deaths in patients with type 1 diabetes. Even mild episodes aggravate pre-existing microvascular complications and lead to cumulative brain damage.

Causes

In patients with diabetes, the most common cause is a relative imbalance of administered vs required insulin or oral hypoglycaemic drug. This may result from undue or unforeseen exertion, insufficient or delayed food intake, and excessive insulin administration (due to time, dose, or type of insulin). Other causes are:

- Alcohol (in addition to alcohol directly causing hypoglycaemia, the features of hypoglycaemia may be mistaken for alcohol intoxication or withdrawal).
- Addison's disease.
- Pituitary insufficiency.
- Post-gastric surgery.
- Liver failure.
- Malaria.
- Insulinomas.
- Extra-pancreatic tumours.
- Attempted suicide or homicide with large doses of insulin or oral hypoglycaemic drug.

Symptoms and signs

Common features Sweating, pallor, tachycardia, hunger, trembling, altered mental state or loss of consciousness, irritability, irrational or violent behaviour, fitting, focal neurological deficit (eg hemiplegia). Look for MedicAlert bracelet/chain.

Diagnosis

Check venous or capillary blood with glucose oxidase strip (BMG). If <3.0 mmol/L, take a venous sample for a formal blood glucose level, but give treatment without waiting for the result. Take appropriate samples if overdose of insulin, oral hypoglycaemic agent, or other drugs is suspected.

Treatment

This depends upon the conscious state and degree of co-operation of the patient. Choose the appropriate option from the following:

- A fast-acting oral carbohydrate 5–15g (eg Lucozade®, sugar lumps, Dextrosol®, followed by biscuits and milk).
- Glucagon 1mg: SC, IM, or IV. Can be administered by relatives, by ambulance crew, and when venous access is difficult. Glucagon is not suitable for treatment of hypoglycaemia due to sulfonylurea drugs, liver failure, or in chronic alcoholism (as there may be little liver glycogen available for mobilization).
- Glucose 10% solution 50mL IV, repeated at 1–2min intervals until the patient is fully conscious or 250mL (25g) has been given.
- Glucose 50% solution (25–50mL IV) is hypertonic, liable to damage veins, and no more effective than glucose 10%. If glucose 50% is used, give it into a large vein and follow with a saline flush.
- The time taken for return of consciousness and the incidence of nausea, vomiting, and other adverse effects are similar for IV glucagon and glucose.

Persistence of an altered conscious level suggests another underlying pathology (eg stroke) or may reflect the development of cerebral oedema due to hypoglycaemia, which has high mortality. Maintain plasma glucose at 7–11mmol/L; contact ICU, and consider mannitol and/or dexamethasone. Arrange urgent investigation (eg CT scan) and search for other causes of altered consciousness.

Overdose Glucose infusions may be needed for 24hr or longer after poisoning with insulin or an oral hypoglycaemic drug, depending upon exactly what and how much has been taken. Hypokalaemia may be a problem. Block excision of the injection site has been used as successful treatment for insulin overdose. Octreotide may be helpful in recurrent hypoglycaemia due to overdose of a sulfonylurea drug (see  Sulfonylurea poisoning, p. 205).

Discharge

90% of patients fully recover in 20min. Provided that the cause for the episode has been identified and fully corrected, it is reasonable to discharge the patient after observation in the ED, with appropriate follow-up.

Arrange follow-up, having considered the following:

- Why did this episode occur?
- Has there been a recent change of regimen, other drugs, alcohol, etc.?
- Is the patient developing hypoglycaemic unawareness or autonomic dysfunction?

If the patient is a driver, advise them to inform the Driver and Vehicle Licensing Agency (DVLA) of the hypoglycaemic episode.

Hyperglycaemic crises

Diabetic ketoacidosis (DKA) is caused by absolute or relative ↓ insulin levels. Plasma glucose ↑ causes an osmotic diuresis, with Na^+ and water loss (up to 8–10L), hypotension, hypoperfusion, and shock. Normal compensatory hormonal mechanisms are overwhelmed and lead to ↑ lipolysis. In the absence of insulin, this results in the production of non-esterified fatty acids, which are oxidized in the liver to ketones.

Younger undiagnosed patients with diabetes often present with DKA developing over 1–3 days. Plasma glucose levels may not be grossly ↑; euglycaemic ketoacidosis can occur. Urinalysis demonstrates ketonuria.

Hyperosmolar hyperglycaemic state (HHS) is caused by intercurrent illness, inadequate diabetic therapy, and dehydration. It develops over days/weeks and is more common in the elderly. HHS is characterized by ↑ glucose levels ($>30\text{ mmol/L}$), ↑ blood osmolality, and a lack of urinary ketones. Mortality is ~5–10% but may be even higher in the elderly.

Causes

Think of the four 'I's separately or (often) in combination:

- *Infection*: common primary foci are the urinary tract, respiratory tract, and skin.
- *Infarction*: myocardial, stroke, GI tract, peripheral vasculature.
- *Insufficient insulin*.
- *Intercurrent illness*: many underlying conditions precipitate or aggravate DKA and HHS.

Clinical features

Hyperglycaemic crisis may present in various ways. Some of the following are usually present:

- *Signs of dehydration*: thirst, polydipsia, polyuria, ↓ skin turgor, dry mouth, hypotension, tachycardia.
- *GI symptoms*: are common in DKA, with nausea, vomiting, and abdominal pain. This can be severe and mimic an 'acute surgical abdomen'.
- *Hyperventilation* (respiratory compensation for metabolic acidosis), with deep rapid breathing (Kussmaul respiration) and the smell of acetone on the breath, is pathognomonic of DKA.
- *True coma* is uncommon, but altered conscious states and/or focal neurological deficits (which may correct with treatment) are seen particularly in older patients with HHS.

Diagnosis and investigations

Aim to confirm the diagnosis and search for possible underlying cause(s):

- Check BMG and test the urine for glucose and ketones.
- Send blood for U&E, blood glucose, creatinine, and osmolality (or calculate it): $m\text{Osm}/\text{L} = (2 \times \text{Na}^+) + \text{glucose} (\text{mmol/L}) + \text{urea} (\text{mmol/L})$.
- Check ABG (look for metabolic acidosis ± respiratory compensation).
- FBC and CXR (to search for pneumonia).
- ECG and cardiac monitoring (look for evidence of hyper-/hypokalaemia).
- Blood cultures and, if appropriate, throat or wound swabs.
- Urine/sputum microscopy and culture.

Treatment of DKA

- If altered consciousness/coma, open/maintain a patent airway.
- Give O₂ by mask, as required. Consider the possible need for GA and IPPV for coma ± severe shock.
- Commence IVI with 0.9% saline. Give 1000mL of 0.9% saline over 0.5–1hr, then 500mL/hr for the next 2–3hr. Persistent hypotension may require ↑ in infusion rate and/or colloid administration. Avoid over-rapid infusion with the risks of pulmonary oedema and ARDS, especially in the elderly and patients with IHD.
- *Insulin:* start an infusion of soluble insulin after IV fluids have started using an IV pump or a paediatric burette at 0.1U/kg/hr (typically 6U/hr). Check blood glucose and ketone levels every hour initially—aim for blood glucose to drop by at least 3mmol/L/hr, and blood ketone by at least 0.5mmol/L/hr. Continue insulin infusion until blood ketone is <0.3mmol/L and blood pH is >7.3.
- When plasma glucose is <14mmol/L, add 10% glucose IVI at a rate of 125mL/hr (through a large vein) to help ketone clearance and acid–base state.
- *Electrolyte balance:* although total body K⁺ is low, plasma K⁺ may be normal, ↑, or ↓. With treatment, K⁺ enters cells and plasma levels ↓—therefore, unless initial K⁺ levels are >5.5mmol/L, give 20mmol/hr of potassium chloride (KCl), monitor ECG, and check K⁺ levels hourly. Despite the presence of metabolic acidosis, do not give sodium bicarbonate. Other electrolytes such as Ca²⁺, Mg²⁺, and phosphate (PO₄²⁻) are commonly disturbed but rarely need emergency correction.
- Consider an NG tube to ↓ the risk of gastric dilation and aspiration.
- Monitor urine output (most accurate with a urinary catheter).
- Consider a central venous catheter to monitor CVP to guide treatment in the elderly or severe illness.
- Arrange admission to ICU, HDU, or acute medical admissions unit.

Other aspects of treatment

Signs of infection Are often masked. T° is rarely ↑, and ↑ WCC may only reflect ketonaemia. If in doubt, treat with a broad-spectrum antibiotic.

Over-rapid fluid replacement Can cause cardiac failure, cerebral oedema, and ARDS, especially in patients with underlying cardiac disease or the elderly. CVP monitoring may be needed.

Clotting Hyperglycaemia causes a hypercoagulable state—DVT or PE may occur. Administer prophylactic anticoagulation with LMWH in DKA or hyperosmolar states.

Treat HHS with IV 0.9% saline. Do not start insulin unless significant keto-naemia, or glucose does not fall with fluid therapy. Seek advice before starting insulin as it risks cardiovascular collapse.

Sodium derangements

Abnormal Na⁺ states can occur with hypervolaemia, euvolaemia, or hypovolaemia, depending on the underlying pathophysiological process.

Hyponatraemia

Causes Include excessive fluid loss replaced by hypotonic fluids (diarrhoea, burns, prolonged exercise such as marathon running), polydipsia, ecstasy ingestion, syndrome of inappropriate antidiuretic (ADH) secretion, nephrotic syndrome, renal impairment, hepatic cirrhosis, cardiac failure, and many prescription drugs (including diuretics, heparin, and ACE inhibitors).

Treatment of acute hyponatraemia (<24hr duration) Those with mild symptoms can be effectively treated by fluid restriction. Patients who present with seizures or signs of ↑ ICP are at risk of death and require more aggressive treatment. Serum Na⁺ <120mmol/L is associated with risk of brain herniation. Give up to 200mL of 2.7% saline IV over 30min and recheck serum Na⁺ levels.

Treatment of chronic hyponatraemia (>24hr duration) Is associated with central pontine myelinolysis, particularly in patients with low K⁺ levels or alcoholic patients. Chronic hyponatraemia should be corrected no faster than 10mmol/L in 24hr. Treat the underlying cause. This may be as simple as discontinuing a diuretic. Patients with cardiac failure, cirrhosis, or nephrotic syndrome (hypervolaemic patients) should be fluid-restricted. Severe hyponatraemia in association with seizures or ↓ GCS may be cautiously treated with hypertonic saline (200mL of 2.7% saline over 30min and recheck serum Na⁺). Aim to ↑ serum Na⁺ by no more than 5mmol/L using this method.

Hypernatraemia

Usually reflects a loss of water in excess of loss of Na⁺.

Causes Include diabetes insipidus (lack of ADH or lack of renal response to ADH), diarrhoea, vomiting, diuretics, hypertonic saline, sodium bicarbonate administration, or Cushing's syndrome.

Treatment Do not correct Na⁺ concentration faster than 1mmol/L/hr. Use 0.9% saline to correct hypovolaemia (patients who have tachycardia, hypertension, or postural hypotension). Once the patient is euvoalaemic, use an infusion of 0.45% saline or 5% glucose. The free water deficit can be calculated using the formula:

$$\text{Free water deficit (L)} = 0.6 \times \text{weight(kg)} \times [(\text{serum Na}^+ / 140) - 1]$$

Replace the deficit over 48hr (in addition to normal maintenance fluids). Check serum Na⁺ after 2–3hr to monitor correction rate.

Complications Include seizures, subdural and intracerebral haemorrhages, ischaemic stroke, and dural sinus thrombosis. Rapid correction of Na⁺ levels (particularly in chronic hypernatraemia) can cause cerebral oedema and further neurological complications.

Addisonian crisis

Acute adrenocortical insufficiency is rare and easily missed. By far, the most common cause is sudden withdrawal of chronic steroid therapy (deliberately or inadvertently). An Addisonian crisis may also be precipitated in these patients by intercurrent injury, infection, or stress—↑ steroid requirement. 80% of Addison's disease in the UK is idiopathic (autoimmune) and may be associated with Graves' disease, Hashimoto's thyroiditis, type 1 diabetes mellitus, pernicious anaemia, hypoparathyroidism, and ovarian failure. Other causes include TB, fungal infections, metastatic disease, congenital adrenal hyperplasia, drugs (eg metyrapone or cytotoxic agents), haemorrhage into the adrenal glands occurring as a complication of anticoagulation, or meningococcal septicaemia (Waterhouse–Friderichsen syndrome). Look for a MedicAlert bracelet indicating that the patient is taking steroids.

Precipitating factors

Infection, trauma, MI, cerebral infarction, asthma, hypothermia, alcohol, pregnancy, exogenous steroid withdrawal or reduction.

Clinical features

Addison's disease frequently has an insidious onset with weakness, apathy, anorexia, weight loss, abdominal pain (which may be severe enough to mimic an acute abdomen), and oligomenorrhoea. In crisis, the main features may be shock (tachycardia, peripheral vasoconstriction, severe postural hypotension occasionally with syncope, oliguria, profound muscle weakness, confusion, altered consciousness leading to coma) and hypoglycaemia. Chronic features of Addison's disease are: areas of vitiligo and hyperpigmentation in the palmar creases, buccal mucosa, areolae, scars, and axillae.

Investigation

Obtain IV access, and send blood to check for hyperkalaemia, hyponatraemia, hypoglycaemia, uraemia, mild acidosis, hypercalcaemia, and eosinophilia which may be present. Also, take blood for cortisol (10mL in a heparinized tube) and adrenocorticotropic hormone (ACTH) if possible—contact the biochemistry lab to warn them that these tests will be required. Take blood cultures, urine cultures, and sputum for culture and sensitivity.

Management

- If an Addisonian crisis is suspected, take appropriate blood samples, but start treatment without waiting for results.
- If features of haemodynamic compromise are present, commence volume replacement with IV 0.9% saline if shocked.
- Give hydrocortisone sodium succinate 100mg IV stat.
- Treat hypoglycaemia with 50mL of 10% glucose IV (repeated if necessary).
- If infection is suspected as a precipitating cause, consider giving broad-spectrum antibiotics.
- Refer for admission.

Thyrotoxic crisis

A rare condition, occurring in 1–2% of patients with established hyperthyroidism (usually toxic diffuse goitre—‘Graves’ disease). Mortality is significant (~10%).

Causes

It is often precipitated by a physiological stressor:

- Premature or inappropriate cessation of anti-thyroid therapy.
- Recent surgery or radio-iodine treatment.
- Intercurrent infection (especially chest infection).
- Trauma.
- Emotional stress.
- DKA, hyperosmolar diabetic crisis, insulin-induced hypoglycaemia.
- Thyroid hormone overdose.
- Pre-eclampsia.

Clinical features

Onset may be sudden with features of hyperthyroidism and adrenergic overactivity. Fever and cardiovascular and neurological symptoms are common. Weight loss, ↑ appetite, tremor, irritability, emotional lability, heat intolerance, sweating, itch, oligomenorrhoea, agitation, anxiety, confusion, coma, palpitations, tachycardia, AF (rarely, complete heart block). It may mimic an ‘acute abdomen’, with abdominal pain, diarrhoea, and vomiting.

Differential diagnosis

Includes acute pulmonary oedema, neuroleptic malignant syndrome, septic shock, anticholinergic or sympathomimetic overdose, drug withdrawal, or acute anxiety states.

Investigation

- U&E, BMG and blood glucose, Ca^{2+} (hypercalcaemia occurs in ~10%).
- FBC, differential WCC, coagulation screen.
- Screen for infection: mid-stream urine (MSU), blood cultures, sputum.
- Thyroxine (T_4) and tri-iodothyronine (T_3) (for later analysis), thyroid-stimulating hormone (TSH).
- CXR (searching for pulmonary infection or congestive heart failure).
- ECG (looking for arrhythmias).

Treatment

- Manage the airway and give O_2 if indicated.
- Obtain IV access and commence IV 0.9% saline (initially 500mL 4-hourly).
- Give propranolol (1mg slow IV over 1 min or 60mg PO) to ↓ heart rate.
- Give hydrocortisone 100mg IV.
- If sedation is required, give small titrated amounts of benzodiazepine (eg diazepam 5–20mg PO/IV) or haloperidol.
- Give broad-spectrum antibiotic if infection is suspected.
- Consider cooling measures in hyperthermia.
- Refer for admission (consider admission to ICU).
- Once admitted, carbimazole will normally be given with iodine.
- Do not give aspirin (this can exacerbate the clinical problem by displacing thyroxine from thyroid-binding globulin).

Acute kidney injury

Background

AKI is diagnosed by:

- A serum creatinine rise ≥ 26 micromoles/L over 48hr.
- A serum creatinine rise $\geq 50\%$ over 7 days.
- \downarrow urine output of <0.5 mL/kg/hr for >6 hr.

AKI may be present if creatinine is high, even if the above criteria are not met. If it is unclear whether a patient has worsening of their chronic kidney disease (CKD) or AKI, treat as the latter.

Causes

- Pre-renal: hypovolaemia (haemorrhage, burns, pancreatitis), cardiogenic shock, sepsis, renal vasoconstriction (drugs).
- Renal: glomerulonephritis, vasculitis, acute tubular necrosis, interstitial nephritis.
- Post-renal: obstruction within renal system (calculi, stricture, tumour) or from outside (prostatic hypertrophy, pelvic malignancy, retroperitoneal fibrosis).

History and examination

Consider AKI in anyone with acute illness aged >65 y, a history of kidney disease, those on nephrotoxic drugs, and patients who are systemically unwell (eg with sepsis). Look for signs of dehydration, diarrhoea and vomiting, and \downarrow urine output.

Investigation

- U&E, VBG, and other bloods, depending on underlying cause (eg CK in suspected rhabdomyolysis).
- Urinalysis (blood and protein may indicate glomerular disease)—if \downarrow urine output, consider a bladder scan and catheterization.
- If a catheter is already *in situ*, consider flushing or replacing it.
- Perform an ECG (looking especially for changes suggesting hyperkalaemia—see  Hyperkalaemia, pp. 170–1).

Management

- Treat the underlying cause.
- Assess hydration status and commence a fluid balance chart. If dehydrated, commence IV fluid therapy with a crystalloid. Stop all nephrotoxic drugs (eg NSAIDs and ACE inhibitors).
- Treat hyperkalaemia (see  Hyperkalaemia, pp. 170–1).
- Refer to the renal team.
- Indications for haemodialysis include intractable hyperkalaemia, severe acidosis, fluid overload, toxin removal, and severe uraemic symptoms. If severely unwell, haemofiltration is more appropriate—refer to ICU.

Chronic kidney disease

Patients with established CKD are likely to be very well known to the hospital. Review old notes and recent blood results, and liaise early with inpatient specialist teams.

Established CKD (not on dialysis)

Patients with mild CKD (GFR >40–100mL/min) are unlikely to have specific problems related to their underlying renal failure. With GFR of <40mL/min, and especially if GFR is <10mL/min, complications may influence presentation and treatment. These patients are prone to pathological fractures.

- Secondary hyperparathyroidism and osteomalacia (lack of active vitamin D) occur in moderate CKD. In severe CKD, aluminium bone disease and β 2-microglobulin-related amyloidosis may be associated with pathological fractures.
- ‘Pseudogout’ due to high $\text{Ca}^{2+}/\text{PO}_4^-$ production and twitching/tetany due to hypocalcaemia may occur.

Other problems include

- Defective regulation of extracellular fluid volume: there is an ↑ risk of fluid depletion in moderate CKD and fluid retention in severe CKD. High-dose diuretics may be required in severe disease—the combination of furosemide and metolazone may be effective, even with very low GFRs.
- Hyperkalaemia: most patients preserve K^+ balance but cannot deal with sudden K^+ loads (eg dietary, tissue damage/catabolism, GI bleed). Associated ↓ Ca^{2+} compounds the cardiac effects. Plasma K^+ may ↑ very quickly, so monitor ECG and check K^+ frequently.
- Hypertension: often severe and resistant, with an ↑ incidence of accelerated phase. Ciclosporin and erythropoietin ↑ BP and can precipitate hypertensive encephalopathy.
- Drug effects: drugs may accumulate (eg opioids, aciclovir, some antibiotics), worsen renal failure (eg NSAIDs, ACE inhibitors, which ↓ renal perfusion), and cause hyperkalaemia (eg K^+ -sparing diuretics, ACE inhibitors, NSAIDs).
- Infections: impaired white blood cell (WBC) function, with ↑ risk of severe infection, and features of infection (eg pain, fever) may be masked by the relative immunocompromised state.
- Bleeding: platelet function is impaired.
- Pericarditis: a sign of severe chronic renal failure (CRF), indicating the need for dialysis.
- Neurological dysfunction: usually a sign of severe uraemia—convulsions and/or altered conscious state indicate a global metabolic disturbance.

Haemodialysis patients' problems

Pulmonary oedema Usually occurs shortly before the next dialysis session and may reflect fluid overload due to non-compliance with diet and fluid restriction. Most are virtually anuric, so diuretics are ineffective. Get the patient on dialysis without delay. Whilst this is being arranged, give O_2 as needed and sublingual (SL), buccal, or IV nitrates.

Pre-dialysis hyperkalaemia May present with neuromuscular symptoms (eg muscle spasms, weakness, paralysis, paraesthesiae) or arrhythmias, including cardiac arrest. Standard treatment (see  Hyperkalaemia, pp. 170–1) can buy time whilst emergency dialysis is arranged. When giving glucose/insulin, give 6U of insulin at most (there is a risk of late hypoglycaemia, since insulin half-life will be ↑).

Complications of vascular access Arteriovenous fistulae are a dialysis patient's lifeline—never occlude the limb with BP cuffs or tourniquets. Do not use for vascular access unless a life-threatening emergency. Acute shunt thrombosis (loss of palpable thrill, often local pain/redness) is a vascular emergency. Arteriovenous fistulae and central lines are common infection sources (usually staphylococcal), often with no overt external abnormality, but presenting with acute 'viral illness' symptoms.

Continuous ambulatory peritoneal dialysis

Bacterial peritonitis Occurs every 12–18 patient-months. Features are cloudy drained dialysate bags, abdominal pain, and peritonism. Systemic sepsis is usually absent or minimal. Staphylococci are the most common organisms. Suspect an underlying surgical cause (most often diverticular abscess) if Gram –ve organisms or anaerobes present in drainage fluid, and particularly if >1 type of organism is found on microscopy or culture.

Hyperglycaemia Diabetic patients on continuous ambulatory peritoneal dialysis can develop acute severe (usually non-ketotic) hyperglycaemia, related to high dialysate glucose concentrations (80–140 mmol/L).

Hernias of all types, leakage of dialysate into the abdominal wall or the pleural cavity, and scrotal swelling (open processus vaginalis) may occur.

Transplant patients

Contact the transplant team whenever any transplant patient presents to the ED. They will know the patient well and will advise about drug therapy and intercurrent problems, and help with follow-up.

Acute rejection Signs include pain, tenderness, and swelling over graft, ↓ urine output, fever, systemic upset, and biochemical deterioration. Often indistinguishable from acute bacterial infection—if in doubt, treat for both, pending results of further testing by specialists (renal biopsy, blood and urine cultures).

Infections May be opportunistic, whilst 'conventional' infections are unduly severe, with response modulated by steroids.

Poor wound healing, avascular necrosis, and pathological fractures May be caused by steroids.

Urinary tract infection

The urinary tract is normally bacteriologically sterile. Urine infection is present if $>10^5$ colony-forming units are present per mL of urine. Except at the extremes of age, UTIs are much more common in ♀ due to the shorter urethral length. Most UTIs occur because of organisms invading the bladder via the urethra. Proximal invasion via the ureter may result in acute or chronic pyelonephritis, particularly if anatomical derangement exists with impaired ureteric or bladder emptying. In both sexes, an underlying structural abnormality ↑ UTI risk. Blood-borne spread of infection to the urinary tract can occur (eg in bacterial endocarditis or systemic Gram -ve infection). UTI is usually caused by a single organism. The most common organism (90%) at all ages is *Escherichia coli*. *Proteus*, *Klebsiella*, and saprophytic staphylococci account for most of the remainder in adults. Other organisms (eg *Pseudomonas*) more commonly cause UTI in hospitalized patients or following instrumentation.

Presentation

Lower UTI (cystitis)

Dysuria, frequency, haematuria, suprapubic discomfort, urgency, burning, cloudy urine with an offensive smell. Patients with acute urethral syndrome have identical symptoms, but -ve urine culture.

Upper UTI (acute pyelonephritis)

Often systemically unwell with malaise, fever, loin and/or back pain, vomiting, rigors, and occasionally Gram -ve septicaemia. Diagnose pyelonephritis if there is evidence of UTI with loin pain and $T^\circ > 38^\circ C$.

Investigations

Traditional investigations are:

- Reagent strip (dipstick) urinalysis may show haematuria, proteinuria, and +ve nitrite and leucocyte esterase tests. A patient with clear urine, -ve on dipstick testing, is extremely unlikely to have a UTI. False +ve results may be secondary to urinary tract tumours or excessive exercise. A false -ve nitrite test may reflect pathogens that do not convert dietary nitrates to nitrites.
- Urine microscopy may show leucocytes ($>100/\text{mL}$ correlates well with infection but may be due to contamination or other urinary tract pathology). Red blood cells (RBCs) are commonly seen on microscopy but, in isolation, have a low degree of sensitivity or specificity for UTI. Underlying renal pathology is suggested by finding urinary crystals, RBCs, or granular casts.
- MSU for culture and sensitivity. Transport the sample to the laboratory without delay to ensure that bacterial overgrowth does not artificially ↑ the count. Dipslides dipped into freshly passed urine and transported in a plastic container to the laboratory are an alternative.

Do not routinely perform urine microscopy and culture for women with uncomplicated UTIs, but do perform them if there is haematuria, impaired renal function, immunosuppression, or abnormality of the renal tract.

Treatment

Lower UTI

- Aim to discharge women with uncomplicated lower UTIs with antibiotics. Commence a 3-day course of trimethoprim (200mg bd) or nitrofurantoin (50mg qds). Provide advice regarding fluid intake, no 'holding on', and voiding after intercourse. (Note: urinary alkalinization renders nitrofurantoin ineffective.)
- Consider a 5 to 10 day course for women with impaired renal function, immunosuppression, or abnormality of the renal tract. Advise the patient to see her GP for review and MSU result.
- In pregnancy, treat symptomatic bacteriuria (eg amoxicillin 250mg tds for 7 days or cefalexin 500mg tds for 7 days), and arrange GP follow-up for a repeat MSU. Also treat with antibiotics a pregnant woman who is asymptomatic and has a +ve urine dipstick for leucocytes and nitrites, again advising GP follow-up. Remember that trimethoprim is contraindicated in the first trimester and nitrofurantoin is contraindicated in the third trimester.
- Do not give antibiotics to elderly men and women with asymptomatic bacteriuria, unless they show signs of being unwell.
- Treat men with symptoms of a lower UTI with a 7-day course of nitrofurantoin (50mg qds) or trimethoprim (200mg bd). Consider the possibility of alternative diagnoses: chlamydial infection, prostatitis, and epididymitis.

Upper UTI

- Assess and treat for severe sepsis (see  Shock, pp. 64–5). Admit if there is evidence of sepsis and/or systemic symptoms, dehydration, or no response to oral antibiotics. Provide parenteral antibiotics (eg gentamicin + amoxicillin IV), fluid replacement, and analgesia.

Older patients with suspected UTI

UTI is likely if an elderly person without obvious infection elsewhere has two or more of: dysuria, urgency, frequency, urinary incontinence, rigors, flank or suprapubic pain, frank haematuria, and new confusion (or worsening of pre-existing confusion). Do not use urinalysis to diagnose UTI, but if clinically suspected, send an MSU. Treat with antibiotics (eg trimethoprim or nitrofurantoin). Consider analgesia and the need to admit to hospital (eg signs of sepsis and/or fever with rigors). If there is an indwelling catheter, remove and replace it. (Note: do not treat catheterized patients with asymptomatic bacteriuria with antibiotics.)

(See  <https://www.sign.ac.uk>)

Hyperkalaemia

Hyperkalaemia is classified as follows: mild (K^+ 5.5–6.0 mmol/L), moderate (K^+ 6.1–6.9 mmol/L), or severe ($K^+ > 7.0 \text{ mmol/L}$).

Causes

- Spurious: sample haemolysed or taken from limb with IVI containing K^+ .
- ↓ renal excretion: AKI, patients with CKD or on dialysis with K^+ load, K^+ sparing diuretics (eg spironolactone, amiloride).
- Cell injury: crush injury and other causes of rhabdomyolysis, burns, tumour cell necrosis, massive or incompatible blood transfusion.
- K^+ cellular shifts: acidosis from any cause (eg DKA), drugs (suxamethonium, β -blockers).
- Hyperaldosteronism: Addison's disease, drug-induced (NSAIDs, ACE inhibitors).

Clinical features

There may be muscle weakness/cramps, paraesthesiae, hypotonia, focal neurological deficits. Dangerous hyperkalaemia may be asymptomatic.

ECG changes

ECG changes typically progress as hyperkalaemia worsens, as follows (see Fig. 3.35):

- Peaked T waves.
- Small, broad, or absent P waves.
- Widening QRS complex.
- Sinusoidal ('sine wave' pattern) QRST.
- AV dissociation or VT/VF.

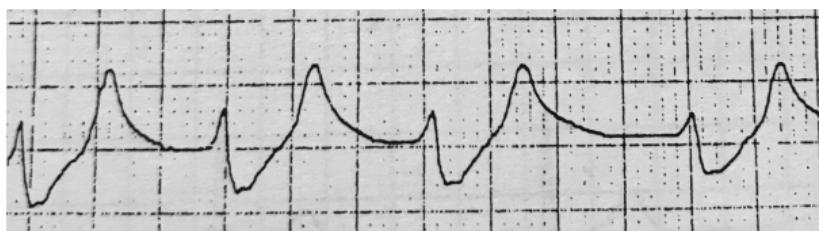


Fig. 3.35 ECG strip of severe hyperkalaemia: peaked T waves, absent P waves, and widened QRS.

Management of hyperkalaemic cardiac arrest

If a patient in cardiac arrest is known to have hyperkalaemia, follow the standard ALS guidelines (see Cardiac arrest, p. 48), plus one or more of the following:

- Give 10mL of 10% calcium chloride IV by rapid bolus injection.
- Consider giving 10U of short-acting insulin + 100mL of 50% glucose rapidly IV.
- If there is severe acidosis, give 50mL of 8.4% sodium bicarbonate rapidly IV.
- Consider haemodialysis for cardiac arrest induced by hyperkalaemia which is resistant to medical treatment.

Management of severe hyperkalaemia

Urgent treatment is needed if K^+ is $>6.5\text{mmol/L}$, unless this is a spurious and incorrect result. If K^+ is reported as $>6.5\text{mmol/L}$, obtain venous access; monitor and review the ECG. If there are no ECG signs of hyperkalaemia, take another blood sample for U&E, with care to avoid haemolysis, and a heparinized sample to measure K^+ on a blood gas machine.

Start treatment immediately if there are ECG changes of hyperkalaemia:

- Give 10mL of 10% calcium chloride slowly IV (over 5min). This does not lower K^+ but antagonizes cardiac membrane excitability. Hypercalcaemia may possibly potentiate toxicity in patients on digoxin, so give as an IV over 30min in these patients.
- Give 10U of short-acting human soluble insulin (eg Actrapid[®]) with 50mL of 50% glucose IV. This helps ↑ cellular uptake of K^+ , lowering serum levels by up to 1mmol/L within 1hr and lasting up to 4hr.
- Give nebulized salbutamol 5mg, repeated once as necessary. This will lower K^+ in most patients, acting in ~30min.
- Correct volume deficits/acidosis with IV fluids and isotonic (1.26%) sodium bicarbonate or aliquots (25–50mL) of 8.4%. Beware fluid overload/osmolar effects, especially in dialysis patients.
- Correct the underlying cause, if possible (eg steroid therapy for Addison's disease).
- Contact the nephrology team urgently for patients with acute or chronic renal failure, as emergency dialysis may be needed.

Hyperkalaemia in children See  Hyperkalaemia, p. 714.

Management of moderate hyperkalaemia

Provided that the result is not spurious, a K^+ level of 6–6.5mmol/L may be regarded as 'moderately' severe hyperkalaemia.

- Obtain venous access and monitor ECG.
- If there are ECG changes, treat as for severe elevation (as outlined previously).
- If there are no ECG changes, give 10U of short-acting human soluble insulin with 50mL of 50% glucose IV over 15–30min.
- Look for and treat the underlying cause and consider diuretics (eg furosemide 1mg/kg IV slowly) and dialysis.

Management of mild hyperkalaemia

K^+ level of 5.5–6mmol/L. Treat the underlying cause and any associated hypovolaemia. Discuss the need for specific intervention (diuretic, dialysis) with the medical team.

Hypokalaemia

Defined as $K^+ < 3.5 \text{ mmol/L}$; it is relatively common. Moderate hypokalaemia may result in lethargy, weakness, and leg cramps. In severe cases ($K^+ < 2.5 \text{ mmol/L}$), rhabdomyolysis and respiratory difficulties may occur.

ECG changes include prominent U waves and flattened T waves (see Fig. 3.36). The U waves can be mistaken for T waves and so give the erroneous impression of a long QT interval.

Treatment

In most instances, aim to replace K^+ gradually. The maximum recommended IVI rate of K^+ is 20 mmol/hr . Restrict more rapid rates of IVI (eg 20 mmol in $20\text{--}30 \text{ min}$) to those patients who have unstable arrhythmias when cardiac arrest is imminent (obtain senior/expert advice). Ensure cardiac monitoring occurs during any K^+ IVI.

Associated magnesium deficiency

Many patients with K^+ deficiency are also Mg^{2+} deficient. Consider replacing Mg^{2+} in those patients who have severe hypokalaemia.

(See also  <https://www.resus.org.uk>)

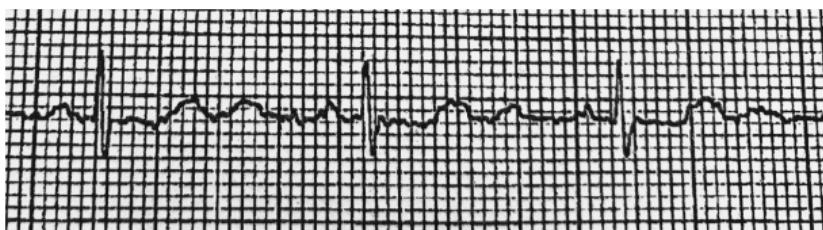


Fig. 3.36 ECG strip of hypokalaemia with prominent U waves.

Porphyria

Porphyrias are haem biosynthesis disorders in which enzyme deficiencies cause accumulation of porphyrin and porphyrin precursors. Most cases are hereditary, but abnormal porphyrin metabolism may develop in iron deficiency, alcohol excess, and lead poisoning. Acute porphyrias (acute intermittent porphyria, variegate porphyria, and hereditary coproporphyria) affect ~1 in 10,000 people in the UK. Non-acute porphyrias (eg porphyria cutanea tarda) do not produce acute attacks but cause skin photosensitivity sometimes associated with liver disease.

Attacks of acute porphyria are often caused by drugs: barbiturates, oestrogens, progesterones, sulfonamides, methyldopa, carbamazepine, phenytoin, sulfonylureas, chloramphenicol, tetracyclines, danazol, and some antihistamines. Other precipitants include: alcohol, smoking, dieting, emotional and physical stress, infection, substance misuse, and pregnancy.

Clinical features of acute porphyria

- Abdominal pain is common and can be severe, with nausea, vomiting, and constipation. Abdominal examination may be normal or there may be mild generalized tenderness.
- Peripheral neuropathy is usually motor, rather than sensory, and may progress to paralysis and respiratory failure.
- Tachycardia, hypertension, and postural hypotension.
- Psychiatric manifestations: agitation, depression, mania, and hallucinations.
- Hyponatraemia due to inappropriate ADH secretion can cause fits or coma.

Investigation and management of acute porphyria

Look for a MedicAlert bracelet. Review old medical notes.

If an acute attack is suspected, send a fresh urine sample (protected from light) to test for aminolevulinic acid and porphobilinogen. In an attack, urine goes dark red or brown, especially if left exposed to light (due to polymerization of porphobilinogen).

Treat acute attacks supportively (if necessary in ICU). Maintain carbohydrate intake (PO or IV). Control mild pain with paracetamol or aspirin; moderate/severe pain with morphine (\pm antiemetic). Consider chlorpromazine for agitation, and propranolol to control severe hypertension. Management of status epilepticus is difficult as many anticonvulsants are contraindicated—choose IV diazepam in the first instance. Haem arginate helps some patients with acute crises (take specialist advice).

Prescribing for patients with porphyria

Many drugs can precipitate attacks, so check with the patient and the BNF.

However, the safety of many drugs in porphyria is uncertain and effects vary between patients. If in doubt, obtain specialist advice. In addition to those mentioned earlier, safe drugs appear to be: ibuprofen, penicillin, ciprofloxacin, and bupivacaine.

Data are also available on the Internet at  <http://www.porphyrria.org.uk>

Bleeding disorders: assessment

- Contact a haematologist whenever treating a patient with a known or suspected bleeding disorder.

Haemostasis requires co-ordination between the vascular system, platelets, and coagulation pathways to limit blood loss from the circulation. Platelets interact with the vascular subendothelium, forming a primary platelet plug, which is strengthened by cross-linked fibrin strands formed via the coagulation cascade to allow restoration of vascular integrity (see Fig. 3.37). The fibrinolytic systems prevent excess clot formation and inappropriate local or generalized thrombosis by promoting lysis of fibrin.

Recognition of bleeding

Bleeding is expected after trauma, but suspect a bleeding disorder if spontaneous or excess haemorrhage occurs from multiple or uninjured sites into deep tissues and joints, or delayed bleeding occurs (hours/days). Bleeding disorders may be congenital or acquired. Ask about previous bleeding after trauma, dentistry, or surgery and about the family history.

Congenital disorders Haemophilia A (factor VIII deficiency), haemophilia B (factor IX deficiency), and von Willebrand's disease. Most adults with a congenital disorder know the nature of it and carry a National Haemophilia card or a MedicAlert bracelet giving details. Many haemophiliacs know more about their required treatment than the ED clinician! They will be registered and known at a haemophilia centre.

Acquired disorders May be due to liver disease, uraemia, drug use (ask specifically about aspirin, NSAIDs, warfarin/anticoagulants, alcohol), or unrecognized conditions such as haematological malignancy.

Hypothermia From whatever cause—aggravates any bleeding tendency. For example, an INR assay performed at 32°C will be prolonged to the same extent as would occur with a factor IX level of 2.5% of normal. The severity of this may not be recognized merely from standard tests as these are performed at 37°C. (See  Hypothermia: presentation, pp. 264–5.)

Site of bleeding Can give a clue as to the abnormality. Platelet problems (usually thrombocytopenia) often present with mucocutaneous bleeding (eg epistaxis, GI, GU, or heavy menstrual bleeding, bruising, purpura, and petechial haemorrhages). Bleeding into joints or potential spaces (eg retroperitoneal) and delayed bleeding are more often due to coagulation factor deficiencies. Patients with mucocutaneous bleeding and haemorrhage into deep spaces may have a combined platelet and coagulation factor abnormality (eg DIC).

Investigations

FBC Remember that in acute bleeds, Hb and Hct values fail to demonstrate the severity of red cell loss as haemodilution takes time. Platelet counts $<100 \times 10^9/L$ indicate thrombocytopenia, and those $<20 \times 10^9/L$ are associated with a risk of spontaneous bleeding. If platelet function is abnormal (eg with aspirin or clopidogrel), serious bleeding can occur with normal platelet levels.

INR Used to monitor anticoagulant control in patients on coumarin drugs. May be prolonged in liver disease. A normal INR makes clinically relevant effects of apixaban or rivaroxaban unlikely.

Activated partial thromboplastin time (APTT) Tests components of the intrinsic and common coagulation pathways (essentially all factors, except VII and XIII) but may be normal in the presence of mild deficiency. Used to dose IV unfractionated heparin. A normal APTT makes clinically relevant effects of dabigatran unlikely.

Anti-Xa assays Calibrated for apixaban, edoxaban, rivaroxaban, enoxaparin, dalteparin, and tinzaparin—can be used to measure each drug effect. However, these blood tests may not be available in an emergency.

Dilute thrombin time Very sensitive to dabigatran and remains elevated (for days), even after the clinical effect of dabigatran has gone.

Ecarin clotting time May help in determining the effect of dabigatran.

Individual factor levels Can be determined by specific assays, together with inhibitor screening tests for antibodies that can prolong normal clotting.

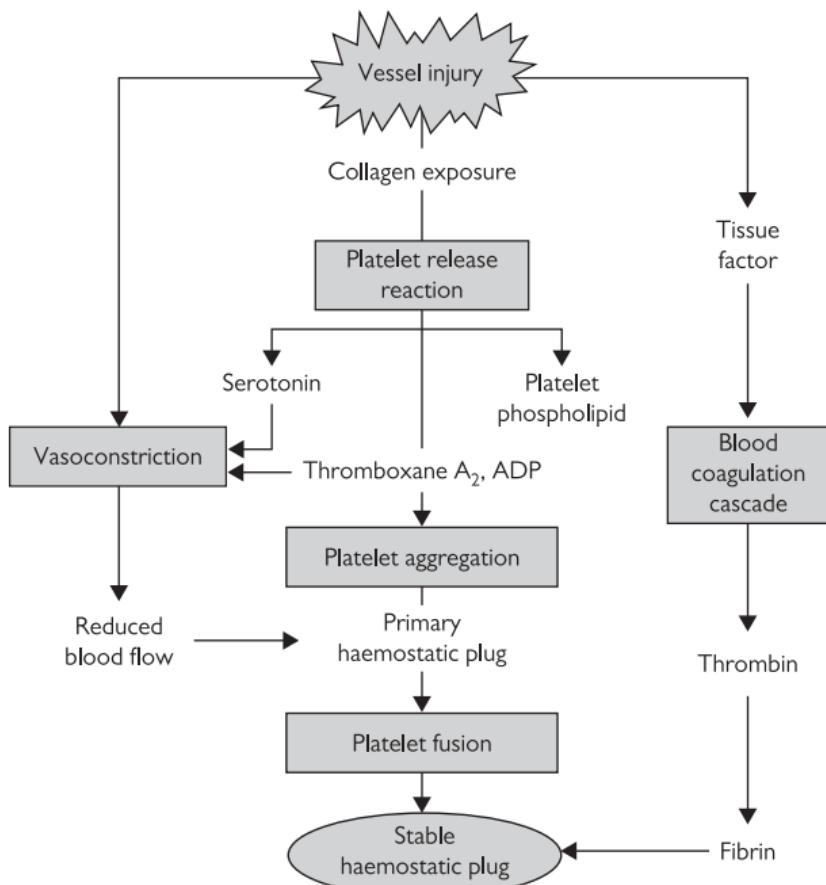


Fig. 3.37 Reactions involved in haemostasis.

Bleeding disorders: treatment

General aspects of treatment

- Liaise with the haematologist for patients with bleeding disorders.
- Perform routine wound/fracture management of patients with bleeding disorders, but consider the need for prior or simultaneous administration of factor concentrates/platelets under haematological guidance.
- Spontaneous or traumatic bleeding into the neck or pharynx may cause rapid airway compromise.
- Always consider intracranial haemorrhage in a patient with headache, neurological symptoms, or minor head trauma.
- Never give IM injections.
- Do not attempt central line placement, except *in extremis*.
- Before giving any drug, check whether it may aggravate the condition or interfere with intercurrent therapy.

Specific conditions

Vascular lesions May be inherited (Ehlers–Danlos syndrome, pseudoxanthoma elasticum, osteogenesis imperfecta, haemorrhagic telangiectasia) or acquired (eg due to steroids, infection such as meningococcaemia, thrombotic thrombocytopenic purpura, vasculitis, scurvy).

Platelet disorders Capillary-related mucocutaneous bleeding is common and may occur immediately after injury/surgery (eg dental extractions). The platelet count may be normal or ↓. Acquired thrombocytopenia may be due to drugs, toxins, infections, autoimmune conditions (eg immune thrombocytopenia), DIC, or massive blood transfusion. Abnormal platelet function occurs with uraemia, myeloproliferative disorders, and drugs (eg aspirin).

Coagulation pathway disorders Congenital coagulation pathway disorders predominate in ♂. They cause intramuscular or deep soft tissue haematomas. Bleeding onset after injury/surgery may be delayed 2–3 days.

von Willebrand's disease The most common congenital bleeding disorder, with von Willebrand (vW) factor and factor VIII deficiency and abnormal platelet function. Clinically, the condition is similar to a platelet disorder, but milder. Bleeding is commonly mucosal (eg epistaxis) and usually treated with desmopressin or factor VIII concentrate (which includes vW factor).

Haemophilia A Caused by a lack of functional factor VIII which is needed for clot formation. Often presents with bleeding into deep muscles, large joints, or the urinary tract. Intracranial bleeding is a major cause of death at all ages. Anticipate bleeding up to 3 days after trauma.

Haemophilia A associated with bleeding or potential bleeding is normally treated with factor VIII concentrate (some patients have 'home supplies' and may bring them to hospital). The volume (dose) depends upon the severity of haemophilia of the individual patient and the purpose of treatment (ie prophylaxis or therapy for current bleeding). Mild haemophilia A may also be treated with desmopressin.

Haemophilia B (Christmas disease) Involves a deficiency of factor IX activity and is genetically and clinically indistinguishable from haemophilia A, but much less common. It is normally treated with factor IX concentrate.

Disseminated intravascular coagulation

Patients may present with DIC due to infection (especially Gram -ve sepsis), trauma, malignancy, pregnancy (amniotic fluid embolism, placental abruption, toxæmia, retained products), any cause of shock, incompatible blood transfusion, or massive volume replacement. Following triggering of the coagulation process, consumption of platelets and coagulation factors (particularly fibrinogen, V, VIII, and XIII) occurs, with thrombin formation overwhelming the normal inhibition system, resulting in systemic fibrin deposition (see Fig. 3.38). Activation of the fibrinolytic system results in dissolution of fibrin and release of fibrin degradation products.

Investigations Platelet count is usually ↓, INR ↑ and APTT ↑, fibrinogen level ↓, fibrin degradation products ↑.

Treatment Is complex and requires control of the primary cause of the DIC to avoid total depletion of clotting factors. Obtain expert advice about replacement therapy with platelets, fresh frozen plasma (FFP), cryoprecipitate, prothrombin complex concentrate, heparin, and red cells (particularly required if the patient is actively bleeding).

EXTRINSIC SYSTEM

Tissue factor
+ VIII
+ Ca^{2+}

X

Prothrombin

Fibrinogen

INTRINSIC SYSTEM

XII (on contact with vascular endothelium)

XI

IX

Xa
+ V
+ Ca^{2+}
+ Phospholipid

XIIa

Xa
+ VIII
+ Ca^{2+}
+ Phospholipid

XIII

XIIIa

Fibrin

Cross-linking

Fig. 3.38 Coagulation cascade.

Patients on anticoagulants

Warfarin

A vitamin K antagonist inhibiting the production of factors II (prothrombin), VII, IX, and X. Expect patients with mechanical prosthetic heart valves to be prescribed warfarin, as this is a contraindication to the newer anticoagulants. Warfarin is also indicated for stroke prevention in AF and in vascular disease. Warfarin treatment cannot be started on its own for the treatment of DVT or PE without heparin (either IV or LMWH), as it initially induces a prothrombotic state. The effect of warfarin (measured by the INR) is influenced by intercurrent illness, liver disease, and changes in diet and/or alcohol consumption and other medications.

Newer oral anticoagulants

Apixaban, rivaroxaban, and edoxaban are all anti-Xa inhibitors. Dabigatran is a direct thrombin inhibitor. Apixaban and rivaroxaban are indicated for the emergency treatment of DVT and PE (see  Treatment of DVT/PE, p. 125), at a higher dose for the initial treatment period. Dabigatran and edoxaban cannot be started for the treatment of acute DVT or PE until 5–7 days of LMWH has been given. Any of the four drugs may be used for stroke prevention in patients with AF who do not have mechanical valves, as well as extended prophylaxis post-arthroplasty. These drugs are preferred by patients over warfarin as they do not require monitoring with blood tests and are not affected by diet.

Renal and hepatic impairment reduces drug clearance and so may promote bleeding. Newer oral anticoagulants have numerous drug interactions, including with antiepileptic, anti-TB, anti-HIV, antibiotic, and anti-arrhythmic drugs.

Low-molecular weight heparin

Enoxaparin, tinzaparin, and dalteparin are short-chain heparins which are given SC. They are often used for cancer-associated DVT and PE. They are also used in acute DVT or PE, if warfarin, dabigatran, or edoxaban oral therapy is planned. LMWH is the drug of choice for prophylaxis in medical inpatients. Syringes are designed for patients to self-administer as an outpatient. Contraindicated in severe renal impairment.

Unfractionated heparin

The anticoagulant of choice in acute DVT/PE in patients with severe renal impairment. Also used when there is a high risk of major bleeding because of its short half-life. The IV rate is adjusted using the APTT as a guide and discontinued when warfarin has achieved a therapeutic INR level.

Fondaparinux

A synthetic pentasaccharide administered as an od SC injection. Indicated in ACS.

Risks of bleeding on anticoagulation

When prescribing an anticoagulant or assessing a patient on anticoagulation, always check their bleeding risk. Patients with a recent bleed have a high risk of recurring bleeding. Patients who are very bruised are also at risk, as are those on antiplatelet (aspirin) or dual antiplatelet drugs. Patients with renal impairment have a high risk of bleeding, especially those on the new anticoagulants, LMWH, or fondaparinux. A platelet count of $<50 \times 10^9/L$ ↑ bleeding risk. If in doubt, discuss with a haematologist.

Managing major bleeding on anticoagulation

- Commence resuscitation measures (IV fluids, massive transfusion protocol as necessary).
- Identify the bleeding source and target haemorrhage control.
- Establish the time of the last anticoagulant dose and the anticoagulant name (new oral drugs will wear off after 24hr in patients with normal renal function).
- Send blood for APTT, INR, and cross-matching.
- Reverse warfarin with prothrombin complex concentrate (eg Beriplex®) and vitamin K 10mg IV.
- Reverse dabigatran with idarucizumab—if it is not available, consider dialysis.
- Andexanet alpha can be used to reverse rivaroxaban and apixaban.
- Give protamine sulfate if the patient has had a dose of LMWH in the past 24hr.
- Consider giving tranexamic acid 1g IV.

Patients with less severe bleeding on anticoagulation

Patients with muscle haematomas, haematuria, or epistaxis may require hospital admission for observation and specific local treatment. Stop anti-coagulant therapy for one or more days. Take expert advice when the patient is also at high risk of thrombosis (prosthetic heart valve or recent DVT/PE). Hold antiplatelet drugs.

(See  <https://www.b-s-h.org.uk/guidelines>)

Anticoagulation control check in inpatients on warfarin

For patients who have INR 4.0–7.0 without haemorrhage, withhold warfarin therapy for 1 or 2 days, and arrange review by an appropriate specialist team or GP. For patients with INR >7.0 without haemorrhage, withhold warfarin and obtain specialist consultation before considering phytomenadione (vitamin K₁) 5mg by slow IV injection or PO.

Blood transfusion overview

► It is better to stop bleeding than to have to replace blood loss.

General aspects

Correctly documenting and labelling blood tubes and forms, combined with checking blood products prior to administration, are crucial for safe patient care. If a patient's name(s), date of birth, clinical details, and address are unknown or uncertain, provide the gender and approximate age and identify them for transfusion purposes by a unique number (usually their unique ED number) and inform the blood transfusion laboratory.

To avoid confusion, the practitioner taking the blood sample must label and sign the tube at the patient's bedside, complete the form, and contact the transfusion service. Only take blood from one patient at a time. Label tubes before leaving the bedside to minimize the risk of mislabelling. Blood banks will refuse to handle incorrectly labelled forms/tubes. If samples are handwritten, the lab usually requires a second sample from fresh venepuncture to be supplied before issuing cross-matched blood.

If you knowingly give a blood product (or an animal product, eg gelatin) to a patient whom you know would not accept this (eg a Jehovah's Witness), you are likely to face an indefensible medicolegal claim. Document verbal consent and the discussion regarding benefits, risks, and alternative treatments. Always consider giving alternatives to blood products (eg iron for iron deficiency anaemia).

What to request

When faced with major haemorrhage (eg major trauma or massive GI bleed), activate the Major Transfusion Protocol (see  Massive blood transfusion, p. 182). ED staff can obtain emergency O rhesus D-negative red cells (this may be O +ve red cells for ♂ patients), platelets, and FFP. Cross-matched blood will follow soon after, at which point, switch from transfusing O -ve to cross-matched blood. Take group and screen samples before giving emergency red cells.

Assessment of a patient with hypovolaemic shock is complex and includes recognition of the clinical situation and the potential blood loss, together with ongoing assessment of the patient and investigations. Hb and Hct values may be misleading—it may take hours for their values to equilibrate to indicate the degree of blood loss.

Group and screen The patient's ABO and rhesus D groups are determined and the serum tested for unexpected red cell antibodies. Subsequently, if required, electronically matched red cells can be provided within 5min, assuming the antibody screen is clear. Request group and screen where a patient does not need transfusion in the ED but may require it later.

Cross-match Full blood compatibility testing may take up to 1hr. If blood is required more urgently, ABO- and Rh-compatible units can usually be provided within 15min, including an 'immediate spin cross-match' as a final check on ABO compatibility. In exsanguinating haemorrhage, uncross-matched group O rhesus -ve blood can be issued immediately.

Blood products

Red cells (additive solution) Each pack (volume 300mL) derives from a single donor and has a Hct of 0.65–0.75 (0.55–0.65 for RBCs in additive solution). A transfusion of 4mL/kg will ↑ circulating Hb by ~10g/L.

Platelet concentrate Either pooled or from a single donor by plateletpheresis.

Fresh frozen plasma Contains clotting factors and fibrinogen.

Cryoprecipitate Derived from FFP when it is thawed. It is rich in factor VIII, fibrinogen, and vWF factor.

Prothrombin complex concentrate A combination of vitamin K-dependent factors II, VII, IX, and X. Use prothrombin complex concentrate to reverse warfarin.

Transfusion precautions

(See UK Blood Safety and Quality Regulations, 2005.)

- A practitioner must confirm all the following steps before commencing transfusion. If there is ANY discrepancy, DO NOT transfuse.
Note: some sites use two practitioners, but these should perform single independent checks (ie not simply reading numbers out to each other).
- Confirm the details on the traceability label on the blood component match the patient's full name, date of birth, and hospital number (all patients must be wearing a wristband before transfusion is given).
- Check that the traceability label is attached to the blood bag.
- Ensure the donation number and the patient's blood group/rhesus D type all match and that any special requirements are covered.
- Check every component before starting transfusion for signs of discolouration, leaks, clots, etc., and the expiry date.
- If all checks are satisfactory, ensure that the component has been prescribed (prescription form and/or fluid balance chart) and sign the front of the traceability label before commencing the transfusion.
- Infuse all components through a giving set with an integral filter to trap large aggregates. Microaggregate filters are not routinely required.
- Never add any drug to a blood component infusion.
- Do not use giving sets which previously contained glucose or gelatin.
- Use a blood warmer for large and/or rapid transfusions.
- Once the transfusion has started, peel off the portion of the signed label and attach to the appropriate place in the prescription chart (or fluid chart).
- Sign the prescription form to confirm the patient identity checks.
- Complete and sign the traceability label and return it to the laboratory.

(See  <https://www.transfusionguidelines.org>)

Routine prescribing rates

- For 'routine' transfusion, prescribe red cells to be given over 90min, unless there is a risk of transfusion-associated circulatory overload (TACO), in which case prolong it to 3.5hr.
- Platelets, FFP, and cryoprecipitate are usually given over 30min.