Advanced Life Support Group

Acute Medical Emergencies

The Practical Approach

SECOND EDITION

WILEY-BLACKWELL

Acute Medical Emergencies

Useful website addresses

Advanced Life Support Group	http://www.alsg.org
Best Evidence in Emergency Medicine	http://www.bestbets.org
Evidence based on-call	http://cebm.jr2.ox.ac.uk/eboc/eboc.html
Royal College of Physicians	http://www.rcplondon.ac.uk/

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Preface to second edition

Since its first publication in 2001, this text has been a key component of an educational package that has trained in excess of 600 providers in the management of acute medical emergencies. The course is now running in the UK, Eire, the Netherlands and Sweden.

Over this time, new guidelines and changes in practice have provided impetus to produce the second edition. The challenge of updating this with ever evolving guidelines has now been met. However, despite evidence-based developments and improvement in medical practice, the foundation of MedicALS – the structured approach – remains unchanged, reassuringly.

The second edition will continue to promote the essence of MedicALS, which is to ensure that the candidate leaves the course equipped with a 'Practical Approach' that is safe to patients with any kind of acute medical emergency.

As new or updated guidelines become available, we will provide those on the ALSG website www.alsg.org to ensure that your Acute Medical Emergencies text remains current.

Over the years, an increasing number of experts have contributed to the work and we extend our thanks both to them and to our instructors, who unceasingly provide helpful feedback. Consultation with instructors has greatly contributed to this new edition and to the revised course.

Preface to first edition

This book has been written to enable health care workers to understand the principles of managing an acute medical emergency safely and effectively. To achieve this aim it provides a structured approach to medical emergencies, describing relevant pathophysiology that will also help to explain physical signs and the rationale behind treatment. The first edition of this manual (written by Terry Wardle) has undergone significant modification directed by the working group and also, in particular, candidates from the first MedicALS courses. The requirements of these contributing doctors have meant that the contents and associated information may initially appear skewed – but this is evidence based.

Most textbooks are out of date by the time they are published – this manual is different in that it is both pragmatic and dynamic. Medicine is a rapidly evolving discipline and in order to ensure that this manual remains dynamic and up to date, reference web sites are available to ensure that the reader has constant access to relevant information. This will facilitate continual professional development that is the responsibility of the individual.

The book provides a structured approach that is applicable to all aspects of acute medicine, ensures the early recognition of signs of critical illness and will empower the individual to take immediate and appropriate action.

The text alone cannot provide all the necessary knowledge and skills to manage an acute medical emergency; therefore, readers are encouraged to attend the MedicALS course to further their theoretical and practical knowledge.

This book will continue to change to include new evidence-based practices and protocols to ensure a solid and safe foundation of knowledge and skills in this era of clinical governance.

Continued professional development is mandatory for all medical practitioners. This manual and the associated course will ensure both new knowledge acquisition and revision – and stimulate further learning.

Acknowledgments

We would like to thank members of faculty and candidates who have completed the MedicALS course for their constructive comments that have shaped both the text and the course.

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Finally, our thanks to Clare Duffy of ALSG and the staff at the Wiley-Blackwell for their ongoing support and invaluable assistance in the production of this text.

PART I
Introduction

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CHAPTER 1
Introduction

INTRODUCTION

After reading this chapter you will understand:
- the current problems in the assessment of acute medical emergencies
- the need for a structured approach to the medical patient.

THE PROBLEM

A medical emergency can arise in any patient, under a variety of circumstances, e.g.:
- previously fit
- acute on chronic illness
- post-surgical
- precipitating or modifying the response to trauma.

The acute problem can be directly or indirectly related to the presenting condition, an associated complication, any treatment and/or the result of inappropriate action.

Key point

Inappropriate action costs lives

Furthermore, with the increase in the elderly population there is a corresponding increase in the number and complexity of medical problems. The management of such patients is compromised by conflicting demands such as financial constraints, limited bed availability, workforce availability and increased medical specialisation.

For the last few years there has been an annual increase of emergency admissions in excess of 5%. These account for over 40% of all acute National Health Service beds. In the UK the mean hospital bed complement is 641, but only 186 are allocated for medical patients, with an average of 95% of these housing patients admitted as emergencies.

The common acute conditions can be broadly classified according to the body system affected (Table 1.1).

This information may be broken down further to reveal the common reasons for admission:
- myocardial infarction
- stroke
- cardiac failure
- acute exacerbation of asthma

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Table 1.1 Classification of medical emergencies

Type	%
Cardiac	29
Respiratory	26
Neurological	21
Gastrointestinal	13

- acute exacerbation of chronic obstructive pulmonary disease
- deliberate self-harm.

Despite the fact that these are common conditions, frequent management errors and inappropriate action result in preventable morbidity and mortality.

A recent risk management study examined the care of medical emergencies. One or more avoidable serious adverse clinical incidents were reported. Common mistakes are listed in the box below.

Common mistakes

Failure to recognise and treat serious infection
Error in investigating – acute headache
– acute breathlessness
– epilepsy
Misinterpretation of investigations
Inadequate assessment of abdominal symptoms

This was only a small study but of the 29 patients who died, 20 would have had a good chance of long-term survival with appropriate management. In addition, out of the 11 patients who survived, 3 were left with serious neurological defects, 3 had avoidable intestinal resection and 4 patients suffered unnecessarily prolonged hospital admission.

The overall problems were identified as follows:

- Medical emergencies were not assessed by sufficiently experienced staff.
- A second opinion was not obtained.
- Assessment was inadequately performed before discharge.
- X-rays were not discussed with radiologists.
- Protocols were not used for standard conditions.

Diagnostic errors were made in 80% of patients because of inadequate interpretation of the clinical picture and initial investigations. Errors in patient assessment are listed in the box below.

Errors in patient assessment

Available clinical evidence incorrectly interpreted
Failure to identify and focus on very sick patients
Investigations misread or ignored
Radiological evidence missed
Standard procedures not followed
Inadequate assessment and/or treatment
Discharge from hospital without proper assessment

Furthermore, the assessment of medical patients requiring intensive care was either incomplete, inappropriate or too late to prevent increased morbidity and mortality.

Therefore, there are problems in the **fundamental** areas of medical patient care, i.e. clinical examination, requesting and interpreting appropriate investigations and communication. However, probably most important of all is knowing when and who to ask for help. One answer to this important problem is to provide a structured approach to patient assessment that will facilitate problem identification and prioritise management.

All that is required to manage medical emergencies is the application of focused knowledge and basic skills. These will ensure prompt accurate assessment and improve patient outcome. Avoidable deaths are due to inappropriate management, indecision or delays in assessment and/or treatment. In the study the average time for initial review after admission is 30 minutes, with a further 130 minutes passing before definitive management occurs.

In the UK, numerous studies have shown that specialist care is better than that provided by a generalist; e.g. prompt review by a respiratory physician has been shown to reduce both morbidity and mortality from asthma. The mortality from gastrointestinal haemorrhage falls from 40% to approximately 5% if the management is provided by a specialist in gastroenterology. Further, supportive evidence has been provided by studies in the US, where mortality from myocardial infarction or unstable angina was greater in patients managed by generalists.

However there are insufficient numbers of 'specialists' to manage all of these conditions. Besides, patients with sudden deterioration in their condition often present as 'undifferentiated medical emergencies', without a clear 'label' identifying which particular specialist is required. Some will require review by a general physician, whilst others will be managed at least initially by colleagues in the rapidly expanding, exciting discipline of acute medicine.

Thus, physicians need to know how to manage medical emergencies. This course will teach a structured approach for assessment that will enable you to deliver safe, effective and appropriate care.

Traditional medical teaching dictates that a history should always be taken from the patient before the clinical examination. This will subsequently allow a diagnosis or differential diagnosis to be postulated and dictate the investigations required. Unfortunately this approach is not always possible; e.g. trying to obtain a history from a patient who presents with breathlessness may not only exacerbate the condition but also delay crucial therapy.

This course has been developed by observing how experienced physicians manage medical emergencies. The results have shown quite an interesting cultural shift. Most of us, as we approach the patient, quickly scan for any obvious physical signs, e.g. breathlessness, and then focus our attention on the symptoms until the diagnosis is identified. Only when the patient's symptoms have been improved can a history be taken and the remainder of the examination performed. This process has been refined and formalised to produce a structured approach to patient assessment so that the most immediately life-threatening problems are identified early and treated promptly. Thus, this structured approach considers the conditions that are most likely to kill the patient.

All other problems will be identified subsequently as part of the overall classical approach to the medical patient, i.e. taking a comprehensive history and examining the patient fully. Being aware at all time that should the patient deteriorate a reassessment should start at the beginning.

The key principles of MedicALS are shown in the box.

Key principles of MedicALS

Do no further harm
Focused knowledge and basic skills are essential for doctors dealing with acute emergencies
A structured approach will identify key problems and prioritise management
Prompt accurate assessment and treatment improves patient outcome

SUMMARY

The number and complexity of acute medical emergencies are increasing along with the potential for medical mishaps. Typically these result from a failure to assess acutely ill patients, interpret relevant investigations and provide appropriate management. This manual, and the associated course, will equip you with a structured approach to deal with these patients.

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CHAPTER 2

Recognition of the medical emergency

OBJECTIVES

After reading this chapter you will be able to:

- understand the clinical features of potential respiratory, cardiac and neurological failure
- describe these clinical features and use them to form the basis of the primary assessment.

Irrespective of the underlying pathology, the acutely ill medical patient who dies does so from failures of the respiratory, circulatory or central neurological systems separately or in combination. It is of paramount importance that the physician can recognise potential failure of these systems, as early intervention will reduce morbidity and mortality. The ultimate failure, a cardiorespiratory arrest, can often be predicted in the hospital setting as it is generally preceded by a period of physiological deterioration.

This chapter will provide an overview of the clinical assessment of patients with potential respiratory, circulatory and neurological failure. The chapters in Part II will then use this format to develop an in-depth assessment that produces a structured approach to the patient with a medical emergency. An underlying principle of the assessment system described below is that it is physiologically based rather than using the more classical format of history taking, examination and investigation.

Time Out 2.1

Think about a patient you have treated recently who was critically ill, and reflect on the good and/or bad aspects of their treatment. List the staff involved and assessments that took place during the management of this patient.

Draw a timeline and place the information from your lists on the line. At the end of the line, write down the outcome of the episode.

RECOGNITION OF POTENTIAL RESPIRATORY FAILURE

This can be assessed by examining the respiratory rate, effort of respiration and effectiveness of ventilation, as well as the effects of respiratory inadequacy.

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Respiratory rate

The normal adult respiratory rate is 14–20 breaths/min. Variation outside this range is an indication of potential respiratory failure. Tachypnoea (greater than 30 breaths/min at rest) generally indicates that increased ventilation is needed because of hypoxaemia associated with disease affecting the airway, breathing or circulation. It can also indicate compensatory hyperventilation due to a metabolic acidosis associated with a non-respiratory problem. Similarly, a respiratory rate of less than 10 breaths/min is an indication of respiratory fatigue or loss of central respiratory drive, both potentially requiring ventilatory support.

Effort of respiration

Assessment of effort gives an indication of how hard a patient is working to breathe. If the patient can count to 10 in one breath, there is usually no significant underlying respiratory problem. Features which suggest increased respiratory effort are tachypnoea, intercostal and subcostal recession and accessory muscle use.

Effectiveness of ventilation

Effectiveness of ventilation is assessed by measurement of chest expansion, percussion and auscultation. Chest expansion indicates the volume of air being moved during the respiratory cycle.

The presence or absence of breath sounds allows assessment of airflow to specific areas of both lung fields. Any asymmetry should be noted. Pathology is generally on the side of abnormal signs.

Any added sounds should be noted. Stridor is a loud inspiratory noise and is indicative of laryngeal/tracheal narrowing or obstruction. During auscultation you may hear wheezing and/or a prolonged expiratory phase due to lower airway narrowing.

> **Key point**
>
> **A silent chest is an extremely worrying sign**

Oxygen saturation

Pulse oximetry is used to measure the arterial oxygen saturation (SpO_2). It is inaccurate in the following circumstances:

- $SpO_2 < 70\%$
- poor peripheral perfusion
- in the presence of methaemoglobin or carboxyhaemoglobin.

Effects of respiratory inadequacy on other organs

Heart rate

Hypoxaemia initially produces a tachycardia. These changes are non-specific as other causes such as anxiety, fever or shock may coexist. However, severe or prolonged hypoxaemia will eventually lead to a bradycardia – a preterminal sign.

Skin colour

Hypoxaemia, via catecholamine release, produces vasoconstriction and hence skin pallor. Decreased oxygen concentration will lead to cyanosis as haemoglobin becomes deoxygenated. Central cyanosis in acute respiratory disease is indicative of imminent respiratory arrest. In the anaemic patient, cyanosis may be

difficult to detect despite profound hypoxaemia, because the reduced total amount of haemoglobin may mean there is not enough deoxygenated haemoglobin to produce the cyanotic colour.

Mental status

The hypoxaemic patient will initially appear agitated and eventually will become drowsy. Similar features will also occur with hypercapnoea; in this situation the patient will be vasodilated and have a flapping tremor (asterixis).

RECOGNITION OF POTENTIAL CIRCULATORY FAILURE

Acute circulatory failure can also be defined as shock. Although this has multiple causes, during the initial assessment the overriding priority is to identify shock, rather than find a specific cause.

Circulatory failure is assessed by examining the heart rate, effectiveness of circulation and the effects of shock on other organs.

Heart rate

This increases in the shocked patient due to catecholamine release, generally secondary to a decreased circulatory volume in an effort to increase cardiac output. There are many reasons why a normal adult may experience a tachycardia (pulse rate $>$ 100/minute) and other signs should be sought to confirm the clinical suspicion of circulatory failure.

Be aware that certain drugs (e.g β blockers) can prevent a compensatory tachycardia and very fit patients, in whom a pulse rate of 100/minute may be twice their resting pulse rate.

Effectiveness of circulation

Blood pressure

Compensatory mechanisms will try to maintain blood pressure. Consequently, during the early stages of shock it may be normal or even elevated. For this reason, blood pressure should not be used as the sole indicator of circulatory status. Hypotension in circulatory failure is an indicator of increased mortality. As the Blood pressure is such an important parameter always ensure that an appropriate cuff size is used when it is measured.

Pulses – central and peripheral; pulse volume

Although blood pressure is generally maintained until shock is very severe (loss of at least one third of the circulating volume), a rapid assessment of perfusion can be gained by examining peripheral and central pulses. The combination of absent peripheral pulses and weak central pulses is a sinister sign indicating advanced shock and profound hypotension.

Perfusion

Pressure on a central area (e.g sternum) for 5 s should normally produce a capillary refill within 2 s. A prolonged refill time indicates poor skin perfusion, a sign of shock. This sign is unreliable in hypothermic patients.

Effects of circulatory inadequacy on other organs

Respiratory system

A rapid respiratory rate with an increased tidal volume, without signs of increased respiratory effort, is predominantly caused by a metabolic acidosis associated with circulatory failure.

Skin
Mottled, cold and pale skin especially at the peripheries is an indicator of poor perfusion.

Mental status
Agitation, confusion, drowsiness and unconsciousness are the progressive stages of mental dysfunction associated with circulatory failure due to poor cerebral perfusion.

Urinary output
A urine output of less than 0.5 ml/kg/h indicates inadequate renal perfusion.

RECOGNITION OF POTENTIAL CENTRAL NEUROLOGICAL FAILURE

Maintaining adequate central neurological function is a priority during resuscitation of a critically ill patient. Both respiratory and circulatory failure will affect the assessment of neurological function, and must therefore be addressed before central neurological assessment.

Initial assessment is directed at global rather than specific function. Conscious level, posture, asymmetrical motor signs and pupillary response should be evaluated.

Conscious level
A rapid assessment of the patient's conscious level can be made by assigning the patient to one of the categories shown in the box.

AVPU grading of consciousness

A = Alert
V = response to Voice
P = response to Pain
U = Unresponsive

A painful stimulus should be applied by pressure over the supraorbital ridge on the superior orbital nerve. An adult who either responds only to pain (P) or is unresponsive (U) has a significant degree of coma equivalent to 8 or less on the Glasgow Coma Scale. These patients are at risk of losing control of their airway.

Posture
Abnormal posturing such as decorticate (flexed arms, extended legs) or decerebrate (extended arms, extended legs) is a sinister sign of brain dysfunction. A painful stimulus may be necessary to elicit these signs. Determine also if there is a difference in motor response between the right and left sides as this indicates a localised neurological disorder.

Pupils
The most important pupillary signs to seek are dilation, unreactivity and inequality. These indicate possible serious brain disorders. Many drugs and cerebral lesions have effects on pupil size and reaction.

Respiratory effects of central neurological failure on other systems

There are several recognisable breathing patterns associated with raised intracranial pressure. However, they are often changeable and may vary from hyperventilation to periodic breathing and apnoea. The presence of any abnormal respiratory pattern in a patient with coma suggests brain stem dysfunction.

Circulatory effects of central neurological failure

Systemic hypertension with sinus bradycardia and erratic respiration (Cushing's triad) indicates compression of the medulla oblongata caused by herniation of the cerebellar tonsils through the foramen magnum. This is a late and preterminal sign.

Time Out 2.2

Refer back to the timeline you drew in Time Out 2.1.

Using the system described in this chapter, draw out an ideal timeline for your patient's assessment.

Do you think that this system would have any benefits to the patient's care you charted and particularly to the outcome you wrote at the end of the line?

On your ideal timeline, highlight the number of features that assess more than one system.

Finally, check that your list of clinical features is in a logical order to produce a rapid system for assessment of a critically ill patient.

SUMMARY

In the acutely ill medical patient a rapid examination will detect potential respiratory, circulatory and neurological failure. The clinical features are:

- respiratory – rate, effort and effectiveness of respiration
- circulatory – heart rate and effectiveness of circulation
- neurological – conscious level, posture and pupils.

These features will form the framework of the primary assessment. The components will be discussed in detail in Part II.

PART II
Structured Approach

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CHAPTER 3

A structured approach to medical emergencies

OBJECTIVES

After reading this chapter you will be able to describe the:

- correct sequence of priorities to be followed when assessing an acutely ill medical patient
- primary and secondary phases of assessment
- key components of a patient's history
- techniques used in the initial resuscitation, investigation and definitive care of a medical emergency.

INTRODUCTION

The management of a patient with a medical emergency requires a rapid assessment with appropriate treatment of life-threatening conditions as and when they are found. This can best be achieved using a structured approach. This chapter will give an overview of this approach and each component will be examined in greater detail in subsequent chapters.

Structured approach

Primary assessment and resuscitation
Secondary assessment and emergency treatment
Reassessment
Definitive care

Key point

The aim of the primary assessment is to identify and treat any immediately life-threatening conditions with minimum delay and in a prioritised fashion. Most acutely ill medical patients (75%) do not have an immediately life-threatening problem. However, a rapid primary assessment is still required.

The primary assessment should be repeated immediately in the event of any deterioration in the patient's condition. This allows early appropriate resuscitation to reverse the deterioration. Be prepared to act on a strong clinical suspicion as your intervention will be likely to reverse or halt a deterioration.

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Remember that certain classical signs taught in medical school may be difficult to confirm in a noisy resuscitation area (e.g. quiet heart sounds in life-threatening cardiac tamponade).

Once any immediately life-threatening conditions have been either identified and treated, or excluded (i.e. primary assessment and resuscitation), you can then take a comprehensive history and complete a thorough examination (i.e. secondary assessment and emergency treatment). Following any emergency treatment the patient should be reassessed. Definitive care can then be planned including transportation to the appropriate ward and further investigation.

PRIMARY ASSESSMENT AND RESUSCITATION

> **Key point**
>
> **The aim of the primary assessment is to identify and treat all (there may be more than one) immediately life-threatening conditions**

> **Key point**
>
> **Always use universal precautions before assessing an acutely ill patient**

Key components of the primary assessment (ABCDE) are assessment and management of:

A – Airway
B – Breathing
C – Circulation
D – Disability
E – Exposure

A – Airway

Aims = assess patency and identify any imminent threat, e.g. mucosal oedema in anaphylaxis. If necessary, clear and secure the airway
= administer high concentrations of inspired oxygen
= appreciate the potential for cervical spine injury

Assessment

Assess airway patency by talking to the patient. An appropriate response to 'Are you okay?' indicates that the airway is clear, the patient is breathing and has adequate cerebral perfusion. If no answer is forthcoming, then open the airway with a chin lift or jaw thrust and reassess patency by:

- looking – for chest movement
- listening – for the sounds of breathing
- feeling – for expired air.

A check for upper airway obstruction should include inspection for foreign bodies, including dentures, and macroglossia.

Resuscitation

If a chin lift or jaw thrust is needed, then an airway adjunct may be required to maintain patency. A nasopharyngeal airway is useful in the conscious patient. In contrast an oropharyngeal (Guedel) airway is typically used as a temporary adjunct in the unconscious patient before airway protection is achieved by endotracheal intubation.

Whilst gaining definitive control of the airway, supplemental oxygen should be given to all patients who are breathless, shocked, bleeding or suspected to be acutely ill from any cause. If the patient is not intubated, oxygen should be given using a non-rebreathing mask and reservoir. This enables the fractional inspired oxygen (FiO_2) concentration to reach a level of up to 0.85. All critically ill patients should receive high concentrations of inspired oxygen. For the purposes of this text and course, critically ill is defined as a patient with an early warning score of 3 or above (see Table 3.3). Even critically ill patients who have chronic obstructive pulmonary disease should receive high concentrations of inspired oxygen initially; this can subsequently be reduced according to its clinical effect and arterial blood gas results. You therefore need to maintain close observation of these patients until the optimum FiO_2 is determined.

Cervical spine problems are very rare in medical patients – except in those with rheumatoid disease, ankylosing spondylitis and Down's syndrome. The clinical features of these conditions are usually easily identifiable. However, be wary of the elderly patient found collapsed at the bottom of the stairs after an apparent 'stroke'. If you suspect cervical spine injury, ask for immediate help to provide in-line immobilisation.

Key point

Hypoxaemia kills

Hypercarbia is not a killer, provided the patient is receiving supplemental oxygen in a high dependency setting

Monitoring

Arterial oxygen saturation (SpO_2) monitoring is essential. End tidal carbon dioxide ($ETCO_2$) should be measured after endotracheal intubation, to check correct tube placement and alert you in the event of subsequent tube displacement.

See Chapter 4 for further details.

B – Breathing

Aim = detect and treat:

–life-threatening bronchospasm
–pulmonary oedema
–tension pneumothorax
–the presence of critical oxygen desaturation

Assessment

A patent airway does not ensure adequate ventilation. The latter requires an intact respiratory centre, adequate pulmonary function and the coordinated movement of the diaphragm and chest wall.

Chest inspection

Colour/marks/rash
Rate
Effort
Symmetry

Look for cyanosis, respiratory rate and effort, and symmetry of movement. Feel for tracheal tug or deviation. Tracheal deviation (in a distressed or unconscious patient) indicates mediastinal shift (consider tension pneumothorax and decompress immediately if suspected). Percuss the anterior chest wall in the upper, middle and lower zones, assessing the difference between the left and right hemithoraces. Repeat this on the posterior chest wall and axillae to detect areas of hyper-resonance (air), dullness (interstitial fluid) or stony dullness (pleural fluid). Listen to the chest to establish whether breath sounds are absent, present or masked by added sounds. Further information regarding oxygen saturation of blood will be provided by a pulse oximeter.

Key point

Some physical signs will be elicited that suggest a non-breathing cause for respiratory difficulty. Thus corroborative evidence must be sought to confirm a clinical diagnosis of, e.g., left ventricular failure.

Key point

Remember that if your patient is lying down:
The physical signs might be harder to elicit, e.g. dullness to percussion from an effusion will be posterior
Back examination must occur at some stage!

Resuscitation

Treat life-threatening bronchospasm as soon as it is identified, with nebulised salbutamol (β_2-agonist) and ipratropium bromide (muscarinic antagonist).

A tension pneumothorax requires urgent decompression with a needle thoracocentesis, followed by intravenous access and chest drain insertion.

Further clues to the cause of apparent respiratory difficulty may be found on examination of the patient's circulation.

Monitoring

Arterial oxygen saturation (SpO_2) should be monitored continuously.

Respiratory rate

See Chapter 5 for more comprehensive details.

C – Circulation

Aim = detect and treat shock

Assessment

Measure cardiovascular indices and level of consciousness. Examine a central pulse (ideally the carotid), for rate, rhythm and character. Compare both carotid pulses, but not simultaneously. A reduction or absence in one pulse may reflect focal atheroma or a dissecting aneurysm. Measure the blood pressure and assess peripheral perfusion using the capillary refill time. Assess the height (and character) of the JVP (Jugular Venous Pulse), the apex beat and listen to the heart sounds and for any extra sounds.

Hypotension indicates established decompensation, requiring prompt action to prevent shock becoming irreversible. A normal blood pressure in the young and previously healthy can be maintained despite well-established shock. Look for narrowing of the pulse pressure. This occurs when the systolic blood pressure is 'propped up' by the neuroadrenergic (catecholamine) response to a reduced stroke volume raising the diastolic blood pressure (increased tone/resistance).

Reduced blood volume can impair consciousness due to reduced cerebral perfusion.

Some causes of shock require specific treatment, e.g. adrenaline for anaphylaxis

Resuscitation

Intravenous access is needed in all acutely ill patients. If there is a suspicion of hypovolaemic shock, two large-bore cannulae should be inserted. The antecubital fossa is usually the easiest and most convenient site. Take blood for baseline haematological and biochemical values (including a serum glucose) and, in appropriate cases, a cross-match. You should strongly consider taking arterial blood gases to look for ventilatory inadequacy and metabolic acidosis in particular.

Key point

Fluid, antibiotics, glucose, adrenaline, inotropes, antidotes and electricity are crucial in the management of different types of shock

Hypovolaemic

If you suspect hypovolaemia (depletion of intravascular volume), give a fluid challenge of 2 litres of Ringer's lactate in 500 ml boluses. Further fluid requirements will be determined by the patient's response. If, after 2 litres of fluid, the patient remains hypotensive and haemorrhage is suspected, then blood and a **surgeon** are needed urgently.

Cardiogenic

A similar pale, cold and clammy picture will be found in cardiogenic shock. The presence of pulmonary oedema is a useful differentiating factor. Non-invasive positive pressure ventilation and/or inotropes will be required in this case. Consider emergency primary coronary intervention if myocardial infarction is the cause.

Cardiac rhythm

Cardiac rhythm disturbance causing haemodynamic instability should be treated according to UK and European resuscitation guidelines. This patient will almost certainly require sedation and cardioversion.

Septic

The hypotensive, warm, vasodilated and pyrexial patient is 'septic' until proven otherwise. Look for the non-blanching purpuric rash of meningococcal septicaemia. This condition, if suspected, requires immediate treatment with intravenous benzyl penicillin 2.4 g and ceftriaxone 1 g. Investigations should include blood cultures, C-reactive protein and blood for meningococcal polymerase chain reaction after initial resuscitation.

Diabetic keto acidosis (DKA)

Look (and smell) for diabetic ketoacidosis (raised blood glucose, ketonuria and acidaemia) in the tachypnoeic, dehydrated and hypotensive patient.Following confirmation of the diagnosis, treatment should be started as described in chapter 20.

Anaphylactic

Shock due to anaphylaxis is treated according to the UK and European resuscitation guidelines (see Chapter 9).

Occasionally, shock may have more than one cause. Dehydration is common in acute medical emergencies. If there is no evidence of either ventricular failure or a dysrhythmia, all patients should receive a fluid challenge (200–300 ml immediately). Subsequent management will depend on the patient's response and blood test results.

Monitoring

Continuous monitoring of oxygen saturation (pulse oximetry), pulse, blood pressure and ECG provides valuable baseline information about the patient and the response to treatment (reassessment). Consider a urinary catheter to monitor urinary output, in the shocked patient as this provides a useful indicator as to the adequacy of resuscitation.

See Chapter 6 for more comprehensive details.

D – Disability (neurological examination)

Aim = to detect and treat any immediately life-threatening neurological condition (e.g. prolonged fit, hypoglycaemia, opioid overdose, infection or suspected cerebral ischaemia).

Assessment

Measure pupillary size and reaction to light. Evaluate the conscious level, using either the AVPU system (see Chapter 2) or more commonly the Glasgow Coma Score (Table 3.1). Check the patient's posture and for the presence of lateralising signs in the limbs. Examine for signs of meningeal irritation. Remember FAST; **F**ace, **A**rms, **S**peech, **T**ime as pre-hospital indicators of a potential stroke.

Check serum glucose with either a glucometer or a BM stix in the presence of any neurological dysfunction. Hypoglycaemia is common, readily detectable, easily treatable and has serious implications if therapy is delayed. Hypoglycaemia should be treated immediately, once a venous blood sample has been taken for definitive glucose measurement.

Resuscitation

In the unconscious patient, it is vital to clear and secure the airway. Give supplemental oxygen until further clinical information and the results of investigations

Table 3.1 The Glasgow Coma Scale

Eye Opening	
Spontaneous	4
To speech	3
To painful stimuli	2
Nil	1
Best Verbal Response	
Orientated	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
Nil	1
Best Motor Response	
Obeys commands	6
Localises pain	5
Withdraws from pain	4
Abnormal flexion	3
Abnormal extension	2
Nil	1

Note: If there is focal limb weakness, the best motor response should be recorded.

are available. Prevent secondary brain injury by ensuring optimum management of A, B and C.

'Tonic–clonic' seizures usually resolve spontaneously within a minute or so. Ensure that the patient has a patent airway, is receiving supplemental oxygen and that vital signs are monitored regularly. Place the patient in the recovery position to prevent aspiration and injury on any adjacent objects. It is not uncommon to misinterpret reduction of tonic–clonic movements as the cessation of seizures. If you think the patient has stopped convulsing, check for ease of passive eye opening and absence of abnormal eye movements. Remember to check for hypoglycaemia.

If the fit is prolonged (longer than 2–5 min, depending on the patient's condition), give intravenous benzodiazepines, e.g. lorazepam 4 mg over 2 min (repeat after 10 min if the patient is still fitting), or increments of 2.5 mg of diazemuls (to a maximum of 20 mg). If benzodiazepines fail to control the fit, start intravenous phenytoin at 15 mg/kg over 30 min with ECG monitoring. This drug does not impair the conscious level and will facilitate early neurological assessment. An alternative is fosphenytoin (18 mg/kg phenytoin equivalent IV up to 150 mg/min). If this combination fails to control fitting, request urgent assistance from an anaesthetist regarding rapid sequence induction.

In hypoglycaemia, an infusion of 10% dextrose and/or intravenous glucagon (1 mg) is immediately necessary to prevent recurrence. If underlying alcohol use is suspected, give intravenous thiamine as well.

The unconscious patient showing signs of opioid excess (small and unreactive pupils) should be treated with intravenous naloxone 0.2 mg.

The unconscious or confused patient will need a CT brain scan. However, this must not delay antibiotic and/or antiviral treatment for suspected meningitis/encephalitis, or any other necessary resuscitation (including intubation to

protect the airway, if necessary). As a general rule, unstable and/or inadequately resuscitated patients should not be moved to places of lesser safety (such as CT scan area), without the consultant in charge of the resuscitation agreeing that it is necessary and appropriate.

Patients with suspected cerebral ischaemia should have an early CT and, if indicated, should receive thrombolysis within 4 h.

Monitoring

Glasgow Coma Score, pupillary response and serum glucose.

See Chapter 7 for more comprehensive details.

E – Exposure

Aim = examine the entire patient and prevent hypothermia.

Severe life-threatening skin conditions are associated with problems in 'C', but also occasionally in A, B and D. These include hypovolaemia, vasodilatation, loss of temperature control and risk of infection. Hence you should have already treated these by the time you treat E.

Assessment

Examine for three important rashes (the non-blanching purpura of meningococcal septicaemia, erythroderma and blistering eruptions). Other physical signs may include bleeding or bruising (coagulopathy), injury, swelling and infection. Do not forget to look for needle marks.

Resuscitation

Patients should have received intravenous fluids and antibiotics, if indicated, earlier in the primary assessment. Urgent referral to a dermatologist may be necessary to guide further management and investigation.

Monitoring

- Temperature

It is impossible to do a comprehensive examination unless the patient is fully undressed. However, care must be taken to prevent hypothermia, especially in elderly patients. Therefore, adequately cover patients between examinations and ensure all intravenous fluids are warmed.

MONITORING

The effectiveness of resuscitation is measured by an improvement in the patient's clinical status. It is therefore important that repeat observations are measured and recorded frequently. Table 3.2 shows the minimum level of monitoring required by the end of the primary assessment in an acutely unwell patient.

It is important to reassess the patient regularly, especially after treatment has been started. This will ensure that the patient has responded appropriately, and not deteriorated.

Key point

The most important assessment is the reassessment

Table 3.2 Minimum patient monitoring in an acutely unwell patient

Pulse oximetry
Respiratory rate
Blood pressure
Continuous ECG monitoring, augmented by a 12-lead ECG
Chest X-ray when appropriate
Arterial blood gases when appropriate
Core temperature
Central venous pressure when appropriate
Glasgow Coma Score, lateralising signs and pupillary response
Urinary output

The majority of medical patients will only require a brief primary assessment, to establish that there is no need for aggressive resuscitation. In clinical practice, the usual patient–doctor introduction will provide a rapid assessment of the A, B, Cs. A patient who is sitting up and talking has a patent airway and sufficient cardiorespiratory function to provide oxygenation and cerebral perfusion.

A variety of scoring systems can be used to assess the acutely ill patient rapidly as a measure of' 'how at risk the patient is'. These are based on the B, C, D and E components:

- Respiratory rate
- Pulse rate
- Systolic blood pressure
- Mental response
- Temperature

One such system, the Early Warning Score, is shown in Table 3.3. Scores may trigger actions at different levels in different settings.

In addition, the urine output can be included in patients who are catheterised. Each component is scored between 0 and 3. A patient who has a score of 3 for one component or 4 or more for a combination of components needs a more detailed assessment before physiological deterioration becomes too profound. Local protocols will dictate who does this detailed assessment, e.g. junior doctor, member of the outreach team or critical care team.

Where the early warning score is lower than that described above, one can skip quickly to the traditional style of history taking followed by a physical examination. This is referred to as the secondary assessment.

Table 3.3 Example early warning scoring system

Score	3	2	1	0	1	2	3
Respiratory rate		≤9		10–14	15–20	21–30	>30
Heart rate		<40	40–50	51–100	101–110	111–130	>130
Blood pressure (systolic)	≤70	71–80	81–100	101–199		≥200	
Central nervous system			SC	A	V	P	U
Temperature		<35		35–38		>38	
Urine output (ml/h)	Nil	<30	30–39	40–99	100–150	151–199	≥200

SC = sudden confusion; A = alert; V = responds to voice; P = responds to pain; U = unresponsive

Time Out 3.1

After reading the case history, answer the following question:

A 54-year-old man is referred to the medical assessment unit because of an acute onset of confusion.

Briefly describe your primary assessment of this patient.

SECONDARY ASSESSMENT

The aims of the secondary assessment are to identify and treat all conditions not detected in the primary assessment, seek corroborative evidence to formulate a provisional diagnosis and prioritise the patient's management.

The secondary assessment starts once vital functions have been stabilised and immediately life-threatening conditions have been identified and treated.

History

Nearly all medical diagnoses are made after a good history has been obtained from the patient. Occasionally, for a variety of reasons this may not be possible. Therefore information should be sought from relatives, the patient's medical notes, the general practitioner, friends or the police and ambulance service. A well-'phrased' history is required, and also serves as a useful mnemonic to remember the key features.

A well-'phrased' history

P Problem
H History of presenting problem
R Relevant medical history
A Allergies
S Systems review
E Essential family and social history
D Drugs

The history of the presenting problem is of paramount importance. A comprehensive systems review will ensure that significant, relevant information is not excluded. In addition, it will ensure that the secondary assessment focuses on the relevant systems.

Examination

Aims = find new features – often related to clues in the history
= comprehensively reassess conditions identified in the primary assessment
= seek corroborative evidence to support findings from the primary assessment and to formulate a diagnosis

The examination should be directed by the history and primary assessment findings. It is a methodical, structured approach comprising a general overview and the detection of specific features.

General

A clinical overview of the patient's overall appearance 'from the end of the bed' can give clues to underlying pathology.

Clinical overview

Posture
Pigmentation
Pallor
Pattern of respiration
Pronunciation
Pulsations

Specific features include the following.

Hands

Inspect the hands for stigmata of infective endocarditis, chronic liver disease, thyrotoxicosis, carbon dioxide retention, polyarthropathy and multisystem disease. Palpate the radial pulse for rate, rhythm, character and symmetry, comparing it to the contralateral radial pulse and the femoral pulse.

Face

Examine for facial asymmetry, cyanosis, and the presence of any pigmentation, stigmata of hyperlipidaemia, titubation, and cutaneous features of internal pathology. Inspect the mouth, tongue and pharynx for the presence of ulcers, blisters, vesicles and erythema. Pigmentation of the buccal mucosa should be specifically sought (Addison's disease is an uncommon cause of collapse often associated with a delay in making the diagnosis).

Neck

Assess the height, waveform and characteristics of the internal jugular venous pulse. Palpate both internal carotid arteries in turn to compare and determine the pulse character. Check the position of the trachea and the distance between the suprasternal notch and the inferior aspect of the thyroid cartilage. A distance of less than 3 finger breadths indicates hyperexpansion of the chest. Feel for lymphadenopathy.

Chest

Assess the shape of the chest and breathing pattern. Recheck the rate, effort and symmetry of respiration. Look for surgical scars. Palpate the precordium to determine the site and character of the apex beat, the presence of a left and/or right ventricular heave, and the presence of thrills. Listen for the first, second and any additional heart sounds; and murmurs. Percuss the anterior and posterior chest walls bilaterally in upper, middle and lower zones comparing the note from the left and right hemithoraces. Auscultate these areas to determine the presence, type and quality of breath sounds as well as any added sounds. Check for evidence of peripheral oedema.

Abdomen

Systematically examine the abdomen according to the nine anatomical divisions. Specific features that should be sought include hepatosplenomegaly, peritonism/itis, abdominal masses, lymphadenopathy, ascites as well as renal angle tenderness. In appropriate cases examine the hernial orifices, external genitalia and rectum.

Locomotor

Inspect all joints and examine for the presence of tenderness, deformity, restricted movement, synovial thickening and inflammation. The patient's history, however, will indicate the joints that are affected. Although inflammatory polyarthropathies may present suddenly, acute monoarthropathies are potentially more sinister (see Chapter 17). Septic arthritis is an emergency that quickly destroys a joint if not diagnosed and treated.

Neurological

A comprehensive neurological examination is rarely required in the acutely ill patient. A screening examination of the nervous system can be accomplished as follows:

1. Assess the conscious state using the Glasgow Coma Scale.
2. A Mini Mental State Examination (see next box).
3. Examine the external ocular movements for diplopia, nystagmus or fatiguability. Elicit the pupillary response to light and accommodation (PERLA, i.e. pupils equally react to light and accommodation). Examine the fundi. The absence of dolls eye movement (oculocephalic reflex) indicates a brain stem problem. This is obviously only relevant in the unconscious patient and should not be elicited if there is a suspicion of cervical spine instability. Assess muscles of mastication and facial movement followed by palatal movement, gag reflex and tongue protrusion. When appropriate check the corneal reflex and visual fields (see Chapter 7).
4. Test the tone of all four limbs, the power of muscle groups, reflexes (including the Plantar/Babinski response) and coordination.
5. Sensory testing, although subjective, is useful in the acute medical setting, especially when a cord lesion is suspected.
6. Further neurological examination will be dictated by the patient's history and the examination findings, especially from the screening neurological assessment.

Skin

The skin and the buccal mucosa must be thoroughly inspected. Lesions may be a manifestation of internal pathology (e.g. buccal pigmentation in Addison's disease).

REASSESSMENT

The patient's condition should be monitored to detect any changes and assess the effect of treatment. If there is any evidence of deterioration, re-evaluate by returning to A in the primary assessment.

Some patients presenting with an apparent medical problem may require urgent specialist intervention to save their life, e.g. an early surgical opinion when treating patients with upper gastrointestinal haemorrhage.

No.	Question	Assessment	Rating
1	How old are you?	Score for exact age only	
2	What is your date of birth?	Only date and month needed	
3	What is the year now?	Score for exact year only	
4	What is the time of the day?	Score if within 1 h of correct time	
5	Where are we? What is this building?	Score for exact place name, e.g. 'hospital' insufficient	
Now ask subject to remember an address: 42, West Street			
6	Who is the current monarch?	Score only current monarch	
7	What was the date of the First World War?	Score for year of start or finish	
8	Can you count down backwards from 20 to 1?	Score if no mistakes or any mistakes corrected spontaneously	
9	Can you tell me what those 2 people do for a living?	Score if recognises role of 2 people correctly, e.g. doctor, nurse	
10	Can you remember the address I gave you?	Score for exact recall only	
		TOTAL	/10

Key point

Remember to examine the back of the patient either during the primary or secondary assessment

DOCUMENTATION

Always document the findings of the primary and secondary assessments. This record, along with subsequent entries into the patient's notes, should be dated, timed and signed. The patient's records must also contain a management plan, a list of investigations requested and the related results, as well as details of any treatment and its effect. This will not only provide an aide-mémoire but will also enable the patient's condition to be monitored and provide colleagues with an accurate account of a patient's hospital admission. This is assuming greater importance as patient care becomes more fragmented with increasing shift working and more patient movement between wards. Excellent written notes, and hence communication, are essential for good patient management.

DEFINITIVE CARE

Management plan

This should comprise a list of further investigations and treatment required for the particular patient. This is a dynamic plan that may change according to the clinical condition and test results. It needs to be reviewed regularly and updated.

Investigations

These will be dictated by the findings from the initial assessment and liaison with colleagues. Tests are not without risks; they should only be done if they directly benefit patient care.

Transport

All patients will be transferred sometime during their hospital stay. Irrespective of the transfer distance, appropriate numbers and grades of staff are required along with relevant equipment. Any period of transport is a period of potential patient instability. See Chapter 22 for further details.

Time Out 3.2

Your primary assessment of the confused 54-year-old man has revealed the following:

A	Assessment	Patent
	Resuscitation	$FiO_2 = 0{\cdot}85$
	Monitor	Pulse oximetry ($SpO_2 = 99\%$)
B	Assessment	Rate 30/min No accessory muscle use Symmetrical expansion No focal features
	Resuscitation	Not required, as yet
	Monitor	Respiration rate
C	Assessment	Pulse – radial 140 beats/min Apex – 160 beats/min – atrial fibrillation Blood pressure 90/60 Jugular venous pulse only visible when patient is lying flat Remainder of examination was normal
	Resuscitation	Following sedation cardioversion ($\times 3$) failed to convert patient to sinus rhythm therefore intravenous β blocker or amiodarone should be started
	Monitor	Radial pulse and apical rate Blood pressure Blood glucose, haemoglobin, urea and electrolytes
D	Assessment	PERLA Glasgow Coma Score 14/15: E = 4; V = 4; M = 6

a. What would be your next action?
b. What is the problem with this patient?
c. What is your management plan?

A WORD (OR TWO) OF COMMON SENSE

The structured approach is a safe comprehensive method of assessing any acutely ill patient. It should be regarded as the 'default method' in that it will prevent any further harm and cater for all medical problems. However, as most patients do not have an immediately life-threatening problem, a rapid primary assessment and/or an early warning score is all that is needed. Many patients do not require high

concentrations of inspired oxygen, intravenous access (×2) and a fluid challenge. Clinical judgement is still needed, combined with a modicum of common sense. If in doubt, revert to A.

SUMMARY

The acutely ill patient must be evaluated quickly and accurately. Thus, you must develop a structured method for assessment and treatment. In most acutely ill medical patients, the primary assessment is rapid and resuscitation is not required. Diagnosis is based on a well-'phrased' medical history obtained from the patient. However, if this is not possible then further information must be sought from medical records, relatives, general practitioners or colleagues from the emergency services.

Assessment and treatment are divided into two key assessment phases.

Primary assessment and resuscitation

The aim of the primary assessment is to identify and treat immediately life-threatening conditions.

- In most medical patients this can be done rapidly.
- Do not proceed to the secondary assessment until the patient's vital signs are normal or are moving towards normality.
- The most important assessment is the reassessment.

Assessment of:

A – Airway
B – Breathing
C – Circulation
D – Disability
E – Exposure

Resuscitation by:

- clearing and securing the airway and oxygenation
- ventilation
- intravenous access and shock therapy, including fluids, antibiotics, glucose, inotropes, dysrhythmia management
- exclude/correct hypoglycaemia
- consider anti-epileptic drugs, specific antidotes.

Monitoring to include oxygen saturation, respiration rate, pulse, blood pressure, cardiac rhythm, urinary output, pupillary response and Glasgow Coma Score; glucose and blood gases (if indicated).

Secondary assessment and emergency treatment

To gain corroborative evidence for primary diagnosis; to identify and treat new conditions.

Comprehensive physical examination including:

- general overview
- hands and radial pulse
- facial appearance
- neck – jugular venous pulse, carotid pulse, trachea
- chest – precordium and both lungs
- abdomen and genitalia
- locomotor system
- nervous system
- skin.

Reassessment
Now or if patient deteriorates at any stage.

Definitive care
- management plan
- investigations
- transport.

AIDE MEMOIRE

The flow diagrams depicted in Figs 3.1–3.6 are designed to aid your revision and provide an overview of the structured approach.

Note: *'BIG RED FLAGS' are findings that should alert you to an immediate life-threat and the need for urgent corrective action. The list is intended to be helpful, but not exhaustive.

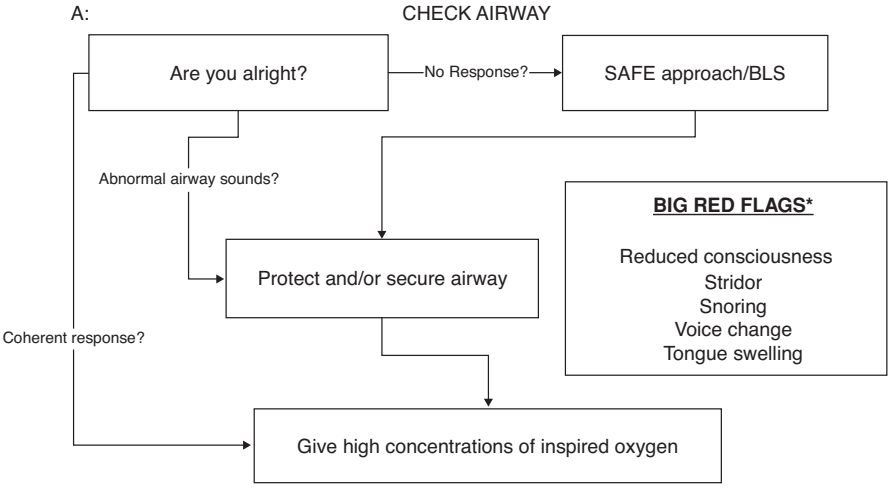

Fig. 3.1 Summary of airway assessment.

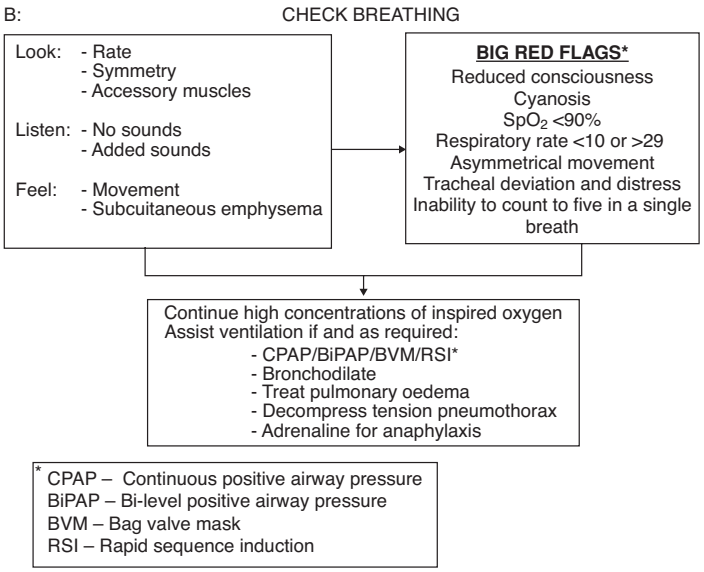

Fig. 3.2 Summary of breathing assessment.

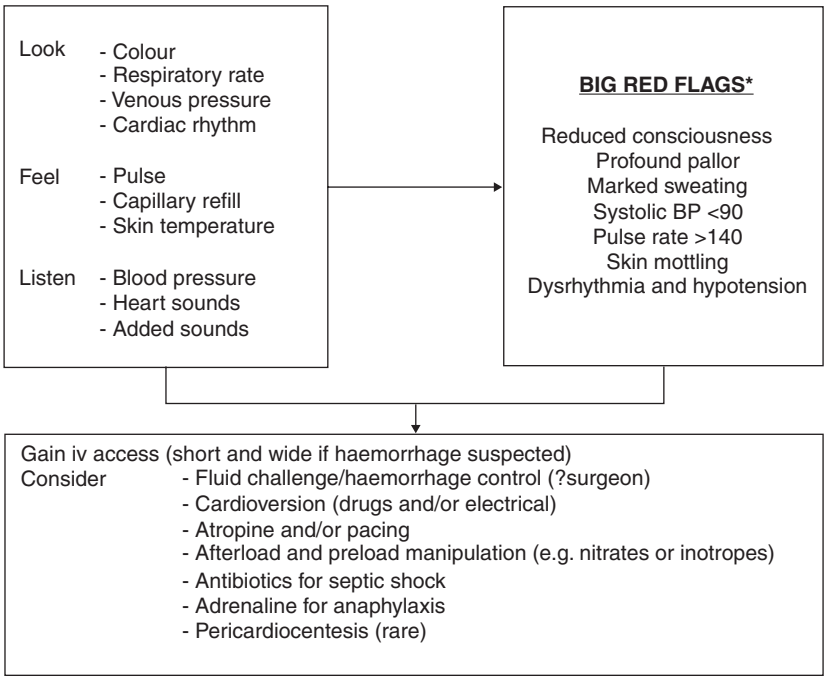

Fig. 3.3 Summary of circulation assessment.

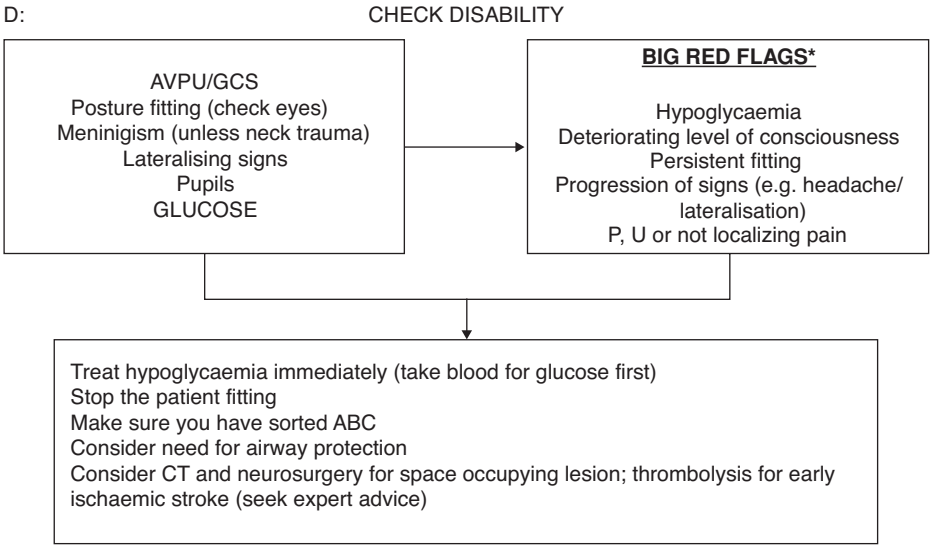

Fig. 3.4 Summary of disability assessment.

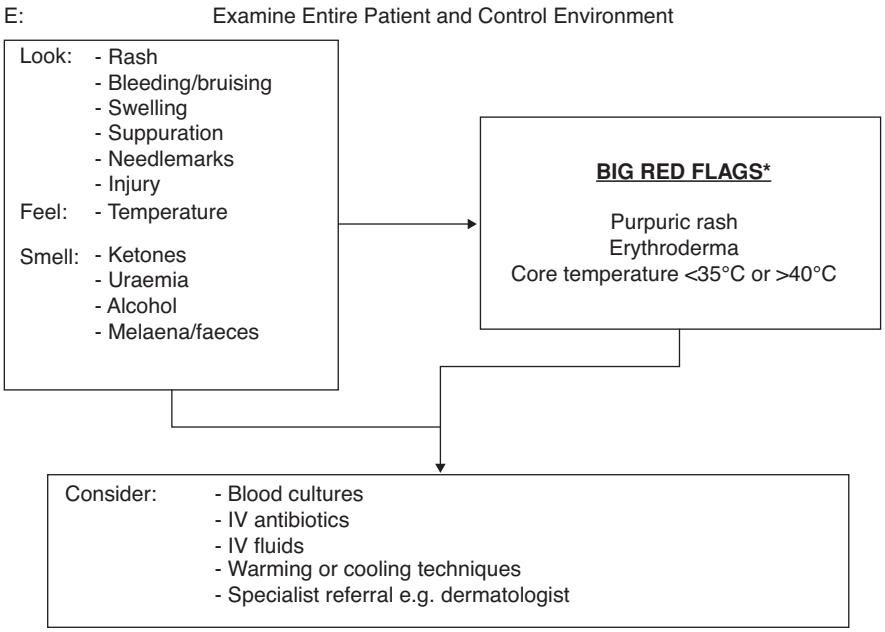

Fig. 3.5 Examine entire patient and control environment.

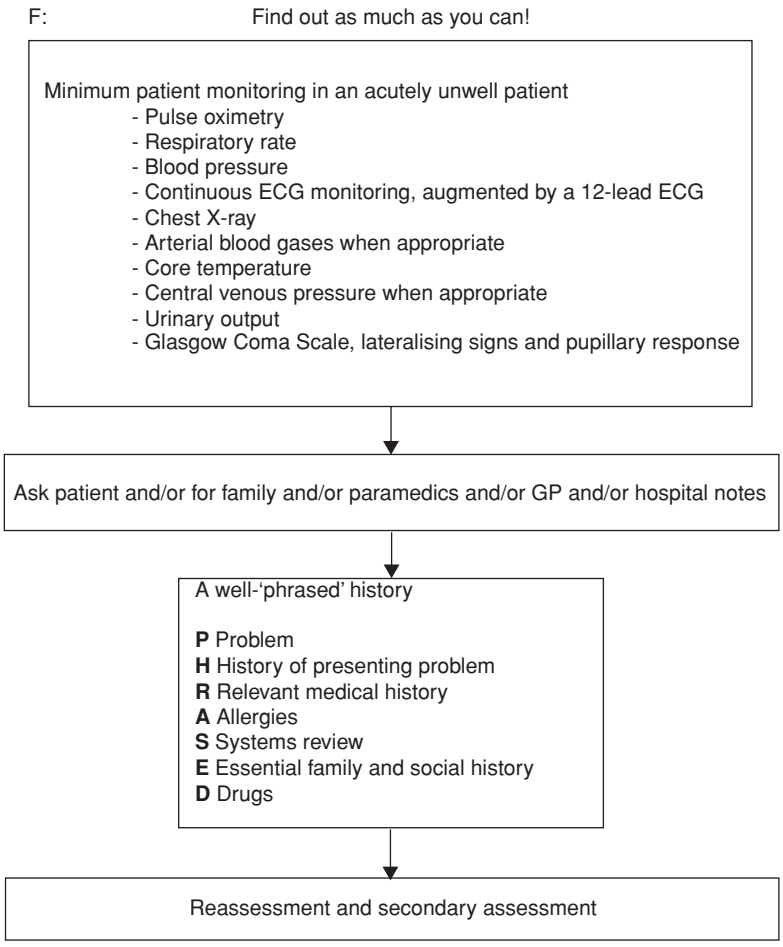

Fig. 3.6 Find out as much as you can.

Advanced Life Support Group

CHAPTER 4
Airway assessment

OBJECTIVES

After reading this chapter you will be able to:
- recognise the signs of airway obstruction
- understand how to use simple airway adjuncts
- describe advanced airway control and ventilation.

INTRODUCTION

- Airway problems are common in acute medical emergencies.
- Airway obstruction is the immediately life-threatening problem.

In the unconscious patient, it is essential to rapidly assess and control the airway; these simple manoeuvres can be life-saving. Therefore, in both basic and advanced life support, management of the airway is the first priority – the A of 'ABC'.

An obstructed airway can result in, or be caused by, a loss of consciousness. The obstruction can occur at many levels.

Immediately life-threatening causes of airway obstruction

Pharynx	**Tongue swelling**
	Swelling of the epiglottis or soft tissues
Larynx	**Oedema**
	Spasm of the vocal cords (laryngospasm)
	Foreign body
	Trauma
Subglottic	**Secretions or foreign body**
	Swelling
Bronchial	**Aspiration**
	Tension pneumothorax
	Foreign body

In the unconscious patient, the most common level of obstruction is the pharynx due to:
- a reduction in muscle tone, allowing the tongue to fall backwards (Fig. 4.1)
- abnormal muscle activity in the pharynx, larynx and neck. This explains why obstruction may still occur when the patient is prone.

This situation can be rectified and an airway provided by using manoeuvres described in this chapter.

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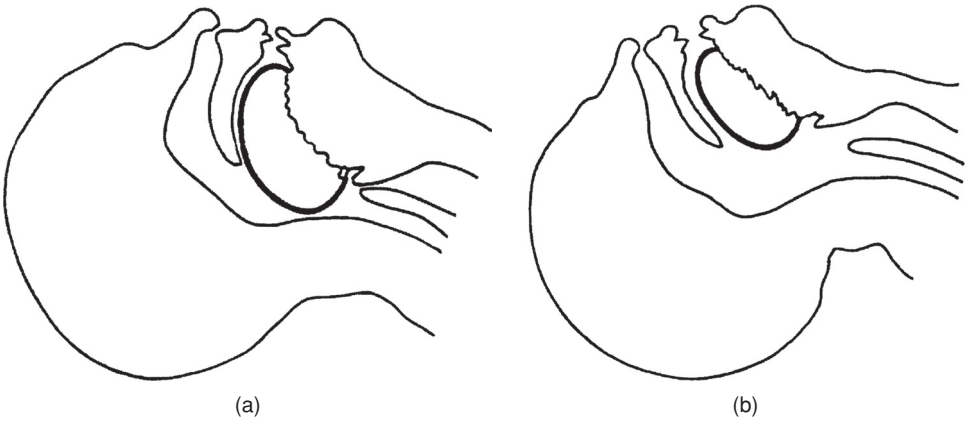

Fig. 4.1 (a) Obstruction of the airway by the tongue. (b) Head tilt, chin lift to clear the airway.

PRIMARY ASSESSMENT AND RESUSCITATION

This comprises:

Look at the chest for rate, depth and symmetry of movement. In complete airway obstruction, paradoxical movement of the chest and abdomen (see-sawing) will occur as a result of the increased respiratory effort. In addition, there may be use of accessory muscles; intercostal and supraclavicular recession may be visible and a tracheal tug may be palpable. Look in the mouth for blood, gastric contents, frothy sputum (pulmonary oedema) and foreign bodies.

Listen for breath sounds. Partial obstruction may be accompanied by the following:

- Inspiratory noises (stridor) commonly indicate upper airway obstruction.
- Expiratory noises, particularly wheezing, usually occur in obstruction of the lower airways as they collapse during expiration.
- 'Crowing' signifies laryngeal spasm.
- 'Gurgling' indicates the presence of liquid or semisolid material.
- 'Snoring' indicates that the pharynx is still partially occluded by the tongue.

Feel for:

- expired air against the side of your cheek
- chest movement, comparing one side with the other
- the position and 'tugging' of the trachea
- any subcutaneous emphysema.

Remove broken and very loose dentures, but leave well-fitting ones as they help to maintain the contour of the mouth and make using a bag–mask system easier (see later).

The possibility of an injury to the cervical spine must always be considered. However, medical patients are more likely to die from hypoxaemia than be rendered quadriplegic as a consequence of carefully conducted airway opening manoeuvres.

Airway adjuncts

These are often helpful to improve and maintain airway patency either during resuscitation or in a spontaneously breathing patient. Both oropharyngeal and nasopharyngeal airways are designed to overcome backward tongue displacement in the unconscious patient. In both cases, either the head tilt or jaw thrust techniques usually need to be maintained.

Oropharyngeal (Guedel) airways

These are curved, rigid, plastic tubes, flanged at the oral end and flattened in cross section to fit between the tongue and the hard palate. They are available in a variety of sizes which are suitable for all patients, from newborn babies to large adults. An estimate of the size required can be obtained by comparing the airway with the vertical distance from the centre of the incisors to the angle of the jaw (see Chapter 31 for further details).

The oropharyngeal airway is inserted either upside down and rotated through 180° or under direct vision with the aid of a tongue depressor or laryngoscope (see Chapter 31 for further details).

Incorrect insertion can push the tongue further back into the pharynx and produce airway obstruction, trauma, bleeding and force unrecognised foreign bodies further into the larynx. An oral airway may irritate the pharynx and larynx and cause vomiting and laryngospasm, respectively, especially in patients who are not deeply unconscious.

Nasopharyngeal airways

This airway is made from malleable plastic that is bevelled at one end and flanged at the other, and is round in cross section to aid insertion through the nose. Nasopharyngeal airways are sized according to the diameter of the patient's nares or the size of their little finger.

Nasopharyngeal airways are often better tolerated than oropharyngeal airways. They may be life-saving in a patient whose mouth cannot be opened, e.g. with trismus or in the presence of maxillary injuries. They should, however, be used with extreme caution in patients with a suspected base of skull fracture (very rare in the acutely ill medical patient).

Even with careful insertion, bleeding can occur from tissues in the nasopharynx. If the airway is too long, both vomiting and laryngospasm can be induced in patients who are not deeply unconscious.

A further problem with both of these types of airway is that air may be directed into the oesophagus during assisted ventilation. This results in inefficient ventilation of the lungs and gastric dilatation, which splints the diaphragm, making ventilation difficult and increasing the risk of regurgitation. This commonly occurs when high inflation pressures are used to try and ventilate a patient. In these circumstances, carefully check that ventilation is adequate and gastric distension is minimised.

If adequate spontaneous ventilation follows these airway opening manoeuvres, place the patient in an appropriate recovery position – provided there are no contraindications. This will reduce the risk of further obstruction.

Ventilatory support

If spontaneous ventilation is inadequate or absent, start artificial ventilation.

Exhaled air resuscitation

If no equipment is available, expired air ventilation will provide 16% oxygen. This can be made more pleasant, and the risks of cross-infection reduced, by the use of simple adjuncts to avoid direct person-to-person contact; an example of this is the Laerdal pocket mask. This device has a unidirectional valve to allow the rescuer's expired air to pass to the patient while the patient's expired air is directed away from the rescuer. The masks are transparent to allow detection of vomit or blood; one version has an additional attachment for supplemental oxygen.

Oxygen

Oxygen should be given to all patients during resuscitation, with the aim of increasing the inspired concentration to greater than 95%. However, this concentration will depend on the system used and the flow available. In spontaneously breathing patients, a Venturi mask will deliver a fixed concentration (24–60%), depending on the mask chosen. A standard concentration mask will deliver up to 60%, provided the flow of oxygen is high enough (12–15 l/min). Some patients are more tolerant of nasal cannulae, but these only raise the inspired concentration to approximately 44%. The most effective system is a mask with a non-rebreathing reservoir in which the inspired concentration can be raised to 85% with an oxygen flow of 12–15 l/min. This is the most desirable method in spontaneously breathing patients.

Advanced airway control and ventilation

Airway control

In the deeply unconscious patient, airway control is best achieved by tracheal intubation. However, the technique requires a greater degree of skill and more equipment than the methods already described.

Tracheal intubation may be indicated for several reasons:

- to protect the airway against contamination from regurgitated stomach contents or blood
- to protect the airway and ensure ventilation when an investigation has to be done, e.g. CT scan
- when an airway cannot be secured by another route
- to allow safe transport of a patient
- to facilitate ventilation without leaks, even when airway resistance is high (e.g. in pulmonary oedema and bronchospasm)
- to achieve a safe environment for the patients (e.g. in drug overdosage)
- to facilitate mechanical ventilation for other reasons (e.g. exhaustion).

Tracheal intubation

This is the preferred method for airway control during cardiopulmonary resuscitation, for the reasons already outlined. Considerable training and practice are required to acquire and maintain the skill of intubation. Repeated attempts by the inexperienced are likely to be unsuccessful and traumatic, compromise oxygenation and delay resuscitation. Orotracheal intubation is the preferred route. Nasotracheal is rarely required and much more difficult than orotracheal intubation.

The technique of orotracheal intubation is described in Chapter 31. Nevertheless this is not intended as a substitute for practice using a manikin or, better still, an anaesthetised patient under the direction of a skilled anaesthetist.

Tracheal intubation may be difficult to perform during cardiac arrest. The patient may be in an awkward position on the floor, equipment may be unfamiliar, assistance limited, cardiopulmonary resuscitation obstructive and vomit copious. In these circumstances, it is all too easy to persist with the ‘almost there’ attitude. This must be strongly resisted. If intubation is not successfully accomplished in approximately 30–40 s (about the time one can breath-hold during the attempt), it should be abandoned. Ventilation with 12–15 l/min (95%) oxygen using a bag–valve–mask should be recommenced before, and in between, any further attempts at intubation. Ideally, the tube should be seen to pass through the cords and then the circuit should be attached to a carbon dioxide monitor (end tidal or colorimetric) to ensure correct placement.

In certain circumstances such as acute epiglottitis, laryngoscopy and attempted intubation are contraindicated because they could lead to deterioration in the patient's condition. Specialist skills will be required, including the use of anaesthetic drugs or fibre-optic laryngoscopy.

Key point

It is not appropriate to learn and practice endotracheal intubation during resuscitation

Currently endotracheal intubation is the optimum method of managing the airway in an unconscious patient. For most people, acquiring this skill is time-consuming; continuous training is unavailable; and skill retention is poor. The laryngeal mask airway is an acceptable alternative.

The laryngeal mask airway

This comprises a 'mask' with an inflatable cuff around its edge that sits over the laryngeal opening. Attached to the mask is a tube that protrudes from the mouth and through which the patient either breathes or is ventilated (Fig. 4.2). This was originally designed for spontaneously breathing anaesthetised patients. However, supported ventilation is possible, provided that inflation pressures are not excessive. The main advantage of the laryngeal mask airway (LMA) is that it is inserted blindly, the technique may be mastered more easily than tracheal intubation and skill retention in the occasional practitioner is better. However, if either the seal around the larynx is poor or the mask is malpositioned, ventilation will be reduced and gastric inflation may occur. Furthermore, there is no guarantee against aspiration. Whenever possible, insertion of an LMA must be preceded by a period of preoxygenation. Any attempt at insertion must be limited to 30–40 s, after which ventilation with 12–15 l/min oxygen using a bag–valve–mask should be recommenced before further attempts.

The LMA can be used as a conduit to allow the insertion of a tracheal tube to secure the airway in cases of difficult tracheal intubation. This technique is described in Chapter 31.

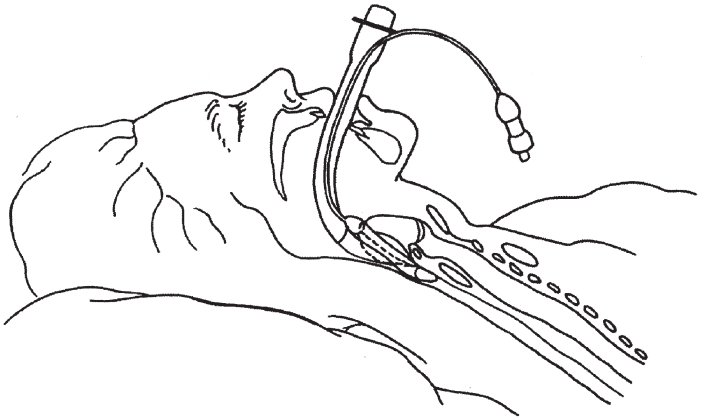

Fig. 4.2 Laryngeal mask *in situ*.

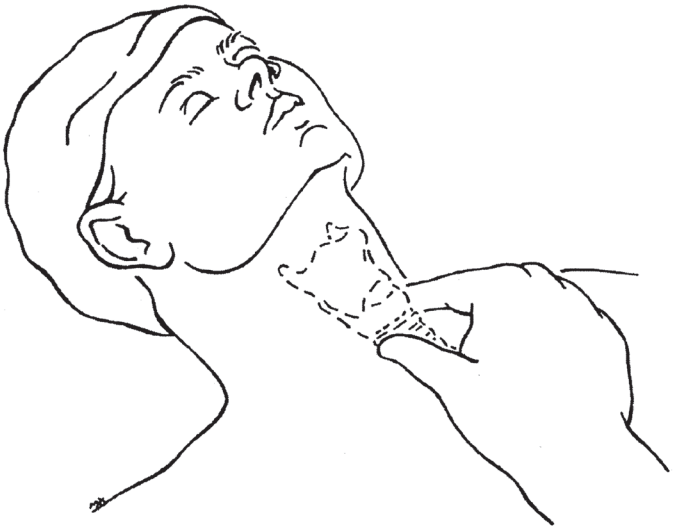

Fig. 4.3 Technique for cricoid pressure.

Cricoid pressure

This is a manoeuvre used by anaesthetists to prevent regurgitation and aspiration of gastric contents during induction of anaesthesia – often in acutely ill patients and/or those with a full stomach.

The cricoid cartilage forms a complete ring immediately below the thyroid cartilage. Pressure is applied on the cricoid by an assistant forcing the ring backwards, occluding the oesophagus against the body of the sixth cervical vertebra (Fig. 4.3), thus preventing the flow of any gastric contents beyond this point. This manoeuvre is maintained until:

- the tracheal tube is inserted into the larynx
- the cuff is inflated
- the person intubating indicates that pressure can be released.

Incorrectly applied pressure will make intubation more difficult. If the patient vomits, cricoid pressure must be released immediately because of the slight risk of oesophageal rupture. In such circumstances the patient needs to be turned onto their side, the trolley tipped head down and the airway cleared with suction.

In contrast to cricoid pressure, pressure on the thyroid cartilage by a trained assistant can facilitate endotracheal intubation. This is known as the BURP (Backward Upward Right Pressure) technique.

Ventilation

The ultimate aim is to achieve an inspired oxygen concentration of greater than 95%. The most common device used is the self-inflating bag with a one-way valve that can be connected to either a facemask or a tracheal tube (Fig. 4.4).

Squeezing the bag delivers its contents to the patient via the one-way valve. On release the bag re-inflates, refilling via the inlet valve at the opposite end. At the same time, the one-way valve diverts the expired gas from the patient to the atmosphere. Using the bag-valve alone (attached to mask or tracheal tube), the patient is ventilated with 21% oxygen as the bag refills with ambient air. This can (and should) be increased during resuscitation to around 50% by connecting an oxygen supply at 12–15 l/min directly to the bag adjacent to the air intake. This can be further increased to 95% by attaching a reservoir bag.

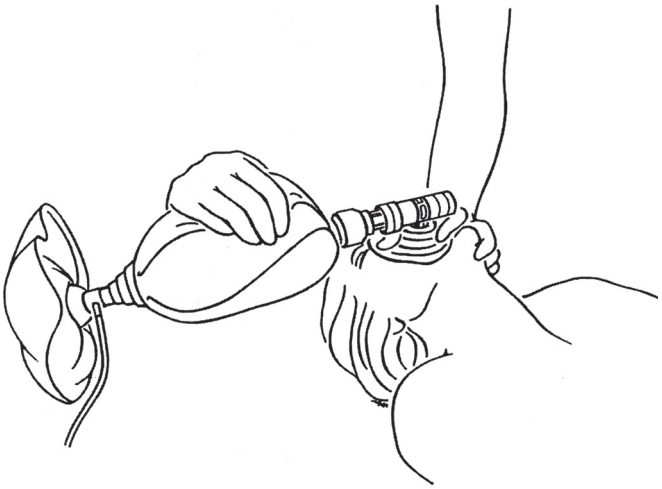

Fig. 4.4 Self-inflating bag–mask and reservoir.

Although the self-inflating bag–valve–mask will allow ventilation with higher concentrations of oxygen, it is associated with several problems.

- It requires considerable skill for one person to maintain a gas-tight seal between the mask and the patient's face, whilst at the same time lifting the jaw with one hand and squeezing the bag with the other.
- Any air leak will result in hypoventilation, no matter how energetically the bag is compressed.
- Excessive compression of the bag when attached to a facemask results in gas passing into the stomach. This further reduces effective ventilation and increases the risk of regurgitation and aspiration.
- The valve mechanism may 'stick' if it becomes blocked with secretions, vomit or heavy moisture contamination.

As a result of some of these problems, a two-person technique is recommended during ventilation of a patient with a bag–valve–mask. One person holds the facemask in place using both hands and an assistant squeezes the bag. Thus a better seal is achieved, the jaw thrust manoeuvre is easier to maintain and the patient can be ventilated more easily.

Clearly these problems can be overcome by tracheal intubation, which eliminates leaks and ensures that oxygen is delivered directly and only into the lungs (always assuming the tube is in the trachea!).

Patients requiring prolonged ventilation can be placed on a mechanical ventilator. When using these devices, the most important feature to remember is that they are good servants, but poor masters. Ventilators will only do what they are set to do, and will not automatically compensate for changes in the patient's condition during resuscitation. Therefore, it is imperative that they are set correctly and checked regularly.

A variety of small portable ventilators are used during resuscitation and are generally gas powered. If an oxygen cylinder is used as both the supply of respiratory gas for the patient and the power for the ventilator, its contents will be used more rapidly. This is particularly important if patients are being transported over long distances because adequate oxygen supplies must be taken.

Gas-powered portable ventilators are classified as time cycled and often have a fixed inspiratory/expiratory ratio. They provide a constant flow of gas to the patient during inspiration; expiration occurs passively to the atmosphere. The volume delivered depends on the inspiratory time (i.e. longer times and larger

breaths) with the pressure in the airway rising during inspiration. As a safety feature, these devices can often be 'pressure limited' by a relief valve opening to protect the lungs against excessive pressures (barotrauma).

A ventilator should initially be set to deliver 7–10 ml/kg tidal volume at a rate of 12 breaths/min with 100% oxygen. Some ventilators have coordinated markings on the controls for rapid initial setting for different-sized patients. The correct setting will ultimately be determined by analysis of arterial blood gases.

Key point

Care should be taken when using ventilators which have relief valves fixed to open at relatively low pressures. These may be exceeded during cardiopulmonary resuscitation if a chest compression coincides with a breath from the ventilator, resulting in inadequate ventilation. Furthermore, by the same mechanism, ventilators with adjustable pressure relief valves, if set too high, may subject patients to excessively high pressures. These risks can be reduced by decreasing the rate of ventilation (breaths/min) to allow coordination of breaths and compressions.

If there is any doubt about the performance of the ventilator, the safest option is to temporarily disconnect it and continue by using a self-inflating bag–valve assembly with oxygen and reservoir, until skilled help is available.

Suction (endotracheal)

Once the trachea has been intubated, suction is performed to remove secretions, vomit or blood. To avoid making the patient hypoxaemic and bradycardic, this must be done carefully in the following way:

- Wear gloves.
- Ventilate the patient with 100% oxygen.
- Introduce a sterile catheter through the airway into the trachea without suction applied.
- The diameter of the catheter should be less than half that of the tracheal tube.
- Intermittent suction and withdraw the catheter using a rotating motion over 10–15 s.
- Irrespective of the amount of blood/mucus removed, do not reintroduce the catheter without a further period of oxygenation.
- Loosen tenacious secretions by instilling 10 ml of sterile saline, followed by five vigorous manual ventilations. Suction as described earlier.

Key point

Suction must not be applied directly to the tracheal tube as this will result in life-threatening hypoxaemia and dysrhythmias

Time Out 4.1

Take a break and list the clinical features of airway obstruction

SUMMARY

Airway control and ventilation are essential prerequisites for successful management of the acutely ill patient. Airway obstruction should be recognised and managed immediately. Endotracheal intubation remains the best method of securing and controlling the airway, but requires additional equipment, skill and practice.

Advanced Life Support Group

CHAPTER 5
Breathing assessment

OBJECTIVES

After reading this chapter you will be able to:
- understand the physiology of oxygen delivery
- describe a structured approach to breathing assessment
- identify immediately life-threatening causes of breathlessness
- describe the immediate management of these patients.

INTRODUCTION

The acutely breathless patient is a common medical emergency that is distressing for both the patient and the clinician. Often the effort required for breathing makes it virtually impossible for the patient to provide any form of medical history and questioning may only make the situation worse. Information should be sought from any available source. The clinician's skills will help to determine the underlying cause and dictate appropriate management.

Key point

Breathlessness can result from a problem in airway (A), breathing (B), circulation (C) and disability (D)

Immediately life-threatening causes of breathlessness

Airway
- **Obstruction (see full list in box 1 in Chapter 4)**

Breathing
- **Acute severe asthma**
- **Acute exacerbation of chronic obstructive pulmonary disease (COPD)**
- **Pulmonary oedema**
- **Tension pneumothorax**
- **Critical oxygen desaturation**

Circulation
- **Shock**

Disruption of oxygen delivery is a fundamental problem in these conditions. Therefore it is important to understand the mechanisms that maintain oxygen delivery in health.

Acute Medical Emergencies: The Practical Approach, Second Edition
Edited by Advanced Life Support Group
 ISBN: 978-0-727-91854-3

RELEVANT PHYSIOLOGY

Oxygen delivery

The normal respiratory rate is 14–20 breaths/min. With each breath, 500 ml of air (7–10 l/min) is inhaled and exhaled. This air mixes with alveolar gas and, by diffusion, oxygen enters the pulmonary circulation to combine mainly with haemoglobin in the red cells. The erythrocyte-bound oxygen is transported via the systemic circulation to the tissues, where it is taken up and used by the cells.

The delivery of oxygen (DO_2) to the tissues depends on:

- concentration of oxygen reaching the alveoli
- pulmonary perfusion
- adequacy of pulmonary gas exchange
- capacity of blood to carry oxygen
- blood flow to the tissues.

Concentration of oxygen reaching the alveoli

The two most important factors determining the amount of oxygen reaching the alveoli are:

- the fraction of inspired oxygen (FiO_2)
- ventilation.

Providing supplementary oxygen to a person increases the number of oxygen molecules getting to the alveoli. A variety of oxygen masks can be used to increase the **fraction of inspired oxygen**.

The effectiveness of this procedure depends on the lungs' ability to draw the inspired gas into the alveoli. The mechanism for transporting inspired air to, and expired gas from, the alveoli is called **ventilation** (V). Ventilation is subject to several regulatory processes which are summarised in the box below.

Key components in ventilation

Brain stem	**Central respiratory chemoreceptors in medulla oblongata**
Peripheral chemoreceptors	**Carotid and aortic bodies for CO_2, O_2 and H^+**
Vagus, phrenic and intercostal nerves	**Ventilatory drive**
Respiratory muscles	**Intercostal muscles and diaphragm**
Mechanics and compliance	**Airways, lungs and chest wall**

The normal ventilatory volumes and rates are summarised in Figs 5.1 and 5.2.

The **normal resting respiratory rate** is 15 (range 14–20) breaths/min. The amount of air inspired per breath is called the **tidal volume** and is equivalent to 7–8 ml/kg body weight (or 500 ml for the 70 kg patient). Therefore the amount of air inspired each minute, the **minute volume**, can be calculated by multiplying the **respiratory rate** by the **tidal volume** (15 × 500 ml) to produce a value of 7.5 l/min.

The tidal volume (500 ml) is distributed throughout the respiratory system but only 350 ml (70%) mixes with alveolar air. The remainder (150 ml) occupies the airways that are not involved in gas transfer. This volume is referred to as the **anatomical dead space**. In addition, there are certain areas within the lungs which are not involved with gas transfer because they are ventilated but not

perfused. The volume produced by the combination of these areas and the anatomical dead space is called the **total or physiological dead space**. In healthy individuals, these two dead spaces are virtually identical because ventilation and perfusion are well matched.

It follows that the amount of air reaching the alveoli, i.e. **alveolar ventilation**, can be calculated from:

respiratory rate × (tidal volume − anatomical dead space)

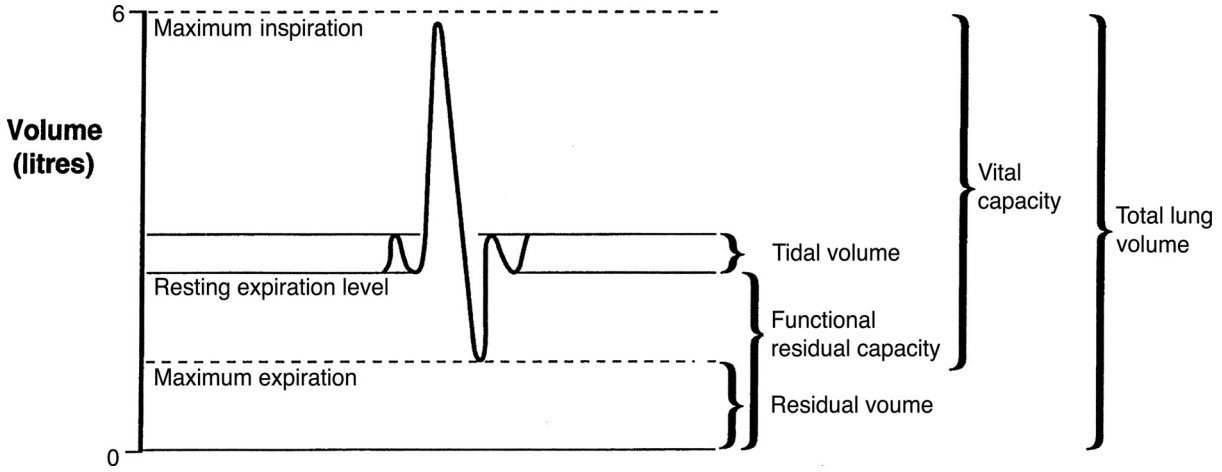

Fig. 5.1 Normal ventilatory volumes.

Using data from Fig. 5.2, this corresponds to 15 × (500 – 150) = 5250 ml/min.

Rapid shallow respiration causes a marked reduction in alveolar ventilation because the anatomical dead space is fixed, i.e. 30 × (200 – 150) = 1500. This is demonstrated further in Table 5.1, where the effect of different respiratory rates can be seen.

Finally, it is important to be aware of a crucial volume known as the **functional residual capacity** (FRC) (2.5–3.01). This is the amount of air remaining in the lungs at the end of a normal expiration. As 350 ml of each tidal volume is

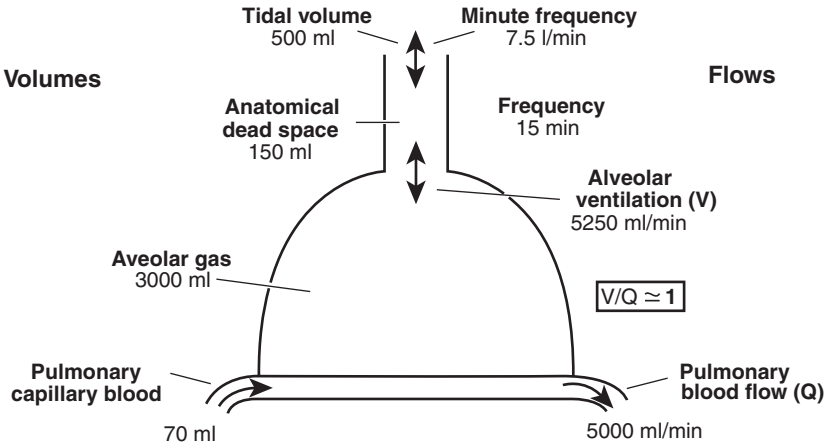

Fig. 5.2 Normal volumes and flows.

available for gas transfer, fresh alveolar air will only replace 12–14% of the functional residual capacity. The FRC therefore acts as a large reservoir, preventing sudden changes in blood oxygen and carbon dioxide concentration.

Table 5.1 The effect of respiratory rate on alveolar ventilation

Respiratory rate (breaths/min)	10	20	30
Tidal volume (ml)	600	400	200
Anatomical dead space (ml)	150	150	150
Alveolar ventilation (ml/min)	4500	5000	1500

Pulmonary perfusion

At rest, the cardiac output from the right ventricle is delivered to the pulmonary circulation at approximately 5.5 l/min. As alveolar ventilation is 5.25 l/min, the ventilation/pulmonary perfusion ratio is equal to 0.95 (5.25/5.5).

The pressures in the pulmonary vascular bed are low (around 20/9 mm Hg) and therefore affected by posture. As a result, there are differences in blood flow to different lung regions, contributing to the physiological dead space. In the upright position, basal alveoli are well perfused but relatively poorly ventilated. Consequently, in these areas, venous blood comes into contact with alveoli filled with low concentrations of oxygen and so less oxygen can be taken up. This effect is minimised in healthy individuals by pulmonary vasoconstriction which diverts blood to areas of the lungs that have better ventilation. In the apical regions of the lungs, there is ventilation but relatively poor perfusion.

There are also direct links between the right and left sides of the heart (mainly from blood supplying lung parenchymal tissue). These normally allow 2% of the right ventricle's output to bypass the lungs completely and are collectively known as the **physiological shunt**. As the blood in this shunt has had no contact with the alveoli, its oxygen and carbon dioxide concentrations will remain the same as those found in the right ventricle.

Pulmonary gas exchange

Oxygen continuously diffuses out of the alveolar gas into the pulmonary capillaries, with carbon dioxide going in the opposite direction. The rate of diffusion is governed by the following factors:

- partial pressure gradient of the gas
- solubility of the gas
- alveolar surface area
- alveolar capillary wall thickness.

The lungs are ideally suited for diffusion as they have both a large alveolar surface area (approximately 50 m^2) and a thin alveolar capillary wall. It is easy to understand why gas exchange would be compromised by a reduction in the former (e.g. pneumothorax) or an increase in the latter (e.g. interstitial pulmonary oedema).

Gases move passively down gradients from areas of high to low partial pressure. The partial pressure of oxygen in the alveoli (PaO_2) is approximately 13.4 kPa (100 mm Hg), whereas that in the pulmonary artery is 5.3 kPa (40 mm Hg). In contrast, the gradient for carbon dioxide is only small, with the alveolar partial pressure being 5.3 kPa (40 mm Hg), compared with 6.0 kPa (46 mm Hg) in the

pulmonary artery. However, carbon dioxide passes through biological membranes 20 times more easily than oxygen. In health, the net effect is that the time taken for exchange of oxygen and carbon dioxide is virtually identical.

Although alveolar ventilation, diffusion and pulmonary perfusion will all affect the alveolar PO_2 (PaO_2) and hence the arterial PO_2 (PaO_2), the most important factor in determining the PaO_2 is the ratio of ventilation to perfusion.

Ventilation/perfusion ratio

To understand this concept, it is helpful to consider the normal situation and divide each lung into three functional areas: apical (a), middle (b) and basal (c) (Fig. 5.3).

The apical segment is well ventilated, but relatively poorly perfused. Therefore, not enough blood is available to accept all the alveolar oxygen. However, the red cells that are available are fully laden (saturated) with oxygen with the extra oxygen dissolved in plasma.

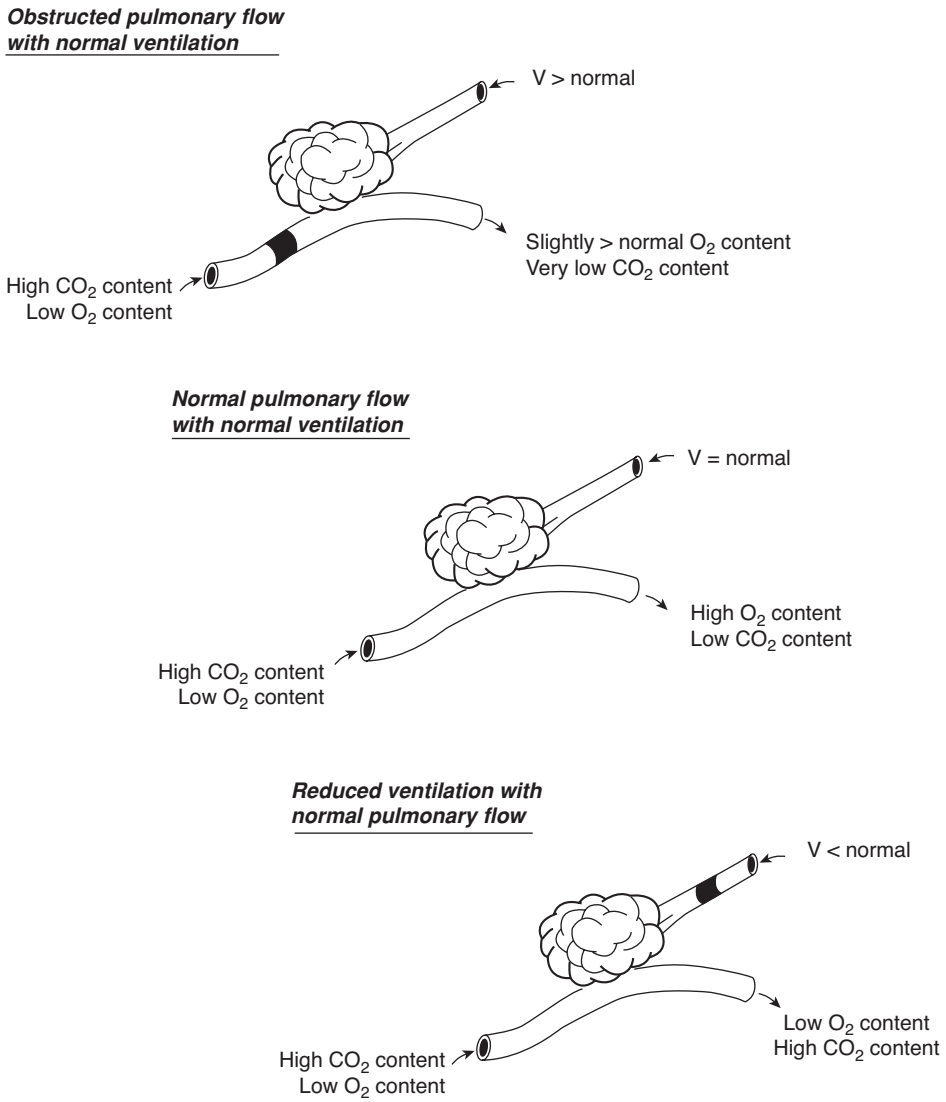

Fig. 5.3 Three different ventilation (V)/perfusion (Q) ratios: (a) normal ventilation with reduced perfusion; (b) normal ventilation with normal perfusion; (c) reduced ventilation with normal perfusion.

Key point

In the apical segment, there is more ventilation than perfusion, i.e. the V/Q ratio > 1

The middle segment has ventilation and perfusion perfectly matched. Alveolar oxygen diffuses into – and is correctly balanced by – the pulmonary capillary blood, ensuring that the red cells are fully saturated. There is relatively little oxygen left to dissolve in plasma.

Key point

In the middle segment, ventilation and perfusion are matched, i.e. the V/Q ratio = 1

The basal segment alveoli are well perfused, but poorly ventilated.

Key point

In the basal segment, ventilation is reduced when compared with perfusion, i.e. the V/Q ratio < 1

Remember that the overall ratio of ventilation to perfusion is nearly one (0.95).

Key point

An area of lung with a high V/Q ratio cannot offset the fall in oxygen content produced by an area of lung with a low V/Q ratio

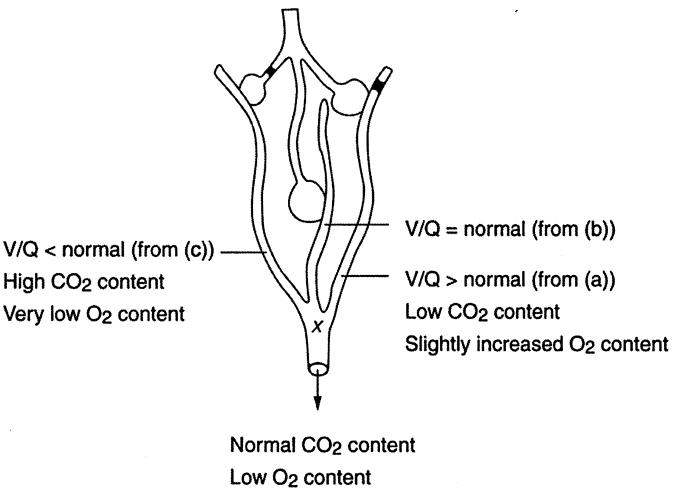

Fig. 5.4 Mixed blood returning from three sites at point X.
To understand this point we need to consider how oxygen is carried in the blood.

Oxygen content of arterial blood

The oxygen content of haemoglobin (Hb) going to tissues depends on the:

- saturation of haemoglobin with oxygen
- haemoglobin concentration
- oxygen-carrying capacity
- oxygen dissolved in plasma.

Haemoglobin is a protein comprising four subunits, each of which contains a haem molecule attached to a polypeptide chain. The haem molecule contains iron which reversibly binds oxygen; hence it is oxygenated but **not** oxidised (it carries oxygen, but does not react with it, i.e. it is not rust!). Each haemoglobin molecule can carry up to four oxygen molecules. Blood has a haemoglobin concentration of approximately 15 g/100 ml. Under normal conditions, each gram of haemoglobin can carry 1.34 ml of oxygen if it is fully saturated. Therefore, the **oxygen carrying capacity** of blood is:

$$\text{Hb} \times 1.34 \times 1$$

$$15 \times 1.34 \times 1 = 20.1 \text{ ml } O_2/100 \text{ ml of blood}$$

(A value of 1 indicates that Hb is fully saturated.)

This is approximately 60 times greater than the amount of oxygen dissolved in plasma.

Key point

Nearly all of the oxygen carried in the blood is taken up by haemoglobin, with only a small amount dissolved in the plasma

The relationship between the PaO_2 and oxygen uptake by haemoglobin is not linear, because the addition of each O_2 molecule facilitates the uptake of the next O_2 molecule. This produces a sigmoid-shaped oxygen dissociation curve (Fig. 5.5). Furthermore, because haemoglobin is 97.5% saturated at a PaO_2 of 13.4 kPa (100 mm Hg) (i.e. that found in the normal healthy state), increasing the PaO_2 further has little effect on oxygen transport.

The affinity of haemoglobin for oxygen at a particular PO_2 (commonly known as the O_2–Hb association) is also affected by other factors. A decreased affinity

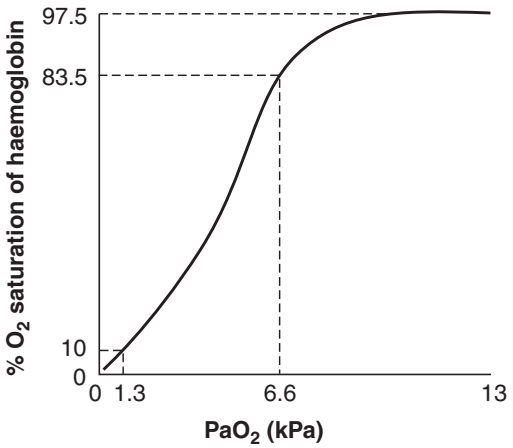

Fig. 5.5 The haemoglobin–oxygen dissociation curve.

means that oxygen is more readily released. Thus the oxygen dissociation curve is shifted to the right.

Factors that decrease the affinity of haemoglobin for oxygen:

- hydrogen ion concentration (i.e. a fall in pH)
- $PaCO_2$
- concentration of red cell 2,3-diphosphoglycerate (2, 3-DPG)
- temperature.

(The opposite of these factors increases the affinity and moves the curve to the left. These will be discussed later.)

The normal haemoglobin concentration is usually just above the point at which the oxygen transportation is optimal. Consequently, a slight fall in haemoglobin concentration will actually increase oxygen transportation by decreasing blood viscosity.

In addition to the oxygen combined with haemoglobin, there is a smaller amount dissolved in plasma. This amount is directly proportional to the PaO_2 and is approximately 0.23 ml/l blood/kPa PaO_2 (0.003 ml/100 ml blood/mm Hg of PaO_2).

It follows from the description above that the total content of oxygen in blood is equal to the oxygen associated with haemoglobin (per litre) and that dissolved in plasma.

$$\text{Oxygen blood content} = (\text{Hb} \times 1.34 \times \text{saturation}) + (0.23 \times PaO_2)\ \text{ml/l}$$

For example, in arterial blood with a haemoglobin content of 150 g/l and a PaO_2 of 15 kPa the oxygen content would be:

$$\text{Oxygen blood concentration} = (150 \times 1.34 \times 0.975) + (0.23 \times 15) = 199\ \text{ml/l}$$

Alternatively, in venous blood with a haemoglobin content of 150 g/l and a PaO_2 of 5 kPa the oxygen content would be:

$$\text{Oxygen blood concentration} = (150 \times 1.34 \times 0.75) + (0.23 \times 5) = 151\ \text{ml/l}$$

PRIMARY ASSESSMENT AND RESUSCITATION

Airway

This has been described in detail in Chapter 4. The following summary contains the relevant facts relating to the breathless patient.

Assessment

Most breathless patients will have a patent airway. The number of words said with each breath is a useful indicator of illness severity and the effects of treatment. If the patient can count to 10 in one breath, then the underlying condition is unlikely to warrant immediate intervention. Occasionally, however, the patient will be severely distressed with stridor, possibly coughing and making enormous but ineffectual respiratory efforts. Stridor is a sinister sign and should be regarded as indicating impending airway obstruction.

Resuscitation

If established or impending airway obstruction is suspected, immediate review by an anaesthetically competent person is required. If foreign body inhalation is suspected, then a Heimlich (or modified Heimlich in pregnant and obese patients) manoeuvre should be attempted. If the patient has a respiratory arrest, perform laryngoscopy and remove any identifiable foreign body. If this is unsuccessful,

proceed to needle cricothyroidotomy and jet insufflation, followed by surgical referral for a surgical cricothyroidotomy.

Breathing

Assessment

This is summarised in the box.

Summary of breathing assessment

Look	**Colour, sweating**
	Posture
	Respiratory rate, effort
	Symmetry
Feel	**Tracheal position**
	Tracheal tug
	Chest expansion
Percuss	**Anterior and posterior aspects of both lungs in**
Listen	**the upper, middle and lower zones**

The immediately life-threatening conditions need to be identified and treated during the primary assessment of breathing.

Specific clinical features

By the time 'B' is assessed, all critically ill patients should be receiving high concentrations of inspired oxygen (FiO_2 = 0.85 at 15 l/min) (Fig. 5.6). Do not be concerned about patients who retain CO_2. Provided that FiO_2 equals 0.85, a rise in $PaCO_2$ will not increase mortality – but untreated hypoxaemia will!

After the primary assessment has been completed, the FiO_2 can be titrated according to either the arterial blood gas results or the pulse oximeter reading.

A hyperinflated chest is indicative of asthma or COPD. **In an acute exacerbation** of these conditions, the trachea moves downwards during inspiration. This is referred to as tracheal tug and implies airway obstruction or increased respiratory effort. In addition, the internal jugular pressure may be elevated and accessory muscle use will be prominent, as will intercostal recession over the lower part of the chest during inspiration. Patients often adopt a seated or standing posture to aid breathing.

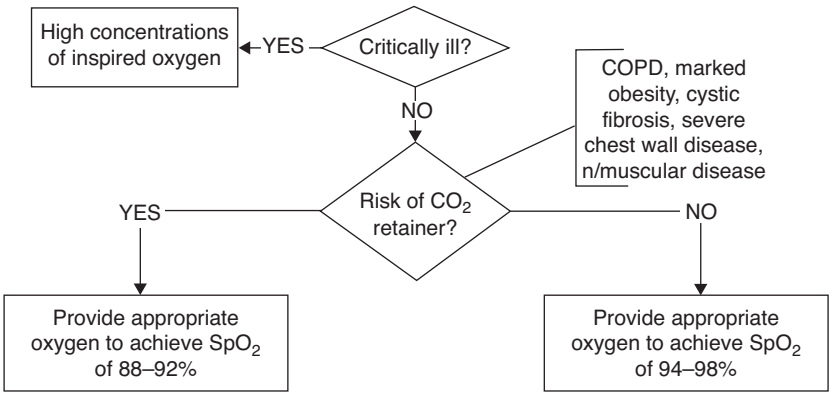

Fig. 5.6 Oxygen requirements.

Bronchospasm is common to both asthma and COPD. In acute asthma, the inspiratory phase is snatched and expiration is prolonged. With COPD, however, the clinical picture ranges widely from a patient with preserved respiratory drive with pursed-lip breathing to one who is cyanosed, lethargic and mildly dyspnoeic. Wheezes may be heard on inspiration, but especially on expiration.

Acute pulmonary oedema can mimic or coexist with either of these conditions. However, the commonest cause of pulmonary oedema is left ventricular failure associated with ischaemic heart disease. Although there are many other causes, these will be seen only occasionally in most hospitals (see box below).

An idea of the 'chance' of meeting such a condition is displayed on an arbitrary scale in the box.

Causes of acute pulmonary oedema and 'chance' of meeting the condition*

Cause	Chance
Ischaemic heart disease	Daily
Myocardial infarction	
Cardiac dysrhythmias	
Fluid overload	Weekly
Severe hypertension	
Aortic stenosis/regurgitation	Monthly
Mitral stenosis/regurgitation	
Cardiomyopathy	
Non-cardiac	
Pulmonary oedema (ARDS, pneumonia)	
Left ventricular aneurysm	Annually
Infective endocarditis	
Cardiac tamponade	
Left atrial myxoma	Only in examinations

*Patients with some of the more common causes of pulmonary oedema may also feature in examinations.

Features that would support a diagnosis of pulmonary oedema include poor peripheral perfusion, absence of both neck vein distension and chest hyperexpansion. In addition, the percussion note is often dull, particularly at the lung bases. There are usually fine inspiratory crackles on auscultation and occasionally signs of a pleural effusion.

A deviated trachea (a very late sign) should alert the clinician to the possibility of a tension pneumothorax. Other signs, in particular tachypnoea, raised neck veins, reduced expansion, a hyperresonant percussion note and absent breath sounds, should be sought on the opposite side to the tracheal deviation.

Resuscitation

Irrespective of the underlying cause of the bronchospasm, treat the patients with nebulised bronchodilators whilst clues to the cause are sought. The clinical features described above will have helped distinguish bronchospasm due to asthma, COPD and pulmonary oedema.

Immediate management of a tension pneumothorax is needle thoracentesis, followed by intravenous access and then chest drain insertion.

More comprehensive details of these conditions including pathophysiology, assessment and management can be found in Chapter 8.

Time Out 5.1

a Define: (i) tidal volume; (ii) minute volume
b How does the respiratory rate affect alveolar ventilation?
c Sketch a graph showing the relationship between PaO_2 and $\%SpO_2$.
d List the immediately life-threatening conditions that affect 'B'.

SUMMARY

Breathing is rapidly assessed using the look, feel, percuss and listen sequence to identify life-threatening:

- bronchospasm
- pulmonary oedema
- tension pneumothorax
- critical oxygen desaturation.

Life-threatening bronchospasm is common and treated initially with oxygen-driven bronchodilators. Tension pneumothorax is rare but, when present, requires immediate decompression with a needle thoracocentesis.

CHAPTER 6
Circulation assessment

OBJECTIVES

After reading this chapter you will be able to:
- understand the physiology of tissue perfusion
- describe a structured approach to circulatory assessment
- identify the immediately life-threatening causes of shock
- identify the anatomy for peripheral and central venous cannulation.

INTRODUCTION

The specific aim in 'C' is to identify and treat shock. The related immediately life-threatening conditions are shown in the box.

Immediately life-threatening conditions

Airway
- **Obstruction (see full list in box 1 in Chapter 4)**

Breathing
- **Acute severe asthma**
- **Acute exacerbation of chronic obstructive pulmonary disease (COPD)**
- **Pulmonary oedema**
- **Tension pneumothorax**
- **Critical oxygen desaturation**

Circulation
- **Shock**

It is important to understand the mechanisms that maintain tissue perfusion in health before considering the effects of disrupting the circulation.

RELEVANT PHYSIOLOGY

Blood flow to the tissues

The amount of blood reaching a particular organ depends on several factors:
- venous system
- cardiac output
- arterial system
- organ autoregulation.

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Venous system

This acts as a reservoir for over 70% of the circulating blood volume and is therefore often referred to as a **capacitance system**. The volume of blood stored at any one time depends on the size of the vessel lumen. This is controlled by sympathetic innervation and local factors (see later), which can alter the tone of the vessel walls. If the veins dilate, more blood remains in the venous system and less returns to the heart. Should there be a need to increase venous return, sympathetic stimulation reduces the diameter of the veins and hence the capacity of the venous system. A change from minimal to maximal tone can increase the venous return by approximately 1 litre in an adult.

Cardiac output

This is defined as the volume of blood ejected by each ventricle per minute. Clearly, over a period of time, the output of the two ventricles must be the same (or else all the circulating volume would eventually end up in either the systemic or pulmonary circulation). The cardiac output equates to the volume of blood ejected with each beat (stroke volume in ml) multiplied by the heart rate (beats per minute) and is expressed in litres per minute.

Cardiac output = stroke volume × heart rate = 4 − 6 l/min (normal adult)

To allow a comparison between patients of different sizes, the term cardiac index is used. This is the cardiac output divided by the surface area of the person and hence is measured in litres per square metre.

Cardiac index = cardiac output/body surface area
= 2.8 − 4.2 l/min/m^2 (normal adult)

The cardiac output can be affected by:

- preload
- myocardial contractility
- afterload
- heart rate.

Preload

This is the volume of blood in the ventricle at the end of diastole. The left ventricular end diastolic volume is about 140 ml and the stroke volume (SV) is 90 ml. Therefore, the end systolic volume is approximately 50 ml and the left ventricular ejection fraction (SV/EDV) ranges from 50 to 70%.

During diastole, the cardiac muscle fibres are progressively stretched as the ventricular volume increases in proportion to the venous return. Remember that **the more the myocardial fibres are stretched during diastole, the more forcibly they contract during systole; hence more blood will be expelled** (Starling's law). Therefore, the greater the preload, the greater the stroke volume. However, this phenomenon has an upper limit (due to the internal molecular structure of muscle cells) so that if the muscle is stretched beyond this point then a smaller contraction is produced (a situation that exists in ventricular failure).

Thus, the end diastolic fibre length is proportional to the end diastolic volume or force distending the ventricle. A clinical estimate of this volume, or force, is the end diastolic pressure (EDP). As the ventricular end diastolic pressure increases so does the stroke volume. If the end diastolic pressure exceeds a critical level then the force of contraction declines and eventually ventricular failure ensues (Fig. 6.1).

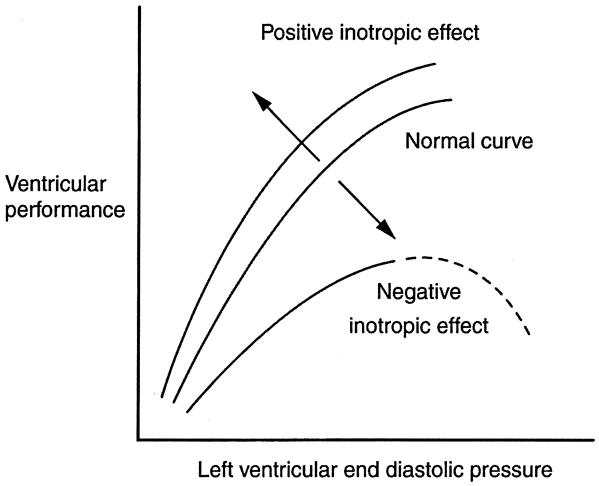

Fig. 6.1 Ventricular performance.

Current haemodynamic monitoring is based on measurements from a pulmonary artery flotation catheter. A commonly used recording is the pulmonary artery occlusion pressure because it is a useful estimate of the left ventricular end diastolic pressure.

Myocardial contractility

This is the rate at which the myocardial fibres contract for a given degree of stretch. Substances affecting myocardial contractility are termed inotropes, and they can be positive or negative in their actions. A positive inotrope produces a greater contraction for a given length (or end diastolic pressure (EDP)) (Fig. 6.1). Adrenaline and dopamine are naturally produced substances which have this effect. Dobutamine is a synthetic catecholamine with positive inotropic activity (however, it also has a potent vasodilator effect and must be used with caution). Therefore, depending on where you work, you may find some (or all) of these agents are used as part of the treatment of cardiogenic and septic shock.

Negative inotropes reduce contractility for a given muscle length (Fig. 6.1). These substances are often drugs, e.g. antiarrhythmics and anaesthetic agents. Rapid sequence induction of intravenous anaesthesia can precipitate circulatory collapse, if consideration has not been given to adequate fluid resuscitation and adjustment of dosage of induction agent (e.g. propofol or thiopentone) in the shocked patient. Many pathological states will also depress contractility,e.g. hypoxaemia, acidosis and sepsis. Myocardial damage also has a negative inotropic effect.

Afterload

As the left and right ventricular muscle contracts, pressures within the chambers increase until they exceed those in the aorta and pulmonary artery, respectively. The aortic and pulmonary valves open and the blood is ejected. The resistance faced by the ventricular myocardium during ejection is termed the afterload. In the left ventricle, this is mainly due to the resistance offered by the aortic valve and the compliance (stiffness) of the arterial tree. Usually this latter component is the most important and is estimated by calculating the **systemic vascular resistance**.

Using Ohm's law, where resistance equals pressure divided by flow, the systemic vascular resistance is defined as the mean arterial and venous pressure difference divided by the cardiac output.

(mean arterial pressure − central venous pressure) × 80/cardiac output =
770 − 1500 dyn/s/cm^5 (normal adult)

The value of 80 comes from converting mm Hg to SI units.

Key point

Reducing the afterload for a given preload will allow the myocardial fibres to shorten more quickly and by a greater amount. It therefore increases the stroke volume and cardiac output

Heart rate

An increase in heart rate is mediated via β_1 adrenoreceptors. These can be stimulated by the sympathetic nervous system, the release of catecholamines from the adrenal medulla and drugs (e.g. isoprenaline). This is termed a **positive chronotropic effect**. Conversely, the parasympathetic nervous system (PSNS) supplies the sinoatrial node and atrioventricular node via the vagus nerve. Stimulation of the PSNS decreases heart rate; i.e. it has a **negative chronotropic effect**. This effect can also be produced by drugs that inhibit the sympathetic nervous system, such as β blockers. In contrast, an increased heart rate may follow inhibition of the parasympathetic nervous system muscarinic receptors, e.g. atropine.

An increase in the heart rate can lead to an increase in cardiac output (see equation earlier). However, ventricular filling occurs during diastole and this phase of the cardiac cycle is shortened as the heart rate increases. A sinus tachycardia, above 160 beats/m in the young adult, drastically reduces the time during diastole for ventricular filling. This leads to a progressively smaller stroke volume and a fall in cardiac output. The critical heart rate also depends on the age of the patient and the condition of the heart; e.g. rates over 120 beats/min may cause inadequate filling in the elderly.

Key point

Increasing the heart rate will only lead to rise in the cardiac output if the rate is below a critical level (which differs in different individuals depending on age and the condition of the myocardium)

The main factors affecting the cardiac output are summarised in the box.

Main factors affecting cardiac output (of the left ventricle)

Preload, or left ventricular end diastolic volume
Myocardial contractility
Afterload, or systemic vascular resistance
Heart rate

The arterial system

The walls of the aorta and other large arteries contain relatively large amounts of elastic tissue that stretches during systole and recoils during diastole. In contrast, the walls of arterioles contain relatively more smooth muscle. This is innervated by the sympathetic nervous system that maintains vasomotor tone. Stimulation of α adrenoreceptors causes vasoconstriction. A total loss of arterial tone would increase the capacity of the circulatory system so much that the total blood volume would be insufficient to fill it. As a consequence, the blood pressure would fall and the flow through organs would depend on their resistance. Some organs would receive more than normal amounts of oxygenated blood (e.g. skin) at the expense of others which would receive less (e.g. brain). To prevent this, the arterial system is under constant control by the sympathetic nervous system and local factors, to ensure that blood goes where it is needed most. This is exemplified in the shocked patient, where differential vasoconstriction is aimed at maintaining supply to the vital circuit of heart, lungs and brain at the expense of others (e.g. skin). Hence the skin is cold and looks pale in some states of shock (e.g. hypovolaemic shock).

Blood volume

Adult male = 70 ml/kg ideal body weight
Adult female = 60 ml/kg ideal body weight

Systemic arterial blood pressure

This is the pressure exerted on the walls of the arteries. Systolic is the maximum and diastolic is the minimum pressure generated in the large arteries during the cardiac cycle. The difference between them is the pulse pressure. The **mean arterial pressure** is the average pressure during the cardiac cycle and is approximately equal to the diastolic pressure plus one third of the pulse pressure. As the mean arterial pressure is the product of the cardiac output and the systemic vascular resistance, it is affected by all the factors already discussed.

Autoregulation

Organs have a limited ability to regulate their own blood supply so that perfusion is maintained as blood pressure varies. This process, known as autoregulation, depends on the physiological control of smooth muscle tone in arteriolar walls. By altering the calibre of the vessels, flow to the organ is maintained over a wide range of arterial pressures. Other local factors, such as products of anaerobic metabolism, acidosis and a rise in temperature, all cause the local vascular tree to dilate. These local factors also shift the oxygen/haemoglobin dissociation curve to the right (see page 49).

Such effects enable active tissues to receive increased quantities of oxygenated blood and nutrients, and for this blood to 'give up' its oxygen more easily. Thus, the body attempts to halt the transition from reversible (where appropriate resuscitation will succeed) to irreversible shock. In the latter, the local tissue circulation has become so disrupted with large amounts of toxins from anaerobic metabolism, that the body becomes overwhelmed by negative inotropes, prothrombotic and procoagulant substances.

Remember that autoregulation may not work under pathological conditions.

PRIMARY ASSESSMENT AND RESUSCITATION

A summary of the circulatory assessment is shown in the box.

Summary of circulatory assessment

Look	**Pallor, sweating, venous pressure**
Feel	**Pulse – rate, rhythm and character**
	Capillary refill time
	Blood pressure
	Apex beat
Listen	**Blood pressure, heart sounds, extra sounds, lung bases**

The aim of this brief assessment is to identify the patient who is shocked. This is a clinical syndrome resulting from inadequate delivery, or use, of essential substrates (e.g. oxygen) by vital organs. The causes of shock are described in detail in Chapter 9 and summarised in the next box.

Causes of shock

Preload reduction	**Hypovolaemia**	**Haemorrhage**
		Diarrhoea
		Excessive vomiting
		High fistulae loss
		Reduced intake
		Endothelial leak
	Impaired venous return	**Pregnancy**
		Pulmonary embolus
		Severe asthma
		Tension pneumothorax
Pump failure	**Endocardial**	**Acute valve lesion**
	Myocardial	**Infarction**
		Inflammation
		Dysrhythmia
	Epicardial	**Tamponade**
After load reduction	**Vasodilation**	**Sepsis**
		Anaphylaxis
		Toxic

SPECIFIC CLINICAL FEATURES

All patients with respiratory distress will have a tachycardia as described in the previous chapter. With severe airways obstruction, however, **pulsus paradoxus** may be present. Normally there is a reduction in systolic blood pressure of up to 10 mm Hg on inspiration. This is attributed to a fall in intrathoracic pressure (i.e. it becomes more negative on inspiration), which enlarges the pulmonary vascular bed and reduces return of blood to the left ventricle.

There is partial compensation by a simultaneous increase in right ventricular output. In severe asthma and COPD there is a more substantial fall in intrathoracic pressure on inspiration. This greatly increases the capacity of the pulmonary

vascular bed that in turn reduces output from the left ventricle, resulting in pulsus paradoxus. This is an exaggeration of the **normal** systolic fall on inspiration and not a paradoxical change in the pulse as the name would imply. The abnormality is the extent by which the arterial pressure falls. If severe, the pulse may disappear on inspiration and this can easily be palpated at the radial artery. In contrast, if the fall in systolic pressure is not so marked, it can be detected using the sphygmomanometer. This physical sign indicates critical circulatory volume deficiency and can also occur in patients with cardiac tamponade.

Another pulse abnormality is **pulsus alternans** where evenly spaced beats (in time) are alternately large and small in volume. As this can indicate left ventricular failure, the clinician should check for corroborative signs such as a displaced apex beat, a **third heart sound** and a pansystolic murmur of mitral regurgitation. A third heart sound in patients over 40 years usually indicates elevated ventricular end diastolic pressure. This is because with increasing age, the myocardium and associated valvular structures become less compliant, i.e. stiffer. Thus, an increase in end diastolic pressure is needed to ensure adequate ventricular filling, during which sudden tension in these structures generates vibrations which correspond to the third heart sound.

Hypovolaemia

Hypovolaemia, commonly due to blood loss, can present with tachypnoea (an early sign, resulting from metabolic acidaemia) and a variety of other physical signs including tachycardia, hypotension (often a late sign of decompensation) and reduced urine output. Bradycardia occurs in advanced/preterminal shock (see Chapter 9 for further details).

Pulmonary embolus

The size and position of the pulmonary embolus will determine its haemodynamic effects. Non-fatal emboli blocking the major branches of the pulmonary artery (PA) provoke a rise in PA pressure due to hypoxaemia and vasoconstriction. In addition, tachypnoea follows stimulation of alveolar and capillary receptors. An acute increase in pulmonary vascular resistance and hence right ventricular afterload causes a sudden rise in end diastolic pressure and dilatation of the right ventricle. This produces a raised jugular venous pressure, a fall in systemic arterial pressure and a compensatory tachycardia (see Chapter 9 on Shock for further details).

Dysrhythmia

Shock resulting from a dysrhythmia is due to either pulmonary oedema or hypotension or a combination of these conditions. Under these circumstances tachydysrhythmias, irrespective of the QRS complex width, will require cardioversion. Unfortunately, atrial fibrillation may either fail to cardiovert (especially when chronic) or only transiently return to sinus rhythm. Remember to anticoagulate the patient with chronic atrial fibrillation (>48 h) before cardioversion.

The remaining options include:

- chemical cardioversion. Of the many drugs available, intravenous amiodarone is well tolerated. Flecainide is an excellent alternative, but has been shown to increase mortality in patients with ischaemic heart disease
- control the ventricular response with intravenous digoxin or β blockers.

In contrast, a patient with a bradydysrhythmia may require temporary support with either atropine and an inotrope (e.g. adrenaline) or external pacing whilst preparations are made for transvenous pacing (see Chapter 9 on Shock for further details).

Cardiac tamponade

The signs of cardiac tamponade include pulsus paradoxus, raised internal jugular venous pressure that increases on inspiration (the opposite of normal; known as Kussmaul's sign) and an impalpable apex beat. As fluid accumulates, the elevated pressure in the pericardial sac is raised further during inspiration (this may be related to the downward displacement of the diaphragm). A corresponding increase is seen in the right atrial and central venous pressures. In contrast, pressures on the left side of the heart may be lower than that in the pericardium. As a consequence, filling of the left ventricle is compromised, stroke volume is reduced and the interventricular septum bulges into the left ventricular cavity. Thus, the stroke volume of the right ventricle is maintained at the expense of the left ventricle, which collapses on inspiration. With further increases in pericardial pressure there is diastolic collapse of the right atrium and ventricle. The venous pressure is always raised and is due to abnormal right heart filling. Kussmaul's sign can also be seen in right ventricular disease and pulmonary hypertension.

TREATMENT

All critically ill patients should receive high concentrations of inspired oxygen and have their oxygen saturation, pulse, blood pressure and cardiac rhythm monitored. Intravenous access is needed and at least one large cannula (12–14 gauge) is required.

Hypovolaemia

In acute hypovolaemia a fluid challenge should be given whilst the cause, usually haemorrhage, is sought (see Chapter 9). Chronic fluid depletion often presents as dehydration with features of acute renal impairment. Oxygen and careful fluid replacement are required, especially in patients with pre-existing cardiac conditions. Diuretics and angiotensin-converting enzyme inhibitors are a common cause of this problem in patients with a history of left ventricular failure. These drugs should be stopped and fluid replacement titrated against the patient's clinical condition and central venous pressure.

Acute severe left ventricular failure

The blood pressure is probably the most important feature in determining treatment. The combination of acute pulmonary oedema and hypotension requires inotropic support. Any patient who has a systolic pressure of less than 90 mm Hg should not be given diuretics, nitrates or opiates as their immediate action is to cause venodilatation. This, in turn, will reduce the cardiac preload and potentially exacerbate hypotension. However, once an inotrope has been started and the patient's condition is improving, a diuretic may be used to 'clear' pulmonary oedema.

Dysrhythmia – tachycardia

The acutely ill patient can develop a tachydysrhythmia in response to a variety of non-cardiac conditions:

- Hypoxaemia
- Hypovolaemia
- Hypokalaemia
- Hypomagnesaemia
- Acidosis
- Hypercarbia.

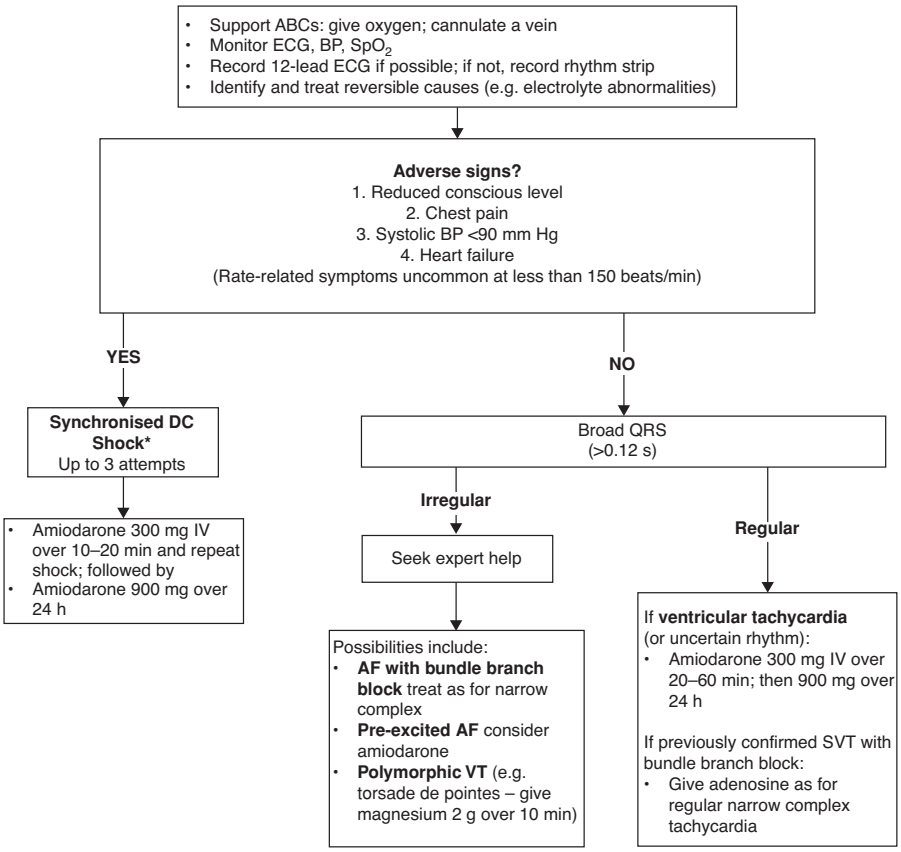

Fig. 6.2 Management of a broad complex tachycardia (UK and European guidelines). [Reproduced with permission from the Resuscitation Council (UK) and extracted from the Adult Tachycardia Algorithm.]

A tachydysrhythmia in the shocked or haemodynamically compromised patient necessitates electrical cardioversion (see Chapter 32). This is more likely to be successful if non-cardiac conditions have been treated. If cardio-version fails then drug treatment according to UK and European resuscitation committee guidelines is advocated (Figs 6.2 and 6.3). Remember that a sinus tachycardia can be the response of a failing ventricle or any other cause of shock. However, if the patient has another baseline rhythm such as atrial fibrillation, the increased sympathetic drive would result in atrial fibrillation with a rapid ventricular response. It can be difficult to decide whether a dysrhythmia is the cause of shock or vice versa. Previous ECGs are invaluable in this circumstance. If no such information is available, the treatment is dictated by the clinician's judgement. The following key points can help in this dilemma:

- A supraventricular tachycardia with a ventricular response of less than 150 is unlikely to cause failure.
- A broad complex tachycardia is almost always ventricular in a patient with ischaemic heart disease (>90%).

Time Out 6.1

Ensure that you have a sound understanding of this protocol (Fig. 6.2). If necessary take five minutes and copy the tachydysrhythmia management flow diagram to reinforce your knowledge.

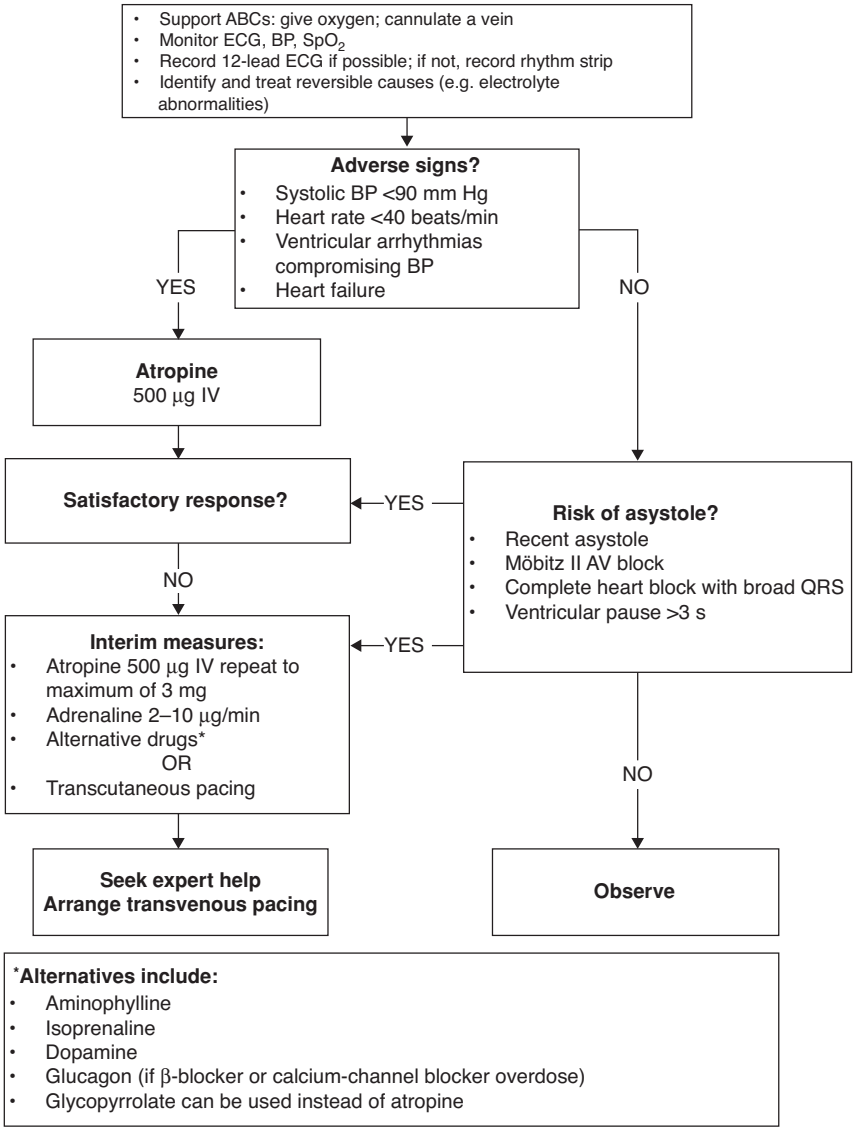

Fig. 6.3 Management of a bradydysrhythmia (UK and European guidelines). (Reproduced with permission from Resuscitation Council (UK).)

Dysrhythmia – bradycardia

This is treated according to UK and European resuscitation committee guidelines (Fig. 6.3).

Time Out 6.2

In a patient with a bradycardia, what are the risk factors for asystole? If you cannot answer this question, copy the bradycardia management flow diagram (Fig. 6.3) to reinforce your knowledge.

Pulmonary embolus

The minimum immediate management comprises high inspired concentrations of oxygen and anticoagulation. More comprehensive treatment details are provided in Chapters 8–10.

Cardiac tamponade

If this diagnosis is suspected clinically, then intravenous fluid should be given to raise the end diastolic pressure and volume to maintain the cardiac output. This is only a temporising procedure and immediate cardiological referral is required for echocardiography and pericardiocentesis (see Chapter 10).

INVESTIGATIONS

Appropriate investigations include:

- a full blood count to exclude anaemia (possibly exacerbating left ventricular failure)
- urea and electrolytes for baseline values particularly in patients who are being treated with vasodilators, diuretics or inotropes
- markers of myocardial injury troponin I or T, cardiac enzymes
- arterial blood gases
- 12-lead ECG
- portable chest X-ray
- echocardiography in selected cases.

Key point

If the patient is still breathless and the cause remains in doubt, a rapid re-evaluation of A, B and C is required

It is important to remember that hypovolaemia is an important cause of breathlessness

Once the patient's condition is stabilised, further information can be obtained from the secondary assessment.

Time Out 6.3

List the major causes of shock.

SUMMARY

- Tissue perfusion relies on venous return, myocardial contractility, afterload and autoregulation.
- Failure of one or more of these components will result in shock.
- In the primary assessment, the immediately life-threatening problems are:

– Airway	Obstruction
– Breathing	Acute severe asthma
	Acute exacerbation of COPD
	Pulmonary oedema
	Tension pneumothorax
	Critical oxygen desaturation
– Circulation	Shock

These conditions can be identified and differentiated clinically.

- All shocked patients require oxygen and intravenous access.

Advanced Life Support Group

CHAPTER 7
Disability assessment

OBJECTIVES

After reading this chapter you will be able to:

- describe the neurological examination in both the primary and secondary assessment phases.

PRIMARY NEUROLOGICAL ASSESSMENT

This is the D component as described in Chapter 3. This brief examination comprises:

- pupil size and response to light
- Glasgow Coma Score
- lateralising signs
- signs of meningeal irritation
- glucose

SECONDARY NEUROLOGICAL ASSESSMENT

The most important component of the neurological examination is the history and this will follow the normal 'phrased' format (see Chapter 3). Particular attention should be directed at the key features shown in the box.

Key neurological features

Define the problem	
Describe the deficit	
Determine the	**Onset**
	Pattern
	Extent and duration
Associated symptoms	**Neurological**
	Other

A comprehensive neurological assessment is not required and so a screening examination will suffice. The components are listed in the box below.

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Components of the screening examination

Higher mental function
Speech
Pupil response
Visual fields
Fundoscopy
Eye movement
Facial sensation
Facial movement
Movement of mouth, tongue and palate
Motor
- **Look for wasting and fasciculation**
- **Test tone**
- **Power**
- **Reflexes**

Sensation
- **Light touch and pinprick test**
- **Joint position sense (proprioception)**

Coordination

Often higher function and speech are assessed whilst taking the patient's history.

Higher mental function tests

Whilst the clinical assessment of higher mental function is necessary for a comprehensive neurological examination, a full examination is rarely done in the first 24 h of admission. A brief assessment of cognition can be carried out using the AMTS (abbreviated mental test score; see Table 7.1). To allow comparison with scores obtained in the past it is important that the correct questions are asked and that they are scored consistently.

Table 7.1 Abbreviated mental test score

	Question	Assessment	Rating
1	How old are you?	Score for exact age only	
2	What is your date of birth?	Only date and month needed	
3	What is the year now?	Score for exact year only	
4	What is the time of the day?	Score if within 1 h of correct time	
5	Where are we? What is this building?	Score for exact place name, e.g. 'hospital' insufficient	
Now ask subject to remember an address: 42, West Street			
6	Who is the current monarch?	Score only current monarch	
7	What was the date of the first world war?	Score for year of start or finish	
8	Can you count down backwards from 20 to 1?	Score if no mistakes or any mistakes corrected spontaneously	
9	Can you tell me what those 2 people do for a living?	Score if recognises role of 2 people correctly, e.g. docor nurse	
10	Can you remember the address I gave you?	Score for exact recall only	
		TOTAL	/10

When to test higher mental function?

Usually when a doctor is suspicious that there is an underlying abnormality or occasionally if a concern is expressed by the patient or, more particularly, the family. Since delirium is frequently missed, there is an argument for assessing the AMTS in all patients who are admitted as an emergency (Table 7.1).

How to test?

Explain that you are going to ask a few questions that might seem strange.

A correct answer scores 1 mark. No half-marks are given. A score of 6 or below is abnormal.

Speech

Particular abnormalities that influence speech are shown in the box.

Important abnormalities affecting speech

Deafness
Dysphasia
Dysarthria
Dysphonia

The 'four D's' of speech can be easily assessed by remembering the following four questions.

Can the patient hear, understand, articulate and vocalise?

Lack of comprehension as well as failure of thought or word generation implies a dysphasia, of which there are two major types. Expressive or motor aphasia is where the patient can understand either verbal or written information but has aphasia or non-fluent speech. In contrast, poor comprehension and occasionally meaningless speech indicates receptive or sensory aphasia. These conditions are often referred to as Broca's or Wernicke's dysphasia respectively and occur predominantly in the dominant hemisphere.

Broca's dysphasia is usually a lesion in Broca's area in the inferior frontal gyrus that can be associated with a hemiplegia. In contrast, Wernicke's area (upper part of the temporal lobe and supramarginal gyrus of the parietal lobe) is often associated with a visual field defect. Total aphasia is a lesion of the dominant hemisphere that affects both Broca's and Wernicke's areas.

Key points in assessing aphasia

Establish whether the patient is right or left handed
Discover the first language
Ask the patient simple questions initially, requiring 'yes/no' answers
Increase complexity of questions starting with simple commands such as 'put out your tongue', followed by 'touch your right ear with your left index finger'
Always ensure that the patient has understood your instructions

Dysarthria is a failure of articulation that normally requires the coordination of breathing with movement of the vocal cords, larynx, tongue, palate and lips.

When taking the history, listen for slurring and the rhythm of speech along with the words or sounds which cause the greatest difficulty.

Types of dysarthria

Spastic – slurred, like 'Donald Duck' speech (also called pseudobulbar palsy)
Extrapyramidal – slurred and monotonous, e.g. in Parkinson's disease
Cerebellar – slurred, disjointed, scanning or staccato (equal emphasis on each syllable) seen in alcohol intoxication and disseminated sclerosis

Lower motor neurone lesions affecting speech include:

- facial (VII) – difficulty with the letters B, P, M and W
- palate (IX) – nasal speech (like nasal congestion)
- tongue (XII) – distorted speech with difficulty with T, S and D (bilateral lower motor neurone lesions of XII cause bulbar palsy).

KLM is an easier way of remembering (X, XII, VII):

- X – affects the soft palate so impairs the sound, 'kuh, kuh, kuh . . .'
- XII – affects the tongue so impairs the sound, 'la, la, la . . .'
- VII – affects the lips so impairs the sound, 'mi, mi, mi . . .'

Dysphonia is a disturbance of voice production that may indicate laryngopharyngeal pathology or an abnormality of the vagus. Dysphonia is not formally assessed unless the patient is unable to produce a normal volume of sound or speaks in a whisper.

Pupil response

Check the pupils for:

- symmetry
- reaction to both light and accommodation
- both direct and consensual reflexes.

Be careful to shine the light obliquely; a torch shone directly in front of the eye may produce an accommodation response. Remember that the afferent pathway for light reaction is the optic nerve whilst the efferent pathway is the parasympathetic component of the third nerve. In contrast, the accommodation reaction afferent pathway arises in the occipital cortex but the efferent pathway remains the parasympathetic component of the third nerve bilaterally (see Fig. 7.1).

Examination should include assessment of a relative afferent pupillary defect (RAPD) by moving the light from eye to eye. If there is a RAPD, the pupil in the affected side will dilate when the light moves to it (as the direct stimulus is less intense than the consensual one). RAPD may occur with prechiasmal pathology such as optic neuritis.

A summary of pupillary abnormalities is given in Table 7.2.

Visual acuity

Some effort should always be made to assess acuity. If there is any doubt that visual acuity may be reduced formal assessment using a Snellen chart is needed.

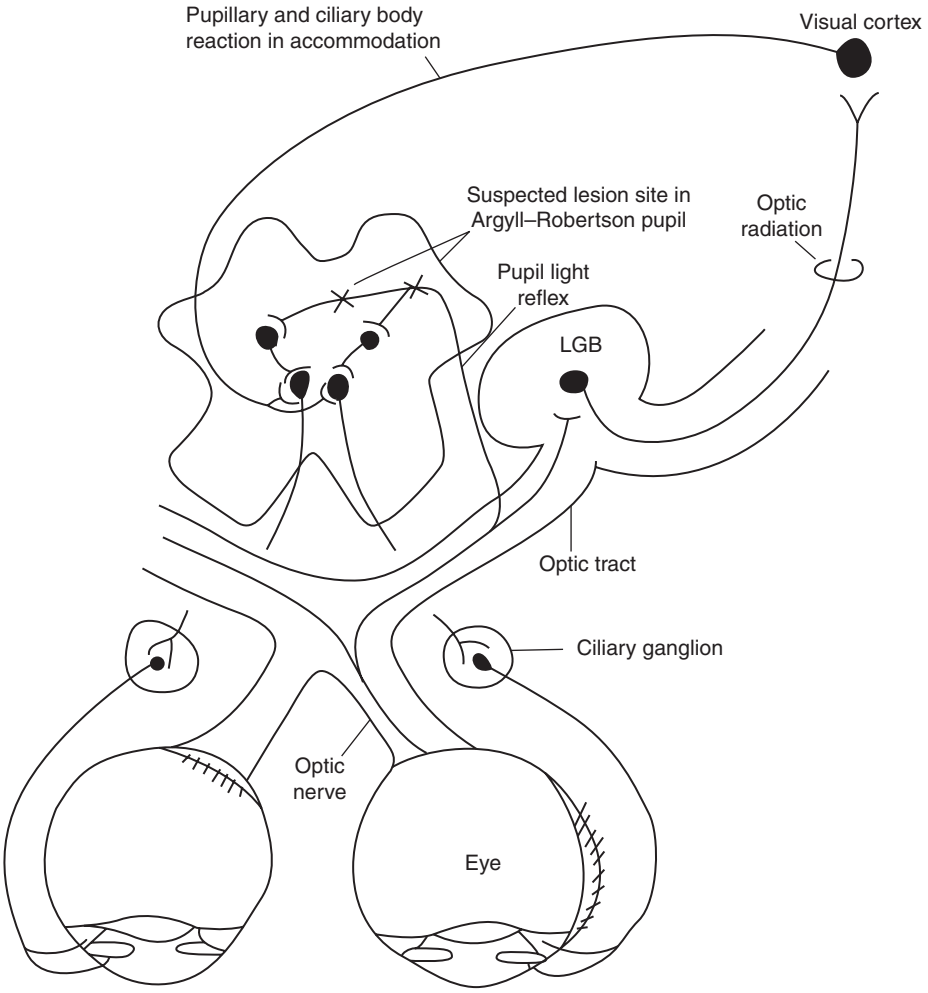

Fig. 7.1 Pathways for pupillary light reflex and accommodation.

Table 7.2 Pupillary abnormalities

	Pupillary response	Cause
Equal pupils	Small + reactive	Metabolic encephalopathy
		Midbrain herniation
		Senile miosis
	Pinpoint + fixed	Pontine lesion
		Opioids, organophosphates
	Dilated + reactive	Metabolic cause
		Midbrain lesion
		Ecstacy, amphetamines
	Dilated + fixed	Peri ictal
		Hypoxaemia
		Hypothermia
		Anticholinergics
Unequal pupils	Small + reactive	Horner's syndrome
	Small + 'non reactive'	Argyll Robertson (tertiary syphilis)
	Dilated + fixed	Uncal herniation
		IIIrd nerve palsy

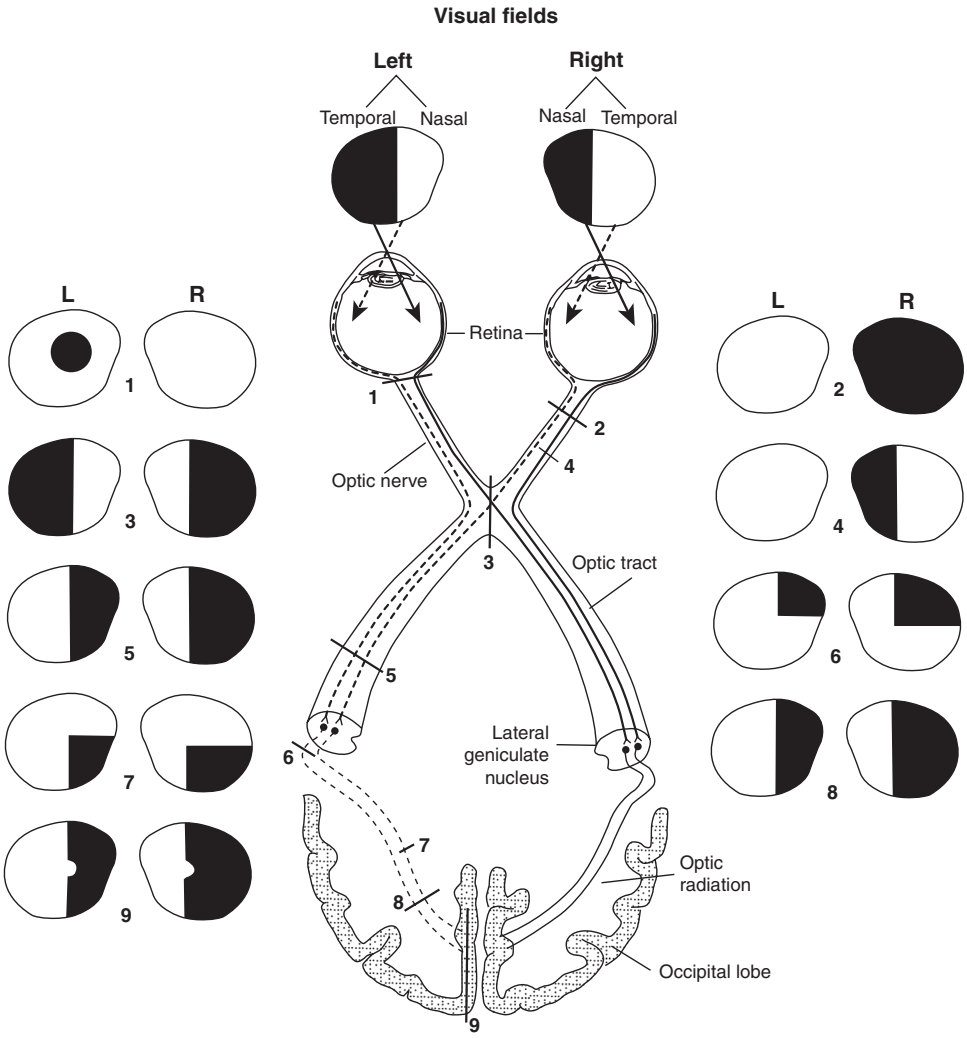

Fig. 7.2 Visual pathway with associated lesions.

Visual fields

These should be tested by confrontation which will identify any gross field defects. This way of assessing visual fields is quick, accurate and easy for patients to understand. To use this method:

- face the patient, who should cover one eye
- ask the patient to look at your corresponding pupil (i.e. their left to your right)
- hold up both hands with one or two fingers extended on each hand and ask the patient how many fingers there are in total.

All four quadrants should be covered. If an error is made, repeat changing the number of fingers in one hand to determine where the patient is not seeing well.

The visual pathway, with associated lesions, is demonstrated in Figs 7.2 and 7.3.

Interpretation

Monocular defects

These usually indicate ocular, retinal or optic nerve pathology.

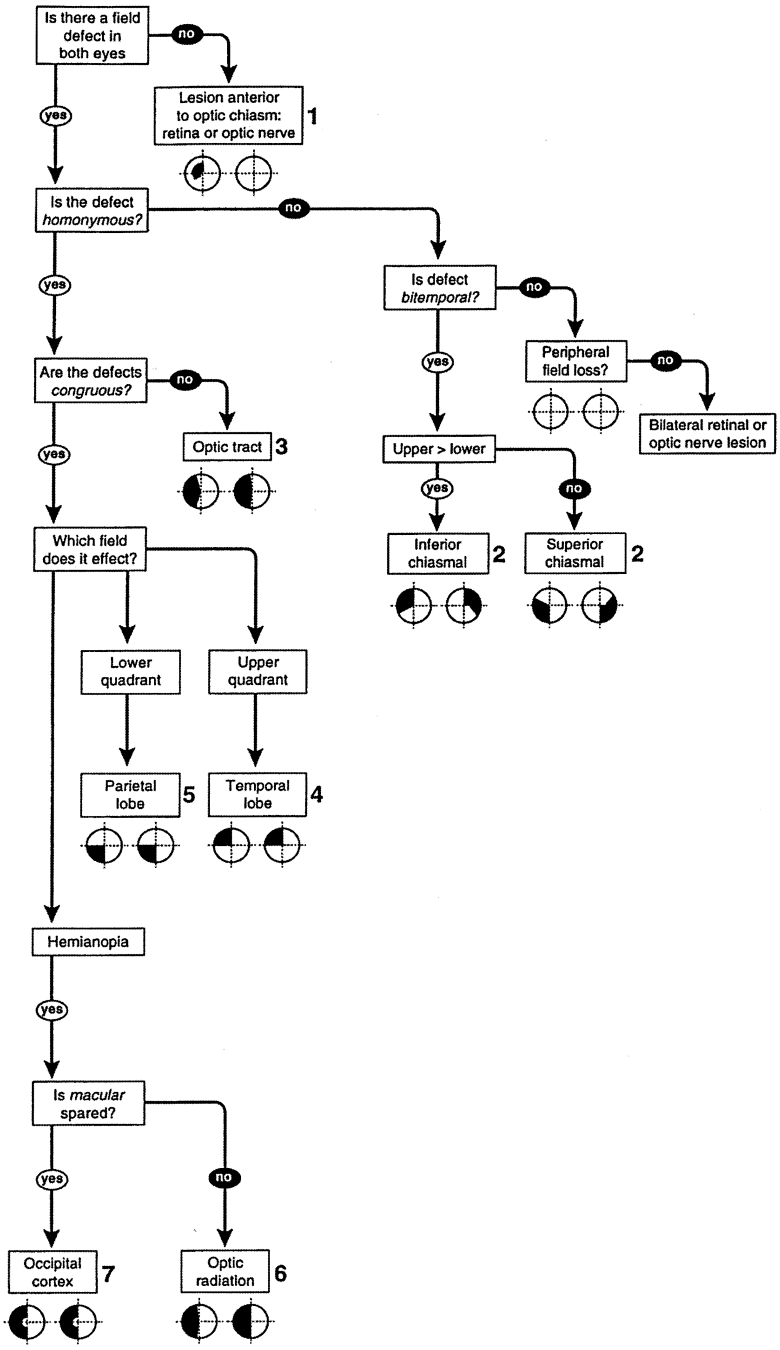

Fig. 7.3 Decision tree to determine the cause of visual field defects.

Key point

In monocular blindness due to a complete lesion of the optic nerve, the direct pupillary response to light is lost but the consensual response is retained

Tunnel vision

The size of the constricted field remains the same irrespective of the distance of the test object from the eye – usually functional.

Constricted field
The field is reduced but is less marked as the object moves away from the eye; seen with chronic papilloedema or glaucoma.

Scotoma
A hole in the visual field, e.g. disseminated sclerosis or neuropathy (toxic or ischaemic), retinal haemorrhage/infarct. This occasionally occurs with migraine but other, more serious causes should be sought and excluded.

Binocular defects
These indicate a lesion at or behind the optic chiasm or very rarely bilateral lesions in front of the chiasm.

Bitemporal hemianopia
It indicates a chiasmal lesion, usually a pituitary adenoma.

Homonymous hemianopia
A lesion anywhere along the optic tract including the occipital cortex where macular sparing may be evident.

Homonymous quandrantanopia
- upper = temporal lobe lesion
- lower = parietal lobe lesion.

Fundoscopy

Many people make fundoscopy harder than it need be. For a successful examination, darken the room to allow the pupils to dilate. Stand to the side of a seated patient rather than in front and check the red reflex. It is important to have a system when examining the fundus. Key features are shown in the box.

Key features when examining the eye

Optic disc – colour, cup and margins; the presence or absence of venous pulsation can be helpful
Blood vessels – arteries, arteriovenous junctions, vascular patterns. Remember that arteries are approximately two thirds the diameter of veins
Background – follow the four groups of vessels from the disc and examine each quadrant systematically

Papilloedema is often sought. Usually the patient will retain normal vision but the optic disc will appear pink with indistinct margins. It is important to remember that papilloedema can be absent even with raised intracranial pressure.

Causes of papilloedema

Raised intracranial pressure
Arterial hypertension
Raised venous pressure due to obstruction of cerebral venous drainage
Others including hypercarbia and severe anaemia

Eye movement

There are three control centres for eye movement.

Control centres for eye movement

Frontal lobe – command
Occipital lobe – pursuit
Cerebellar/vestibular nuclei – positional

The pathways from these three key centres are integrated in the brain stem to ensure that the eyes move together. In addition, the centre for lateral gaze is within the pons. The medial longitudinal fasciculus links the third, fourth and sixth nerve nuclei and these in turn control the external ocular muscles (see Fig. 7.4).

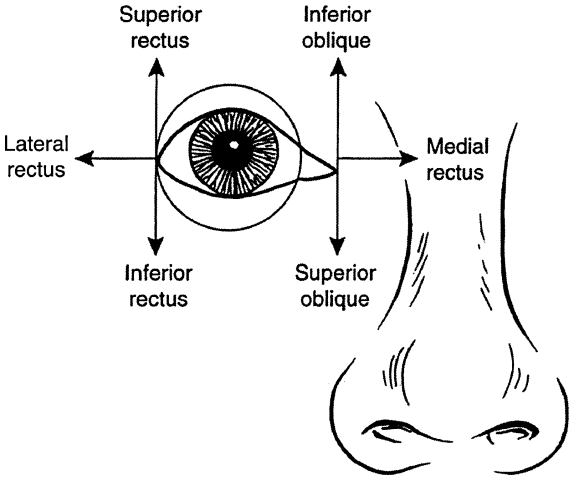

Fig. 7.4 External ocular muscles.

Nystagmus must also be actively sought. To avoid spurious nystagmus, the target should not be moved too quickly and/or too far away from the visual axis.

The commonest form of nystagmus is horizontal; vertical and rotary are rare. Specific examples include:

- Ataxic nystagmus, which is greater in the abducting than the adducting eye; caused by multiple sclerosis and cerebro-vascular disease.
- Multidirectional gaze evoked nystagmus which occurs in the direction of gaze, occurring in more than one direction. This is often seen in cerebellar syndromes, associated with drugs, alcohol or disseminated sclerosis.
- Unidirectional nystagmus, second or third degree horizontal nystagmus usually implies a cerebellar syndrome or central vestibular syndrome.
- Contrasting vertical horizontal nystagmus may be central or peripheral. The latter is usually associated with fatigue and vertigo.
- Downbeat nystagmus normally indicates a lesion at the level of the foramen magnum.

Third nerve palsies

Oculomotor nerve lesions can affect movement of the eye, the pupil and the upper eyelid. In complete third nerve palsy, there is ptosis, paralysis of the eye (except in abduction and intorsion, in which the eye is rotated 'down and out') and a

pupil that is dilated and paralysed to all stimuli. Sometimes the palsy is complete except pupil reaction remains; this indicates a 'medical' problem (e.g. diabetes) rather than compression. This 'pupil sparing rule' should be applied only in isolated third nerve palsies.

Facial sensation

Test for gross deficit using the finger tip in all three divisions of the trigeminal nerve. The corneal reflex should be tested since an abnormal response may be the first sign of trigeminal pathology. It is vital that the cornea rather than the conjunctiva is touched with a fine wisp of cotton wool and that the stimulus is brought in from the side.

Jaw jerk

This can be useful in patients with upper motor neurone signs in all limbs to help determine if the pathology is above or below the fifth nerve nucleus. If above, a pathologically brisk jaw jerk may be found.

Facial movement

After checking for symmetry, ask the patient to screw up their eyes tightly. Asymmetric power will result in the eyelids on the weaker side protruding more than on the stronger side. There are two points to remember: (1) ptosis is not due to a seventh nerve lesion; (2) corneal reflex usually has the ophthalmic branch of the trigeminal nerve as the afferent pathway whilst the efferent pathway uses the seventh nerve.

Seventh nerve palsies can be either upper motor neurone where only movements of the forehead and orbicularis oculi are preserved or lower motor neurone lesions when latter all movements on the affected side are involved.

Movement of the mouth, palate and tongue

Clues to problems in these three areas may have been elicited when testing the integrity of the fifth nerve and the patient's speech.

Abnormalities are shown in the box.

Abnormalities when examining the mouth

Small tongue with fasciculation – lower motor neurone lesion
Reduced range of tongue movement – bilateral upper motor neurone lesion often associated with labile emotions and pseudobulbar palsy
Tongue deviates to one side (with normal bulk) – unilateral upper motor neurone weakness associated with a stroke (common)
Unilateral wasting or fasciculation of tongue – unilateral lower motor neurone lesion (rare)
Uvula does not move on saying 'ah' or gag – bilateral palatal muscle paresis
Uvula moves to one side – contralateral upper or lower motor neurone lesion of the vagus nerve

Risk of aspiration after stroke

The best bedside way to assess the risk of aspiration in patients who have had a stroke is the water swallow test. Testing the gag reflex is much less helpful in this setting. The ability to swallow a small amount of water normally is associated with a low risk of aspiration; inability to do so is associated with a high risk and

such patients should normally be kept nil by mouth until expert assessment (such as by a speech and language therapist) has been made.

Table 7.3 Classification of muscular weakness

Type of weakness	Clinical features
Upper motor neurone lesion	Increased tone, increased reflexes, pyramidal pattern of weakness, i.e. weak arm extensors and leg flexors
Lower motor neurone lesion	Wasting, fasciculation, decreased tone, absent reflexes
Neuromuscular junction	Fatiguable weakness, normal or decreased tone, normal reflexes
Muscle disease	Wasting, decreased tone, impaired or absent reflexes
Functional weakness	No wasting, normal tone, normal reflexes, variable and inconsistent power

Motor system

Examination of the motor system is designed to detect muscular weakness. The five categories are given in Table 7.3.

Thus the sequence of your examination should include the following.

Observation

Look for wasting, fasciculation, posture and gait.

Tone

Ensure that the patient is either relaxed or distracted by conversation but remember that telling the patient to relax often has the opposite effect. Tone in the upper limb is assessed by passive movement of the wrist and elbow joints. Tone in the lower limbs is assessed by rolling the legs (with the hands on the patella) and observing movement at the ankle, and by lifting the knee quickly and observing if the heel slides along the surface (normal) or is lifted in the air (indicates increased tone). Common abnormalities in tone are listed in the box.

Common abnormalities in tone

Increased spasticity – upper motor neurone lesion (the tone will be greater in flexors in upper limb and extensors in lower limb). This is sometimes called 'clasp knife' rigidity

Increased rigidity – this is sometimes called 'lead pipe' rigidity as the resistance to movement is increased in all directions. The combination of increased rigidity and a tremor e.g. extrapyramidal syndromes like Parkinson's disease produces a cogwheel effect.

Reduced tone – lower motor neurone lesion

Power

When assessing any component of the nervous system, patient cooperation is vital. Instructions such as 'pull your foot towards your bottom' can seem complex and difficult to understand, especially when the patient is anxious. It is, therefore, recommended that you not only explain to the patient what you are going to do and what you would like them to do, but also show them. This demonstration can save a lot of time and frustration.

Before formally testing power, ask the patient to hold their arms out in front of their chest with palms uppermost and close their eyes. Watch the position of the arms as this will give you a clue as to underlying abnormalities. For example, pyramidal weakness will cause the arm to pronate and drift downwards. Muscular weakness, which can occur bilaterally, may make the arms drift downwards irrespective of whether the patient's eyes are open or closed. With cerebellar disease the arm may rise spontaneously; or a sharp tap on the back of the hand will cause exaggerated displacement, excessive compensatory return and overshoot. Disorders of joint position sense are manifested by the fingers moving up or down or the arm drifting, particularly when the eyes are closed.

If you suspect the patient's apparent weakness is factitious, ask the patient to lift one leg while you palpate under the other heel. If the patient is trying to lift one leg, there will be downwards pressure from the other side (unless there is bilateral paralysis of the lower limbs).

The key movements to be tested are listed in Table 7.4, along with the muscle, root value, and associated nerve.

The radial nerve supplies all the extensors in the arm. The ulnar nerve supplies all the intrinsic hand muscles except for the lateral two lumbricals, opponens pollicis, abductor pollicis brevis and flexor pollicis brevis. These muscles are supplied by the median nerve and can often be remembered by the acronym LOAF.

Once the muscles in the arms have been tested, it is easier to assess power in the legs rather than sensation in the arms. The reason for that will become apparent. The key movements and the appropriate muscles, nerves and root values for the lower limbs are listed in Table 7.5.

Table 7.4 Key movements of the arm

Movement	Muscle	Nerve	Root value
Shoulder abduction	Deltoid	Axillary	C5
Elbow flexion (with forearm supinated)	Biceps brachialis	Musculocutaneous	C5 C6
Elbow extension	Triceps	Radial	C6 C7
Finger extension	Extensor digitorum	Posterior interosseous	C7
Finger flexion	Flexor digitorum superficials and profundus	Median and ulnar	C8
Thumb abduction	Abductor pollicis brevis	Median	T1
Index finger abduction	First dorsal interosseous	Ulnar	T1
Index finger adduction	Second palmar interosseous	Ulnar	T1

Table 7.5 Key movements of the leg

Movement	Muscle	Nerve	Root
Hip flexion	Iliopsoas	Lumbar plexus	L1, 2
Knee extension	Quadriceps femoris	Femoral	L3, 4
Knee flexion	Hamstrings	Sciatic	L5, S1
Foot dorsiflexion	Tibialis anterior	Deep peroneal	L4/5
Foot plantarflexion	Gastrocnemius	Posterior tibial	S1
Big toe extension	Extensor digitorum longus	Deep peroneal	L5
Hip extension	Gluteus maximus	Inferior gluteal	L5, S1

When testing muscle power, always:

- ensure the joint is pain-free
- allow the patient to move the joint through the full range before testing power
- compare the strength of right side with left side
- grade your findings according to the MRC scale.

Power grading – MRC scale

5 = normal power
4 = moderate movement against resistance
3 = movement against gravity but not resistance
2 = movement with gravity eliminated
1 = flicker
0 = no movement

Reflexes

Remember that tendon reflexes are increased in upper motor neurone lesions and decreased with abnormalities in lower motor neurones and muscles. Reflexes are graded (see Table 7.6 below).

Table 7.6 Grading reflexes

4 = clonus
3 = increased
2 = normal
1 = diminished
0 = absent

When eliciting a tendon reflex:

- first palpate to ensure that the tendon is present and not tender
- make sure that the patient is relaxed
- use the whole length of the patella hammer and let it fall through a gentle arc – the force should come from gravity alone not from your arm muscles
- watch the belly of the muscle you are testing
- use reinforcement if a reflex is unobtainable directly. A way of doing this that patients find easy to understand is to ask them to clench a fist. Remember, reinforcement does not last for long, so ask the patient to clench the fist just as you are about to let the tendon hammer fall.

It may be easier to elicit the triceps reflex if the patient sits forward while their shoulder is passively extended. The triceps will now be on the upper surface of the upper arm and is easily accessible.

Striking the sole of the foot with the tendon hammer rather than striking the Achilles tendon elicits the ankle jerk more easily, comfortably and accurately.

The major reflexes are listed in Table 7.7 (see page 80).

In the presence of increased reflexes, check for ankle and patella clonus and for increased tone. Reflex abnormalities are listed in Table 7.8 (see page 80).

Reflexes can be absent in the early stages of a severe upper motor neurone lesion. This is often, inappropriately, referred to as spinal shock. There is no evidence of shock but the nerves have been in effect stunned. The classic features of an upper motor neurone lesion will develop subsequently.

Table 7.7 Major reflexes

Muscle	Nerve	Root
Triceps	Radial	C7
Brachioradialis (supinator)	Radial	C6
Biceps	Musculocutaneous	C5
Knee	Femoral	L3, 4
Ankle	Tibial	S1, 2

Table 7.8 Reflex abnormalities

Reflex response	Interpretation
Increased, clonus	Upper motor neurone lesion
Absent, generalized	Peripheral neuropathy
Absent, isolated	Lesion of either peripheral nerve or nerve root
Pendular	Cerebellar disease
Slow relaxing	Hypothyroidism
Inverted supinator – no elbow flexion on striking brachioradialis but finger flexion occurs	C6 myelopathy

The plantar or Babinski response completes the assessment of the major reflexes. It is important to be aware of the following:

- A positive Babinski response is manifested by extension of the big toe and spreading of the adjacent toes.
- A negative Babinski response may be found in an upper motor neurone lesion.
- A positive Babinski response that does not fit in with other neurological features should be interpreted with caution.

There are a number of other eponymous manoeuvres that supply the same information as the Babinski. Details do not need to be remembered, but it is useful to know that if the patient is unable to tolerate stimulus applied to the sole of the foot they will usually be able to tolerate the same stimulus applied to the tibial border.

A summary of the common motor abnormalities is shown in Table 7.4.

Sensation

The five basic types of sensation are shown in a cross section of the spinal cord in Fig. 7.5. It is important to remember that on entry to the cord, the spinothalamic tract (pain and temperature) will cross within one or two segments. In contrast, the posterior columns (joint position sense) remain ipsilateral until they cross in the medulla. Pinprick and light touch are rarely lost without discernible symptoms. As the different sensory modalities are carried predominantly in two tracts, the preliminary sensory examination should focus on pinprick and joint position sense.

The sensory examination is used:

- as a screening test
- for assessment of symptomatic patients
- to confirm signs detected on examination of the motor system.

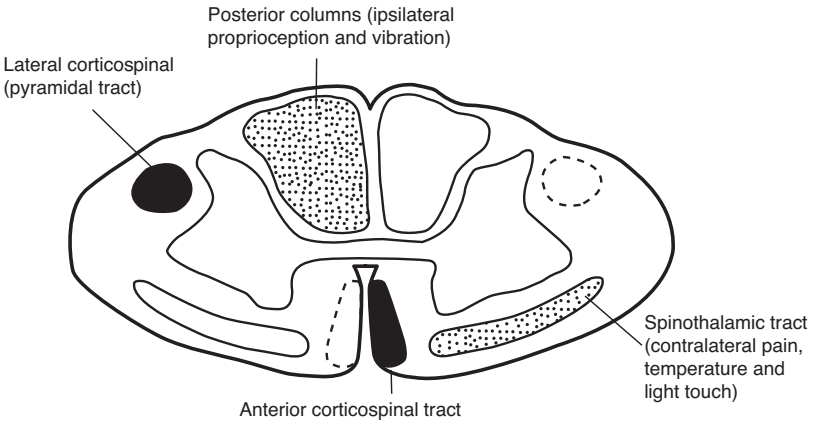

Fig. 7.5 Cross section of the spinal cord.

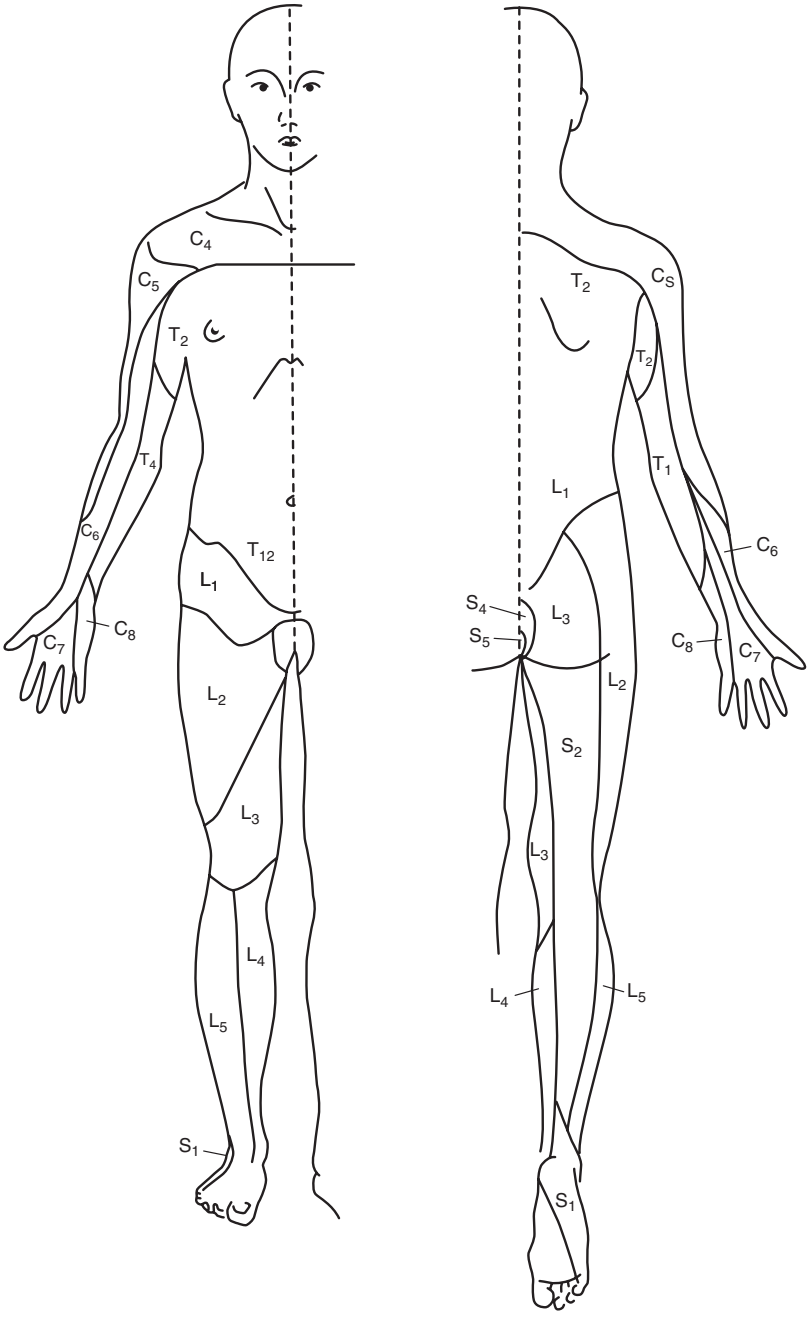

Fig. 7.6 Dermatomes.

There are several key points to consider when examining sensation. These are listed in the next box.

Key points for sensory examination

Explain to the patient what you are going to do
Demonstrate to the patient what you want them to do
Always ensure that the patient can appreciate the sensory modality to be tested
Assess dermatomes in a sequential fashion. The relevant dermatomes are shown in Fig. 7.6
Ask the patient to keep their eyes open but look away during the test. Patients usually find it easier to concentrate when their eyes are open than when closed

Pinprick

Use a 'neuro tip' not a hypodermic needle. Check intermittently using a blunt stimulus that the patient has recognised the 'pin' appropriately. During sensory assessment, test the integrity of each dermatome and whether there is any difference when comparing left and right sides of the body. It is also convenient at this time to test whether the patient can distinguish between two stimuli applied simultaneously to the left and right sides. Failure to do this indicates sensory inattention and therefore a parietal lobe lesion (most commonly non-dominant). Do not forget to test sacral sensation. Whilst this is not part of the screening test, it must be done in patients who have any of the following features (and should be accompanied in each of these cases by assessment of anal tone):

- urinary or bowel symptoms
- bilateral leg weakness
- sensory loss in both legs
- suspicion of a conus medullaris or cauda equina lesion.

Joint position sense (proprioception)

It is important to remember that Romberg's test assesses joint position sense.

Types and causes of sensory loss

Patterns of sensory loss fall into three broad categories:

- peripheral nerves
- spinal cord
- brain.

The major abnormalities for each category are listed in Tables 7.9 and 7.10.

A bizarre distribution of sensory loss which does not conform to an anatomical distribution is suggestive of a functional disorder. This, however, is a difficult diagnosis to make (and a dangerous one to rush into).

Coordination

This requires integration of sensory feedback and motor output. Thus it is logical to assess coordination after any sensory motor abnormalities have been identified. The cerebellum is responsible for integrating information related to coordination. A clue to an underlying abnormality is often present during examination of the motor system. Watch for the exaggerated response when the patient

Table 7.9 Peripheral nerve abnormalities

Lesion	Sensory loss	Cause
Single nerve lesions	Within the distribution of a single nerve	Entrapment neuropathy, diabetes mellitus, rheumatoid disease
Multiple single nerve lesions	Distribution of relevant nerves	Vasculitis or more diffuse neuropathy
Root lesion	Confined to single root or number of roots in close proximity	Prolapsed intervertebral disc
Peripheral nerves	Glove and stocking distribution	Diabetes mellitus, alcohol, thiamine deficiency

Table 7.10 Spinal cord and brain sensory loss

Lesion	Sensory loss	Cause
Complete transverse lesion	Loss of all modalities a few segments below the lesion	Trauma, spinal cord compression by tumour, transverse myelitis
Hemisection	Ipsilateral loss of joint position sense, contralateral loss of pain and temperature a few segments below the lesion	As with complete transection plus subacute combined degeneration of the cord and tabes dorsalis
Posterior column loss	Loss of joint position sense and vibration only	
Central cord syndrome	Loss of pain and temperature sensation at the level of the lesion	Syringomyelia
Anterior spinal syndrome	Loss of pain and temperature below the level of the lesion	Anterior spinal artery lesions
Brain stem	Loss of pain and temperature sensation in the face (ipsilateral) and body (contralateral)	Demyelination, lateral medullary syndrome
Thalamic	Hemicentral loss of all modalities	Stroke, tumour or disseminated sclerosis
Cortical parietal lobe dysfunction	The patient recognises all sensory modalities but localises them poorly. In addition, there is loss of sensory attention, two-point discrimination and astereognosis	Stroke, cerebral tumour, disseminated sclerosis

has outstretched arms and you tap the back of their hands. Tests for demonstrating coordination are complex activities. It is therefore important to tell the patient what you are going to do as well as demonstrate the movement required. Assess the finger–nose test (ensuring they have to extend the elbow to touch your finger), and the heel-shin test (ensuring the heel is slid down to the hallux as poor coordination may be evident only when the heel is moved this far) as well as the presence of truncal ataxia (if the patient is mobile assess their heel-toe walking).

Other signs indicating cerebellar dysfunction are shown in the box.

Signs of cerebellar dysfunction

Speech abnormalities: slurred, scanning, staccato
Nystagmus
Pendular reflexes
Truncal ataxia
Intention tremor
Hypotonia
Dysdiadochokinesis

Interpretation

The presence of unilateral incoordination suggests ipsilateral cerebellar disease, such as demyelination or vascular disease. In contrast bilateral signs usually reflect alcohol, drugs or demyelination. The presence of truncal ataxia and/or gait ataxia without limb coordination indicates a midline (vermis) lesion.

Time Out 7.1

Draw an outline of a man and label the appropriate dermatomes.

SUMMARY

This chapter has provided an overview of the key components of a screening neurological examination. Although there are many other tests that may be relevant to comprehensive neurological assessment, the framework provided in this chapter will enable detection of neurological signs.

The primary neurological assessment (D) comprises:

- pupil size and response to light
- Glasgow Coma Score
- lateralising signs
- signs of meningeal irritation.

The secondary neurological assessment comprises:

- the history, which is the most important component. It is important to develop a simple common personalised structure to neurological assessment. A screening neurological examination is the minimum that should be done
- examine the motor components in a logical, systematic fashion followed by sensory assessment. An isolated abnormality should be interpreted with caution. Neurological problems are associated with a well-described pattern of clinical features
- test coordination after motor and sensory assessment. Clues to the presence of cerebellar disease are obtained from other parts of the examination before coordination is assessed.

Unlike other components of the physical examination, the nervous system is often not examined as there are no clinical indications. It is therefore important to take every opportunity to hone your clinical neurological skills.

PART III

Presenting Complaints

Acute Medical Emergencies: The Practical Approach, Second Edition
Edited by Advanced Life Support Group
 ISBN: 978-0-727-91854-3

Advanced Life Support Group

CHAPTER 8

The patient with breathing difficulties

OBJECTIVES

After reading this chapter you will be able to:

- describe a structured approach to the breathless patient
- understand why a structured approach is important in the management of such patients
- differentiate between the immediately life-threatening and potentially life-threatening causes of breathlessness
- describe the immediate management of these patients and appropriate definitive care.

INTRODUCTION

Acute breathlessness is a common emergency condition. The effort required for breathing often makes it virtually impossible for the patient to provide any form of medical history and questioning may only make the situation worse. The clinician's skills will help to determine the underlying cause and dictate appropriate management.

Immediately life-threatening causes and signs of breathlessness

Airway

- **Obstruction (see full list in box 1 in Chapter 4)**

Breathing

- **Acute severe asthma**
- **Acute exacerbation of chronic obstructive pulmonary disease (COPD)**
- **Pulmonary oedema**
- **Tension pneumothorax**
- **Critical oxygen desaturation**

Circulation

- **Acute severe left ventricular failure**
- **Dysrhythmia**
- **Hypovolaemia**
- **Pulmonary embolus (PE)**
- **Cardiac tamponade**

Key point

It is important to remember that the breathless patient does not always have pathology arising primarily from the respiratory or cardiovascular systems

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PRIMARY ASSESSMENT AND RESUSCITATION

Airway

Assessment

This has been described in Chapter 4 and is summarised in the box below.

Summary of airway assessment

Look	Respiratory	Rate
		Effort
		Symmetry
Feel	Expired air	
	Trachea	
Listen	Count to 10′	
	Breath sounds	
	Added noises	

Resuscitation

High concentrations of inspired oxygen ($FiO_2 = 0{\cdot}85$) may relieve some of the patient's distress. If airway obstruction is suspected, request **immediate** review by a specialist. If a foreign body has been inhaled, attempt a Heimlich or modified Heimlich manoeuvre.

Breathing

Assessment

This has been discussed in Chapter 5 and is summarised in the box.

Summary of breathing assessment

Look	Colour, sweating
	Posture
	Respiratory – rate
	– effort
	– symmetry
Feel	Tracheal position
	Tracheal tug
	Chest expansion
Percuss, Listen	Anterior and posterior aspects of both lungs in the upper, middle and lower zones

Resuscitation

If bronchospasm is suspected, treat patients with oxygen-driven nebulised bronchodilators irrespective of the underlying cause, whilst clues to the cause are sought.

Immediate management of a tension pneumothorax is needle thoracocentesis followed by intravenous access and then chest drain insertion.

Time Out 8.1

Take 30 s to mentally rehearse the key components of your assessment.

Circulation

Assessment

This has been described in Chapter 6 and is summarised in the box.

Summary of circulatory assessment

Look	**Pallor, sweating, venous pressure**
Feel	**Pulse – rate, rhythm and character**
	Capillary refill time
	Blood pressure
	Apex beat
Listen	**Blood pressure, heart sounds, extra sounds, lung bases**

Resuscitation

All patients should receive high concentrations of inspired oxygen, be treated in a 'seated' position (if level of consciousness permits) and have their oxygen saturation, pulse, blood pressure and cardiac rhythm monitored. Intravenous access is necessary and at least one large cannula (12–14 gauge) is required.

The management of the 'shocked' patient will depend on the underlying cause. Treatment options are summarised in the box.

Treatment of shock

Cause	**Treatment**
Acute, severe, left ventricular failure	**Inotropes**
Dysrhythmia – Tachycardia	**Cardioversion**
– Bradycardia	**Atropine**
	Inotropes
	Pacing
Hypovolaemia	**Fluids**
Pulmonary embolus	**Anticoagulation**
	Thrombolysis
	Fluids
Sepsis	**Fluids**
	Antibiotics
	Inotropes
Anaphylaxis	**Adrenaline**
	Fluids
	Chlorpheniramine
	Hydrocortisone
Cardiac tamponade	**Fluids**
	Pericardiocentesis

Once the patient's condition is stabilised and the primary assessment completed, further information can be obtained from the secondary assessment.

Summary

In the breathless patient, the immediately life-threatening problems are:

• Airway	Obstruction
• Breathing	Acute severe asthma
	Acute exacerbation of COPD
	Pulmonary oedema
	Tension pneumothorax
	Critical oxygen desaturation
• Circulation	Acute severe left ventricular failure
	Dysrhythmia
	Hypovolaemia
	Pulmonary embolus
	Cardiac tamponade

These conditions can be identified and differentiated clinically.
All patients require oxygen and intravenous access.

Time Out 8.2

Mentally rehearse your approach to the patient with breathing difficulties. Then list the major components of the primary assessment. Armed with this structure read the following information and then answer the associated questions.

A old man with known ischaemic heart disease was admitted to the coronary care unit after becoming acutely breathless. He denied any chest pain or cough. The following physical signs were elicited:

- respiratory rate 26/min
- fine inspiratory crackles were heard at both bases
- pulse rate 140/min and regular
- blood pressure 80/50
- peripherally shut down
- no other relevant features

a What would be your immediate management?
b What investigations would you request?

SECONDARY ASSESSMENT

Many patients with breathlessness will be able to give a history, albeit fragmented. The conditions diagnosed in this assessment phase are shown in the box.

Potentially life-threatening causes of breathlessness

Respiratory

- **Asthma**
- **Acute on chronic respiratory failure**
- **Pulmonary oedema**

- **Simple pneumothorax**
- **Pneumonia**
- **Pleural effusion**
- **Pulmonary embolus**

Non-respiratory

- **Metabolic acidosis e.g. diabetic ketoacidosis, salicylate overdose**
- **Pontine haemorrhage**

SPECIFIC CONDITIONS

Asthma

Asthma is a chronic inflammatory condition resulting in reversible narrowing of the airways. It affects approximately 5% of the population and can occur for the first time at any age with a male predominance in childhood and females in later life. Asthma in children is usually associated with atopy, whilst in adults it is more commonly non-atopic. Both, however, have an inherited component.

Although there are many potential triggers, asthma is characterised by wheezing due to widespread narrowing of the peripheral airways. There may be an associated increase in sputum volume and viscosity. Occasionally a nocturnal cough will be a prominent symptom and patients may describe tightness in the chest or a sensation of choking rather than wheezing. Furthermore, exposure to external stimuli such as viruses, cold air, cigarette smoke and paint fumes may induce an acute 'asthmatic' attack. This does not indicate an allergic response but demonstrates that the airways are hyperreactive and produce an exaggerated response to non-specific irritants.

Pathophysiology

Acute attacks of bronchospasm may be precipitated by IgE mediated mast cell degranulation. In contrast, when exposed to environmental factors, e.g. allergens and pollutants, the airways of known asthmatics are susceptible to chronic inflammation characterised by eosinophil and T lymphocyte infiltration. These cells are responsible for liberating inflammatory mediators that evoke a variety of responses (see next box) culminating in airways narrowing and hence increased airflow resistance. Since resistance is inversely proportional to the fourth power of the radius (Poiseulle's law) a small increase in airways thickness will have a marked effect on airways resistance and therefore reduce airflow. The change in airway radius is usually due to bronchial muscle contraction, but in the asthmatic this is exacerbated by mucosal oedema, increased mucus production and epithelial cell damage. In addition, the chronic inflammatory response reduces elastic recoil of the airways, further exacerbating the narrowing (Fig. 8.1).

Inflammatory mediator induced changes in asthma

- **Disrupt the functional and structural integrity of the epithelium**
- **Stimulate** **mucus secretion**
  oedema formation
  smooth muscle contraction
- **Induce collagen deposition under the basement membrane.**

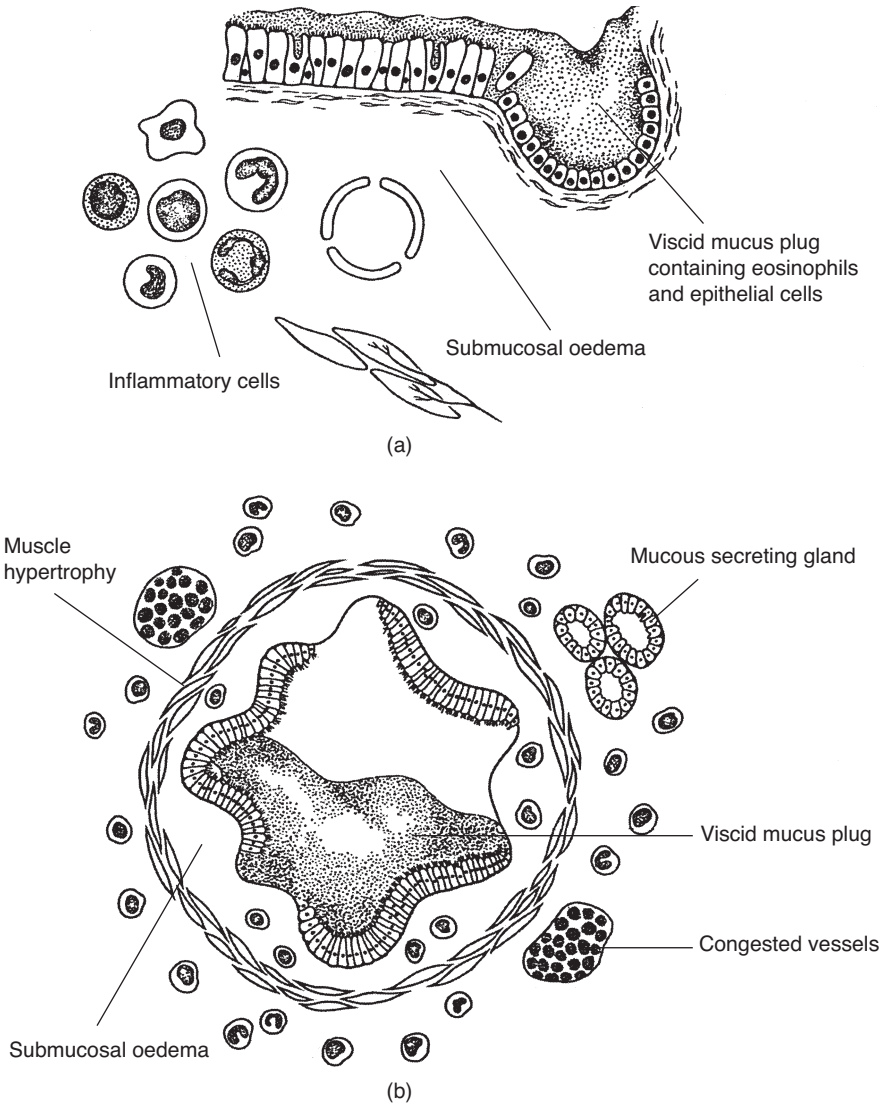

Fig. 8.1 Diagrammatic representation of the pathophysiology of asthma: (a) longitudinal section and (b) cross section.

This disturbed and decreased airflow is manifest clinically as audible wheeze, reduced forced expiratory volume in one second and peak expiratory flow rate, along with increased functional residual capacity due to air trapping. Thus, because of increased airways resistance the work of breathing is increased and the patient feels breathless.

In an acute asthmatic attack some of the airways become blocked by mucus plugs resulting in hypoxaemia due to ventilation perfusion (V/Q) mismatch. This further increases the work of breathing, causing hyperventilation in an attempt to reverse the hypoxaemia.

Key point

Failure to sustain this increased respiratory effort, usually in a severe exacerbation, will be manifest by a silent chest, hypoxaemia and a rising $PaCO_2$

Management

Key point

Preventable deaths from acute asthma still occur due to treatment delay

The management summarised in Fig. 8.2 is from the British Thoracic Society guidelines.

Life-threatening asthma

Assessment

This is characterised by:

- airway – normally patent but can be compromised by exhaustion
- breathing – cyanosed, exhausted, minimal respiratory effort and a silent chest
- circulation – tachycardia greater than 130 beats per minute or bradycardia, hypotension.

In addition, the peak expiratory flow is less than 33% of the predicted or the patient's best.

Key point

A silent chest is a life-threatening sign as it means there is insufficient air being moved (in and out of the chest) to generate a wheeze

Immediate treatment

All patients need high concentrations of inspired oxygen ($FiO_2 = 0.85$); oxygen should also be used to drive nebulised bronchodilators. See Fig. 8.2. It is important to remember that acute breathlessness in an asthmatic is usually due to bronchospasm. However, because of 'gas trapping' there is an increase in positive end expiratory pressure. This increases the potential to develop a pneumothorax that can further inhibit the respiratory and cardiovascular systems. Always be alert to this possibility in asthmatics who either fail to respond to treatment or become acutely breathless. Regular reassessment and an urgent chest X-ray are required.

Role/action management

Intravenous fluids should be given as most patients have coexisting dehydration (increased insensible losses, the possibility of an underlying chest infection and reduced intake). Adequate hydration also helps to render the sputum less tenacious. In addition, hypokalaemia can occur as a consequence of either asthma or coexistent β_2 agonist therapy. Thus, careful monitoring and appropriate replacement therapy are required.

The patient's clinical response to treatment (as described earlier) should be monitored continuously along with frequent arterial blood gases.

If the patient either becomes exhausted, retains CO_2 or adequate oxygenation is not possible then intermittent positive pressure ventilation will be required (see box). Early liaison with the anaesthetist/intensivist is vital. Patients who have previously required intubation and ventilation have an increased risk of requiring this again during an acute severe attack.

Indications for intensive care

Hypoxaemia (PaO_2 < 8 kPa despite FiO_2 > 0.6)
Hypercapnia ($PaCO_2$ > 6 kPa)
Acidaemia
Exhaustion
Altered conscious level (confused, drowsy, unconscious)
Respiratory arrest

MANAGEMENT OF ACUTE ASTHMA IN ADULTS ASSESSMENT OF SEVERE ASTHMA	
B	Health care professionals must be aware that patients with severe asthma and one or more adverse psychosocial factors are at risk of death
☑	• Keep patients who have had near fatal asthma or brittle asthma under specialist supervision indefinitely • A respiratory specialist should follow up patients admitted with severe asthma for at least one year after admission

INITIAL ASSESSMENT	
MODERATE EXACERBATION	LIFE THREATENING
• Increasing symptoms • PEF >50-75% best or predicted • No features of acute severe asthma	In a patient with severe asthma any one of: • PEF <33% best or predicted • SpO_2 <92% • PaO_2 <8 kPa • normal $PaCO_2$ (4.6-6.0 kPa) • silent chest • cyanosis • poor respiratory effort • arrhythmia • exhaustion, altered conscious level
ACUTE SEVERE	NEAR FATAL
Any one of: • PEF 33-50% best or predicted • respiratory rate ≥ 25/min heart rate ≥ 110/min • inability to complete sentences in one breath	Raised $PaCO_2$ and/or requiring mechanical ventilation with raised inflation pressures

Clinical features	Severe breathlessness (including too breathless to compete sentences in one breath), tachypnoea, tachycardia, silent chest, cyanosis or collapse *None of these singly or together is specific and their absence does not exclude a severe attack*
PEF or FEV_1	PEF or FEV_1 are useful and valid measures of airway calibre. PEF expressed as a % of the patient's previous best value is most useful clinically. In the absence of this, PEF as a % of predicted is a rough guide
Pulse oximetry	Oxygen saturation (SpO_2) measured by pulse oximetry determines the adequacy of oxygen therapy and the need for arterial blood gas (ABG). The aim of oxygen therapy is to maintain SpO_2 94-98%
Blood gases (ABG)	Patients with SpO_2 <92% or other features of life threatening asthma require ABG measurement
Chest X-ray	Chest X-ray is not routinely recommended in the absence of: - suspected pneumomediastinum or pneumothorax - suspected consolidation - life threatening asthma - failure to respond to treatment satisfactorily - requirement for ventilation

Taken from The British Thoracic Society – British Guidelines on the Management of Asthma – Revised June 2009

Fig. 8.2 The British Thoracic Society guidelines for asthma assessment [Reproduced with permission from the British Thoracic Society].

Advanced Life Support Group

MANAGEMENT OF ACUTE ASTHMA IN ADULTS

CRITERIA FOR ADMISSION

B	Admit patients with any feature of a life threatening or near fatal attack
B	Admit patients with any feature of a severe attack persisting after initial treatment
C	Patients whose peak flow is greater than 75% best or predicted one hour after initial treatment may be discharged from ED, unless there are other reasons why admission may be appropriate

TREATMENT OF ACUTE ASTHMA

OXYGEN		β_2 AGONIST BRONCHODILATORS	
C	• Give supplementary oxygen to all hypoxaemic patients with acute asthma to maintain an SpO_2 level of 94-98%. Lack of pulse oximetry should not prevent the use of oxygen.	A	Use high dose inhaled β_2 agonists as first line agents in acute asthma and administer as early as possible. Reserve intravenous β_2 agonists for those patients in whom inhaled therapy cannot be used reliably.
A	• In hospital, ambulance and primary care, nebulised β_2 agonist bronchodilators should be driven by oxygen	☑	In acute asthma with life threatening features the nebulised route (oxygen-driven) is recommended.
C	• The absence of supplemental oxygen should not prevent nebulised therapy being given if indicated	A	In patients with severe asthma that is poorly responsive to an initial bolus dose of β2 agonist, consider continuous nebulisation with an appropriate nebuliser.

STEROID THERAPY		IPRATROPIUM BROMIDE	
A	Give steroids in adequate doses in all cases of acute asthma	B	Add nebulised ipratropium bromide (0.5 mg 4-6 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.
☑	Continue prednisolone 40-50 mg daily for at least five days or until recovery		

OTHER THERAPIES		REFERRAL TO INTENSIVE CARE
B	Consider giving a single dose of IV magnesium sulphate for patients with: • Acute severe asthma who have not had a good initial response to inhaled bronchodilator therapy • Life threatening or near fatal asthma.	Refer any patient: • Requiring ventilatory support • With acute severe or life threatening asthma, failing to respond to therapy, evidenced by: - deteriorating PEF - persisting or worsening hypoxia - hypercapnoea - ABG analysis showing ⇩ pH or ⇧ H^+ - exhaustion, feeble respiration - drowsiness, confusion, altered conscious state - respiratory arrest
☑	IV magnesium sulphate (1.2-2g IV infusion over 20 minutes) should only be used following consultation with senior medical staff	
B	Routine prescription of antibiotics is not indicated for patients with acute asthma.	

Taken from The British Thoracic Society – British Guidelines on the Management of Asthma – Revised June 2009

Fig. 8.3 British Thoracic Society's Guidelines for Treatment of Asthma in hospital [Reproduced with permission from the British Thoracic Society].

Please also see Fig. 8.3 for the assessment and management of patients with acute asthma that is not immediately life-threatening.

Time Out 8.3

Have a five-minute break, then answer the following questions.

a What type of condition is asthma?
b List the major components of this response.
c What is the overall effect of this process?
d How does this affect pulmonary physiology?
e Describe how you would manage life-threatening asthma
f What are the indications for ventilation?

Acute on chronic respiratory failure

This is an important cause of breathlessness and is considered in detail in Chapter 20 on organ failure.

Pulmonary oedema

This is an important cause of breathlessness and is considered in detail in Chapter 20.

Pneumothorax

A pneumothorax results from gas entering the potential space between the visceral pleura and the parietal pleura. This may arise spontaneously from the rupture of a bulla or cyst on the lung surface, or as a consequence of underlying lung disease. However, there are a number of invasive procedures such as subclavian vein cannulation that can also be responsible (see box).

Iatrogenic pneumothoraces

Attempted internal jugular/subclavian vein access
Pleural aspiration/biopsy
Percutaneous lung/liver biopsy
Transbronchial biopsy
Intermittent positive pressure ventilation

Pathophysiology

The outward recoil of the chest and inward elastic retraction of the lung produces negative pressure in the potential space between the visceral pleura and parietal pleura. This pressure, with respect to atmosphere, becomes more negative during inspiration. Following a breach of the visceral pleura, air preferentially moves from the alveolus into the pleural space until these pressures equilibrate – hence the lung collapses, resulting in a simple pneumothorax. If, however, the breach in the pleura acts as a one-way valve then air will preferentially enter the pleural space during inspiration and not return during expiration. Thus the pressure in the intrapleural space rises above atmospheric pressure. The

resulting hypoxaemia acts as a respiratory stimulus causing deeper inspiratory efforts, which in turn further increase the intrapleural pressure. This produces a tension pneumothorax, which can impair venous return. If untreated, mediastinal shift occurs, distorting the great vessels, further impairing venous return, and compressing the opposite lung. This process exacerbates hypoxaemia and eventually causes pulseless electrical activity (electromechanical dissociation).

Key point

Tension pneumothorax is a clinical diagnosis. Needle thoracocentesis is the immediate management

Primary pneumothorax

This condition is relatively uncommon (affecting about 9/100,000 patients with a male to female ratio of approximately 4:1). It occurs in previously normal lungs and is attributed to rupture of a surface bulla or cyst, which is often at the apex. About 20% of patients will have recurrent pneumothoraces on both the ipsilateral and the contralateral sides.

Secondary pneumothorax

This condition is associated with pre-existing lung disease (see next box) and medical procedures (see previous box on page 96).

Pre-existing lung conditions associated with pneumothoraces

Emphysema
Chronic obstructive pulmonary disease
Acute exacerbations of asthma
Infections: **Empyema**
Staphylococcal pneumonia
Tuberculosis
Malignancy
Pulmonary fibrosis
Cystic fibrosis

Assessment

Simple pneumothorax

Symptoms and signs may be absent but commonly the patient will present with breathlessness and pleuritic chest pain localised to the affected side. Breathlessness may be related to pain, size of pneumothorax and pre-existing lung disease.

Key point

In a patient with pre-existing lung disease even a small pneumothorax can produce acute respiratory failure

Clinical signs are difficult to detect when the pneumothorax is small or when there is coexistent emphysema. Often there is reduced chest expansion on the affected side (usually due to pain) and the percussion note is typically resonant. Hyperresonance is very difficult to detect even when comparing with the non-affected side. The most consistent sign is a reduction in breath sounds over the pneumothorax. However, a large bulla can easily be misdiagnosed as a pneumothorax. Review of the previous chest X-rays and CT scans will often solve the dilemma and prevent inappropriate chest drain insertion.

Tension pneumothorax

Presenting complaints for this condition differs depending on whether the patient is spontaneously breathing or is receiving Positive Pressure Ventilation (PPV).

Spontaneous

- More gradual onset
- Presents with mainly respiratory symptoms – tachypnoea, increase in effort, chest pain, tachycardia, falling SpO_2, agitation
- Early – contralateral HYPER mobility and Ipsilateral HYPO mobility
- Late – classic physical findings of condition
- Pre-terminal signs are a low SpO_2, fall in blood pressure, decreasing consciousness

Receiving PPV

- Onset quickly
- Fall in SpO_2 (immediate), blood pressure, tachycardia
- Late – pressure alarm (excessive pressure on inspiration)
- Other physical signs include hyperesonance, hyper-expanded chest, chest hypomobility

Radiological features of a tension pneumothorax

- Splayed ribs
- Depressed hemi-diaphragm
- Mediastinal displacement

Key point

Tension pneumothorax is a clinical diagnosis in the non-ventilated patient.

Management

Simple pneumothorax

Spontaneous resolution will occur in an asymptomatic patient with only partial lung collapse (and no deterioration for 24 h) at approximately 1.25% of the volume of the hemithorax per day. Under these circumstances no intervention will be required. Occasionally pain relief is required with non-steroidal anti-inflammatory drugs. Do not forget to reassure the patient!

The management of pneumothorax, however, depends on the size of the collapse and the presence of underlying lung disease. It is summarised in Figs 8.4 and 8.5.

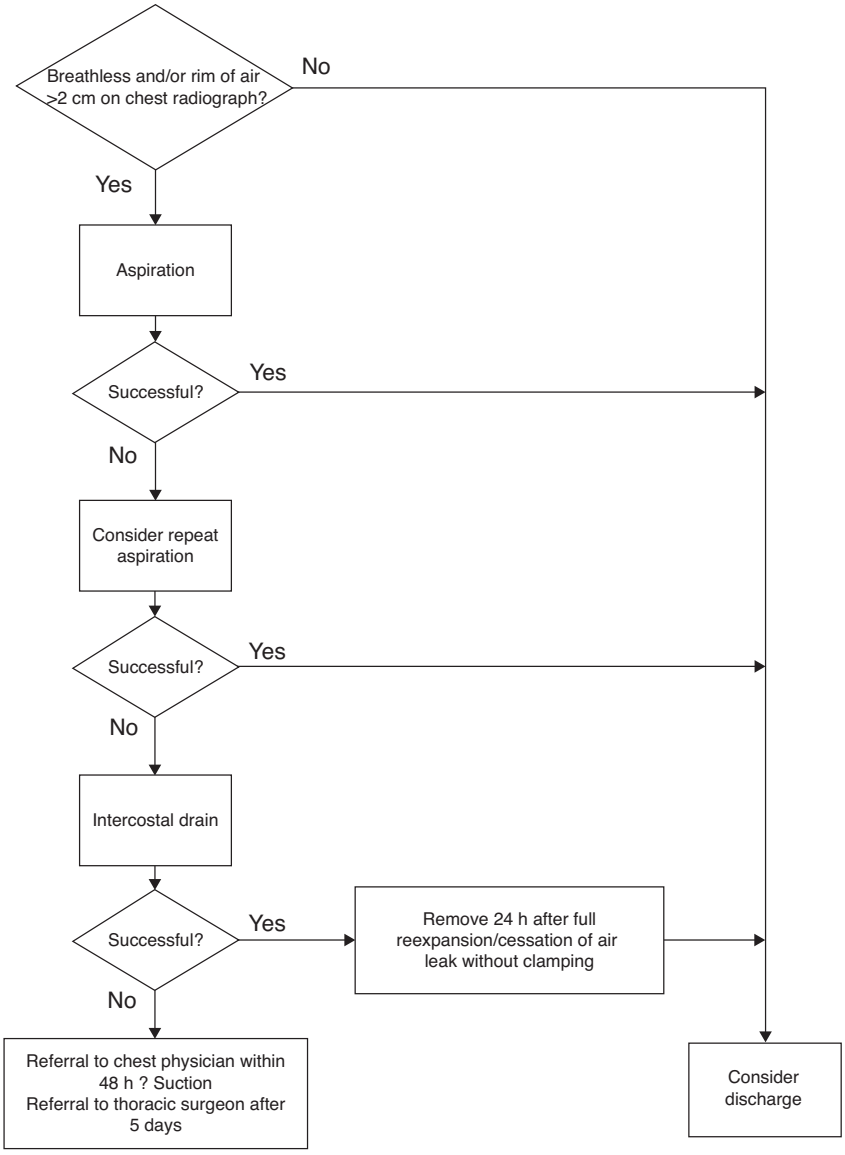

Fig. 8.4 Recommended treatment of a primary pneumothorax.

Aspiration is a simple technique with negligible morbidity. If successful it produces rapid resolution of breathlessness and chest discomfort.

Early drainage and early surgical referral are needed if the patient has either cystic fibrosis or AIDS:

AIDS patients

2–5% of AIDS patients will develop a pneumothorax. Pneumocystis Jiroveci is the most likely cause which produces necrotising alveolitis that impairs healing. This infection leads to a high risk of bilateral and recurrent pneumothorax.

Tension pneumothorax

Immediate needle thoracocentesis is needed. This will relieve the tension and the acute problems, but the residual pneumothorax will need chest drain insertion after securing intravenous access (see Chapter 31). This is a precaution because

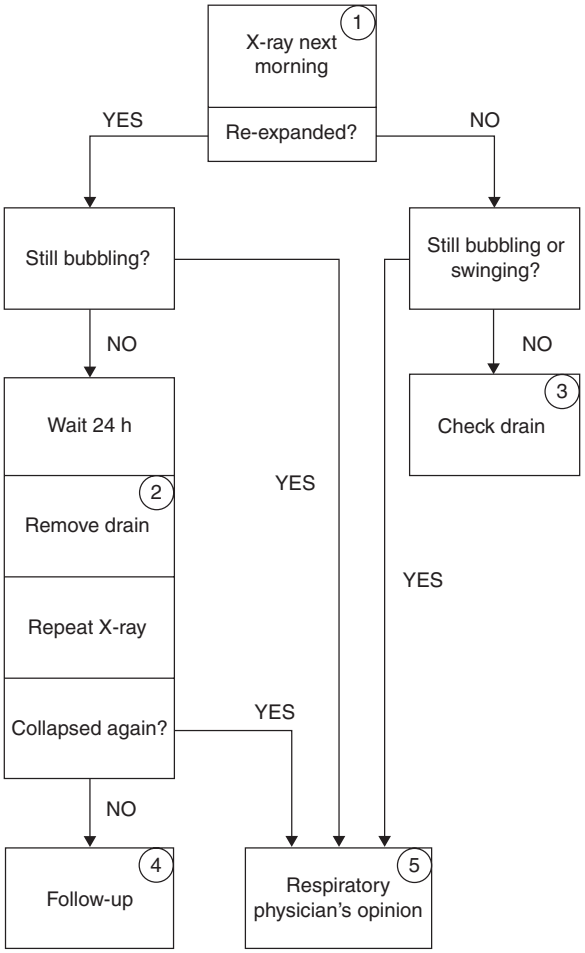

Fig. 8.5 Guidance for removing a chest drain inserted for a pneumothorax.

occasionally a pneumothorax can be complicated by a haemothorax, possibly due to tearing of a pleural lesion or from an adjacent necrotic tumour.

Investigation

Radiological confirmation of a simple pneumothorax is important and will guide appropriate therapy. In contrast, tension pneumothorax is a clinical diagnosis and an X-ray is only needed after chest drain insertion.

Discharge

- Avoid air travel until the pneumothorax has resolved radiologically. Airlines recommend 6 weeks between treatment and flying. If no intervention is necessary then there is a risk of recurrence for up to 1 year. A patient with a secondary pneumothorax without any intervention should therefore avoid flying for up to 1 year.
- Diving should be avoided permanently unless the patient has had a bilateral surgical pleurectomy
- After aspirating a primary pneumothorax – observe the patient to ensure that the signs and symptoms resolve before discharge. After aspiration for a secondary pneumothorax admit the patient for 24 h to ensure that there is no recurrence.

Potential problems

- Lung fails to re-expand after intercostal drain insertion – liaise with respiratory physician regarding use of low pressure suction.
- Recurrent pneumothoraces – liaise with a cardiothoracic surgeon regarding either additional chest drains, chemical endoscopic (VATS – video assisted thorascopic surgery) or formal surgical pleurodesis. Surgical advice should also be sought if there are bilateral pneumothoraces.

Explanatory notes for Fig. 8.5

1 Chest X-ray
If the underwater seal is always kept below the level of the chest, clamping is unnecessary and potentially dangerous. As far as possible, an X-ray film should be taken in the department, rather than on the ward with a portable machine; an expiration film is unnecessary.

2 Removal of chest drain
Bubbling should have stopped for at least 24 h. Some patients find tube removal unpleasant, consider sedation as above. Remove the suture holding the chest drain in place, withdraw the tube while the patient holds their breath in full inspiration. Use the two remaining sutures to seal the wound.

3 Check chest drain
If the lung has not re-inflated and there is no bubbling in the bottle, then the tube is either kinked which can be corrected or blocked. A replacement must be inserted through a clean incision.

4 Follow-up
Arrange for a chest clinic appointment in 7–10 days after discharge. The patient must be given a discharge letter and told to attend again immediately in the event of deterioration. Air travel should be avoided until changes seen on radiographs have resolved.

5 Respiratory physician's opinion
Should advice from a specialist be required, transfer of continuing care is advisable. Important considerations in management are:

- **Assessing why re-expansion has not been achieved (e.g. air leaking around the drain site, tube displaced or blocked, large persistent leak/bronchopleural fistula);**
- **The use of suction to re-expand the lung (this can be lengthy, requires appropriate equipment and pressure settings, influences how and where confirmatory radiographs are taken and involves care from experienced nursing staff);**
- **is a second drain required?**
- **Whether early thoracic surgery would be appropriate (e.g. failure of conservative measures, need to prevent recurrence);**
- **Consideration of chemical pleurodesis in certain cases;**
- **Management of subcutaneous emphysema.**

Time Out 8.4

A 72-year-old lady, who has COPD presents with acute breathlessness. She has a respiratory rate of 28/min, a hyperexpanded chest with scattered wheezes and a

prolonged expiratory phase. Her SpO_2 is 72% on 28% oxygen. What is your immediate management?

Pneumonia

Pneumonia is a general term used to describe a respiratory infection with new chest radiographic shadowing. Traditionally pneumonia has been classified according to its radiological appearance, i.e. lobar, lobular or broncho pneumonia. Unfortunately these do not help in either the diagnosis or the management. In contrast, the circumstances of the illness and the clinical background of the patient, as described in the box, provide helpful clues to aid investigation, management and treatment.

Classification of pneumonia

Community acquired
Hospital acquired (nosocomial)
Aspiration and anaerobic
Recurrent
Immunosuppression associated
Travel related

Management principles – checklist

Diagnosis

- History
- Examination
- Investigations

Chest X-ray	PA
Pulse oximetry	
Arterial blood gases	
Venous blood	Cultures
	Full blood count and film
	Electrolytes, glucose and liver profile
	C-reactive protein
	Initial serology: Mycoplasma, Legionella, Chlamydia
Sputum	Culture and sensitivity
	Microscopy
	Acid and alcohol fast bacilli
Urine	Legionella antigen
	Pneumococcal antigen

Treatment

- Oxygen – unless blood gases are normal
- Antibiotic – choice depends on severity, likely cause, test results and local policies (Fig. 8.6)
- Fluid replacement, either oral or intravenous according to clinical picture
- Analgesia if required
- Consider early liaison with clinical microbiologist, respiratory physician or intensivist.

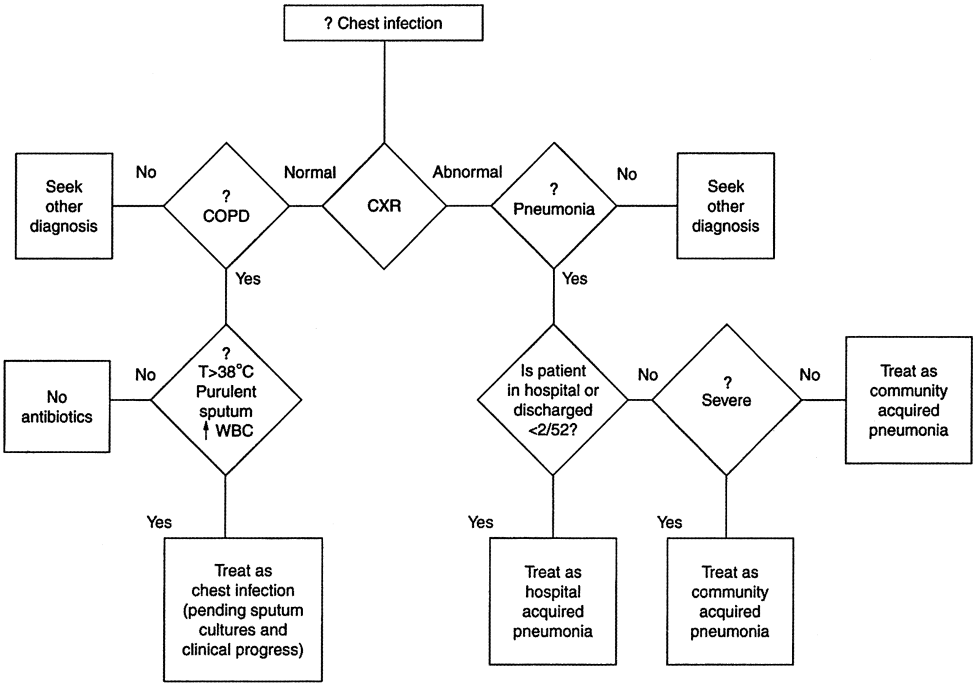

Fig. 8.6 Pneumonia management algorithm.

Key point

Early use of antibiotics in patients with pneumonia reduces morbidity and mortality

Community acquired pneumonia

This is a common cause of acute hospital admission and often occurs in the winter months. Community acquired pneumonia can affect previously healthy individuals or patients with coexisting lung disease. The age of the patient is likely to influence the pathogen involved.

Modes of infection transmission

Extension of bacteria colonising respiratory tract
Droplet, e.g. respiratory viruses from infected individuals
Birds, e.g. *Chlamydia psittaci*
Water droplet, e.g. *Legionella pneumophila*

The organisms likely to cause community acquired pneumonia in the UK are shown in the box.

Organisms causing community acquired pneumonia

Streptococcus pneumoniae
Haemophilus influenzae
Staphylococcus aureus

Influenza virus
Mycoplasma pneumoniae
Chlamydia psittaci
Coxiella burnetti
Legionella species
Moraxella catarrhalis

Streptococcus pneumoniae (pneumococcal pneumonia) is the major pathogen involved, whilst influenza is the commonest viral infection. It is important to realise that viral infections caused by influenza, parainfluenza and respiratory syncytial virus can be associated with superadded bacterial infections.

Assessment
The clinical features are very variable.

Respiratory symptoms

Clinical:	**Cough**	**Prodromal:**	**Pyrexia**
	Sputum production		**Malaise**
	Breathlessness		**Anorexia**
	Pleuritic pain		**Sweating**
	Haemoptysis		**Myalgia**
			Arthralgia
			Headache

On examination most patients appear flushed and unwell with tachypnoea and/or tachycardia. The temperature can exceed 39.5°C and rigors are not uncommon in young people. In contrast, elderly patients may remain afebrile. Herpes simplex labialis is present in approximately one third of patients with pneumococcal pneumonia. Often chest movement is reduced on the affected side, especially if pleuritic pain is present. Inspiratory crackles are the commonest sign and bronchial breathing is infrequent. A pleural rub can be heard even when pleuritic pain is absent. **Occasionally the physical examination is entirely normal;** therefore a chest X-ray is necessary.

It is important to realise that non-respiratory symptoms may predominate; e.g. a patient with lower lobe pneumonia may present with abdominal pain and peritonism. Confusion may be due to hypoxaemia and/or metabolic derangement. In addition, *Legionella pneumophila* is associated with severe headache, cerebellar dysfunction, and amnesia. Vomiting and diarrhoea may occur as a direct manifestation of the illness or related to antibiotic therapy.

Moraxella catarrhalis causes bronchitis or pneumonia in children and adults with underlying chronic lung disease and is occasionally a cause of bacteraemia or meningitis, especially in patients who are immunocompromised.

Unfortunately the mortality from severe pneumonia remains high. Clinical features associated with severe pneumonia are listed in the box and their presence indicates a poor prognosis. Early liaison with an anaesthetist/intensivist is needed if **two** or more are present.

These features have been modified to form the 'CURB 65' score for community acquired pneumonia (Table 8.1). The risk of death from pneumonia ranges from 3% with a score of 1 to 57% with a score of 5.

Table 8.1 The Curb 65 score

Clinical factor	Score
Confusion	1
Urea > 7 mmol/l	1
Respiratory rate > 30/min	1
Systolic blood pressure < 90 or diastolic blood pressure < 60	1
Age > 65 years	1

High risk features in patients with severe pneumonia

Clinical	Investigations
Confusion	Blood urea > 7 mmol/l
Respiratory rate > 30/min	White cell count < 4×10^9 or > 20×10^9
Systolic < 90 mm Hg, diastolic < 60 mm Hg	PaO_2 < 8 kPa (60 mm Hg) (on room air)
Old age and coexistent illness	Serum albumin < 25 g/l
	Multilobe involvement on chest radiograph

Treatment

Patients with scores of 1 or less may not require admission to hospital. Manage all patients in bed; treat fever and pleuritic pain with appropriate non-steroidal anti-inflammatory drugs.

Correction of hypoxaemia and fluid balance is very important as described above. Chest physiotherapy is rarely helpful in the acute phase.

Specific treatment: When the patient presents acutely the microorganism responsible for the pneumonia is not usually known. Therefore, the choice of antibiotic is made according to the limited number of organisms that cause community acquired pneumonia. Most hospitals have devised specific antibiotic policies based on an assessment of the severity of the pneumonia – Fig. 8.6 (page 103). This should be started within 1 h of the diagnosis being made.

- *Severe pneumonia* (CURB 65 $\geq$4). This can affect even previously fit individuals. As the mortality is high parenteral antibiotics must be given immediately. Considering the potential organisms that may be responsible (see earlier) potential treatments include either IV Tazocin (4.5 g tds) or a combination of cefotaxime 1 g tds combined with Clarithromycin 250 mg qds is advised. Rifampicin should be added if legionella is suspected. The duration of intravenous therapy is based on the patient's clinical response.
- *Mild pneumonia* (CURB 65 $\leq$ 1). In most previously fit people the likely organism is *Streptococcus pneumoniae* or occasionally *Mycoplasma pneumoniae*, *Chlamydia* species, *Legionella* species or *Coxiella burnetii*. The combination of amoxycillin and erythromycin is both cheap and effective. Erythromycin alone is appropriate for patients who are allergic to penicillin or if an atypical organism is suspected, e.g. *Mycoplasma, Chlamydia, Legionella*. If the patient fails to respond to this combination, then levofloxacin should be used.

Time Out 8.5

Factual overload? Take a five-minute break and then answer the following questions.

a What is the major pathogen responsible for community acquired pneumonia?
b What are the clinical features?
c How would you manage a patient with severe community acquired pneumonia?
d List the high risk features in patients with severe pneumonia that indicate the need for critical care.

Hospital acquired pneumonia (nosocomial)

This is defined as pneumonia developing more than 48 h after hospital admission, irrespective of the reason.

Pathophysiology

Bacterial colonisation of the nasopharynx changes markedly in hospital patients, particularly those who receive broad spectrum antibiotics and are severely ill. These bacteria arise either from the hospital environment or the patient's gastrointestinal tract. Such pathogens are likely to be aspirated in patients who are ill, bed bound or who have impaired consciousness for whatever reason. This may be exacerbated by an inability to clear bronchial secretions after a general anaesthetic or where coughing is impaired due to thoracic or abdominal surgery. The risk of postoperative pneumonia is associated with increasing age, smoking, obesity, underlying chronic illness and prolonged anaesthesia. Pathogens may also be spread from contaminated equipment such as nebulisers, ventilators, suction equipment and even from the hospital staff.

A large number of pathogens are responsible for hospital acquired pneumonia (see box). Gram negative bacilli are commonly implicated and to a lesser extent, Gram positive bacteria, especially *Staphylococcus aureus*, except if the patient is immunocompromised.

Hospital acquired pneumonia in specific circumstances

Streptococcus pneumoniae	
Haemophilus influenzae	**Early post—elective surgery, especially if chronic chest pathology**
Pseudomonas	
Klebsiella	**Contaminated respiratory equipment**
Pseudomonas	
Klebsiella	
Bacteroides	**Aspiration**
Clostridium	
Legionella	**Contaminated water (cooling towers, heating and showers**

Specific treatment

As there are a wide range of potential organisms responsible for hospital acquired pneumonia, initial therapy is usually Tazocin (Pipparicillin and Tazobactum). If a pseudomonal infection is suspected then an appropriate penicillin derivative such as ticarcillin should be used. Early liaison with a clinical microbiologist is recommended.

Antibiotic therapy can be tailored according to the results of investigations.

Ideally, treatment should be proactive in preventing such infections by scrupulous hygiene practices, appropriate infection control and preoperative advice for the patient.

Time Out 8.6

a Define the term hospital acquired pneumonia.
b List those patients at risk.
c How would you manage a patient with hospital acquired pneumonia?

Aspiration and anaerobic pneumonia

This is commonly associated with impaired consciousness and/or dysphagia. Infection is usually with either *Pseudomonas aeruginosa* or an *Enterobacter* species in the hospital environment. Treatment with Tazocin is advised as true anaerobes do not survive the oxygen rich environment in the lung.

Recurrent pneumonia

If a patient experiences two or more pneumonic episodes, consider the following:

- localised bronchiectasis
- bronchial obstruction, e.g. foreign body, carcinoma or external compression
- a generalised respiratory disorder if the pneumonia recurs in different sites
- COPD with or without bronchiectasis
- aspiration of oesophago-gastric contents in patients with, e.g. motor neurone disease, disseminated sclerosis, achalasia, epilepsy, alcoholism, drug/substance use and oesophagotracheal fistula.
- chronic organising pneumonia (bronchiolitis obliterans organising pneumonia)
- recurrent pulmonary infarction
- immunosuppression/deficiency.

Immunosuppression associated pneumonia

An in-depth discussion on this topic is well beyond the scope of this book. However, the following practical guidelines are suggested.

Pneumonia of acute onset and rapid progression suggests bacterial origin. Therefore, initial treatment should include a combination of cefotaxime and gentamicin to ensure adequate cover against *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Staphylococcus aureus* and many other Gram negative species. If the patient fails to respond or the pace of the illness is less acute then specialist help should be sought early.

Travel related pneumonia

With the increase in worldwide travel, a variety of unexpected respiratory infections may be seen and/or enter into the differential diagnosis of pneumonia. This subject is too extensive to be covered here, but do not forget to:

- take a full:
  - travel history
  - drug history
  - sexual history
- consider bacterial, viral and fungal infections
- consider tuberculosis

- consider an esoteric infection if the patient fails to respond appropriately, but remember that rare manifestations of common infections occur more frequently than common manifestations of rare infections
- liaise early with a consultant in infectious diseases/clinical microbiology/respiratory medicine.

Key point

Antibiotics can cause side effects, and *Clostridium difficile* is a major concern. Thus change from broad spectrum to specific antibiotics as soon as possible

Time Out 8.7

Pneumonia is a common condition. You have already boosted your knowledge by reading this section and answering the associated questions. To complete your understanding review the management of pneumonia as shown in Fig. 8.5

PLEURAL EFFUSION

Pathophysiology

The pleural surfaces are lubricated by a thin layer of fluid that allows the lung and chest wall to move with minimum energy loss. The volume of pleural fluid is a balance between production by the parietal pleura and absorption by the visceral pleura. The increase in hydrostatic pressure within the capillaries of the parietal pleura ensures that fluid passes into the pleural space. Thus, the parietal pleura acts like a plasma ultrafiltrator. In comparison, the pressure within the capillaries of the visceral pleura is lower ensuring that fluid is absorbed. Lymphatic drainage also facilitates removal of fluid and protein from the pleural space.

A pleural effusion results from an excessive accumulation of fluid within the pleural space. Considering the normal production of pleural fluid, as described earlier, the potential factors involved in the production of excess fluid are summarised in the box.

Factors involved in the production of excess pleural fluid

An imbalance between the hydrostatic and oncotic pressures
Alteration in pleural capillary permeability
Impaired lymphatic drainage
Disruption of structural integrity
Transdiaphragmatic passage of fluid

More than one of these factors may be involved in the production of pleural fluid according to the underlying disease process as described below.

Causes of pleural effusion

Some of the reasons why a patient may develop a pleural effusion are listed in the next box. The estimated 'chance' of meeting these causes is given in brackets.

Specific pleural effusions

Transudate

These are characterised by low protein concentrations (<30 g/l). Excess fluid forms when there is an increase in pleural hydrostatic pressure, e.g. in congestive cardiac failure, or when there is a reduction in colloidal osmotic pressure, e.g. with hypoalbuminaemia associated with nephrotic syndrome or liver disease.

Small effusions can be associated with failure of either the left or right or both ventricles.

Elevated left heart pressures will be transmitted to the pulmonary circulation and hence result in reduced fluid absorption from the visceral pleura. In contrast, increased pressure from the right heart is transmitted to the systemic capillaries and this leads to increased production of fluid from the parietal pleura. Resolution occurs with treatment of heart failure, but unilateral effusions that fail to respond to this treatment require further investigation.

Hypoalbuminaemia, as listed earlier, is a major contributory factor in the development of generalised oedema. Thus, both pleural effusions and ascites are common. Formation of pleural fluid is due to a reduction in colloidal osmotic pressure combined with the transdiaphragmatic passage of fluid.

Causes of pleural effusion

(a) Transudate	**Cardiac failure**	**Daily**
Hypoalbuminaemia	**Nephrotic syndrome**	**Weekly**
	Cirrhosis	**Daily**
	Malabsorptions	**Annually**
	Peritoneal dialysis	**Monthly**
	Myxoedema	**Only in exams**
(b) Exudate		
Infective	**Pneumonia**	**Daily**
	Empyema	**Monthly**
	Subphrenic abscess	**Monthly**
	Tuberculosis	**Annually**
Inflammatory	**Pancreatitis**	**Weekly**
	Connective tissue disease	**Monthly**
	Dressler's syndrome	**Annually**
Neoplastic	**Metastatic carcinoma**	**Daily**
	Lymphoma	**Annually**
	Mesothelioma	**Annually**
	Meig's syndrome	**Only in exams**
Haemothorax	**Pulmonary emboli**	**Monthly**
	Trauma	**Monthly**
	Spontaneous bleeding disorders	**Annually**
Chylothorax	**Trauma**	**Annually**
	Carcinoma	**Annually**
	Lymphoma	**Annually**

Key point

In patients with cardiac failure the nature of the pleural effusion can change from transudate to exudate following treatment with diuretics

Exudate

Malignancy The commonest cause of a large pleural effusion is malignant involvement of the pleura. This can occur as:

- direct spread from an adjacent bronchogenic carcinoma
- metastatic spread via the lymphatics, e.g. from breast malignancy
- haematogenous spread from the gastrointestinal tract.

In contrast, primary pleural tumours (mesotheliomas) are rare.

Excess pleural fluid is formed by a combination of mechanisms, including:

- disruption of the integrity of the pleura
- an associated adjacent inflammatory response
- the tumour secreting fluid
- infiltrating malignancy causing haemorrhage
- interference with lymphatic drainage.

Initial treatment is symptomatic and referral to an oncology specialist is recommended.

Connective tissue diseases Pleural involvement is common in patients with systemic lupus erythematosus but to a much lesser extent in those who have rheumatoid disease.

Haemothorax As an acute medical emergency this is rare because most cases occur in association with penetrating or non-penetrating trauma. However, it can occur after:

- attempts at central venous access due to disruption of associated arteries or veins
- secondary to intercostal vessel damage during the course of percutaneous liver or pleural biopsy.

Haemothorax is a rare sequel to bleeding diatheses, overanticoagulation or following a dissection/rupture of the thoracic aorta.

Chylothorax This is rare. It is associated with trauma to, or malignant invasion of, the thoracic duct. This structure can also be damaged during an oesophageal resection or mobilisation of the aortic arch. It is important to differentiate a chylothorax from a pyothorax. A chest drain is the initial management of choice.

Assessment

Symptoms

Pain and breathlessness are the cardinal symptoms of pleural disease. Their presence will, however, vary according to the underlying pathology. Pleuritic pain, which is worse on deep inspiration or coughing, is typical of dry pleurisy. As fluid accumulates, however, the pain spontaneously improves. Breathlessness, as described earlier, only becomes apparent if the pleural effusion is either large or rapidly expanding or there is significant underlying pulmonary pathology.

Signs

Physical signs are often absent unless the effusion is large. Tachypnoea and tachycardia may be present. Chest wall movement and expansion are often reduced on the affected side. The percussion note is 'stoney dull' and both vocal resonance (or tactile vocal fremitus) and breath sounds will be diminished or absent. Above the effusion, however, the lung may collapse with signs of consolidation, i.e. bronchial breathing and increased vocal resonance. Remember that consolidated lung tends to filter out low frequency sounds; thus high pitched bronchial

breathing is prominent. Furthermore, vocal sounds, i.e. '99' or '11' are transmitted by normal lung and, in particular, solid lung but not by air space or fluid.

Investigation of pleural effusion

Radiology

Chest radiographs are of limited value in identifying the cause of a pleural effusion. Ultrasound can help confirm the site and presence of an effusion. It is especially useful when a drain has to be placed into loculated fluid.

Examination of pleural fluid

Macroscopic appearance

- Straw coloured fluid, which does not clot on standing, typifies a transudate.
- Turbid fluid is usually due to the increased protein content, which often reflects an exudate.
- Bloodstained fluid is likely to be associated with an underlying malignancy or pulmonary embolus.
- Chyle is odourless and milky in appearance.
- Empyema fluid is often very viscous, yellow and frequently foul smelling.

Microscopic and cytological examination

- Transudate cell count less than $100/mm^3$
  - often mixed cells, i.e. lymphocytes, neutrophils and mesothelial cells.
- Exudate has high white cell count
  - neutrophil leucocytosis often indicative of bacterial infection
  - lymphocytosis suggests tuberculosis or lymphoma.

The presence of malignant cells is likely to be diagnostic though on occasion the precise cell of origin may be difficult to determine.

Microbiology Fluid should be sent for Gram stain, culture and identification of acid and alcohol-fast bacilli.

Biochemistry Pleural fluid has been described as either a transudate or an exudate based on the protein concentration of less than or greater than 30 g/l, respectively. Unfortunately this is only a rough guide. A better assessment can be obtained by comparing the pleural fluid concentrations of protein and lactate dehydrogenase with those of blood as shown in the box.

Criteria for identifying an exudate

Total protein pleural fluid to serum ratio greater than 0.5
Lactate dehydrogenase concentration in pleural fluid more than two-thirds the upper limit of normal serum LDH
Lactate dehydrogenase pleural fluid to serum ratio greater than 0.6

Other important investigations include:

- glucose – which is consistently low in rheumatoid associated effusions as well as malignancy, empyema and tuberculosis
- amylase – as pancreatitis can result in a pleural effusion which is most frequently on the left
- pleural fluid pH – pH < 7.2 suggests infection and requires tube drainage

Pleural biopsy
This is only indicated when pleural fluid analysis fails to establish a diagnosis and ideally should be done at thoracoscopy, which is usually video assisted.

Specific management

Frequently all that is required, initially, is a sample of pleural fluid for laboratory investigations.

Immediate drainage of pleural fluid is only required if the patient is breathless. This usually occurs if pleural effusion is massive, rapidly accumulating or there is underlying pulmonary disease. The symptoms usually resolve rapidly if 1.5 litres of fluid are aspirated. Chest drains are rarely required, except for empyema, complicated parapneumonic effusions and pleurodesis.

Transudates rarely require direct treatment as they usually resolve with improvement in the underlying condition. In contrast, the management of an **exudate** is governed by the results of investigations. A chest drain may be required for the reasons listed earlier and in the presence of an empyema or pyopneumothorax. The latter should alert the clinician to the presence of either necrotic lung tissue or oesophageal rupture. Antibiotic treatment with intravenous cefotaxime and metronidazole is advocated.

PULMONARY EMBOLISM

Pulmonary embolism is an important condition because it is potentially fatal, often preventable and sometimes treatable. The majority of pulmonary emboli originate in the deep veins of the legs and pelvis. Occasionally, however the right side of the heart can be the source of emboli, e.g. atrial fibrillation, right ventricular infarction or a dilated right ventricle. Major risk factors of pulmonary embolism are shown in the box.

Risk factors for pulmonary embolism and the 'chance' of meeting this factor

Recent surgery	Daily
Immobility for greater than four days	Daily
Age over 40 years	Daily
Previous venous thrombosis/embolism	Daily
Malignant disease	Daily
Lower limb fractures	Daily
Obesity	Daily
Varicose veins	Daily
Pregnancy/oral contraceptive pill	Daily
Nephrotic syndrome*	Weekly
Diabetic ketoacidosis*	Weekly
Resistance to activated protein C*	Annually
Deficiency of antithrombin III*	Annually
Deficiency of protein C and S*	Annually
Prothrombin Gene	Annually
Paroxysmal nocturnal haemoglobinuria*	Only in examinations
Behçet's disease*	Only in examinations

* Risk factors for recurrent thromboembolic disease.

Prothrombin gene mutation

Prothrombin gives rise to thrombin in the coagulation cascade. The mutation leads to an increased amount of thrombin circulating in the blood which leads, by unclear reasons, to a thrombophilic state.

This condition is common in the Caucasian population. About 1–2% of the general population are heterozygous (one copy) for the prothrombin gene mutation.

Risks of the prothrombin gene mutation

Thrombophilic status	Relative risk of venous thrombosis
Normal	1
Oral contraceptive (OCP) use	4
Factor V Leiden, heterozygous	5–7
Factor V Leiden, heterozygous + OCP	30–35
Factor V Leiden, homozygous	80
Factor V Leiden, homozygous + OCP	??? >100
Prothrombin Gene Mutation, heterozygous	3
Prothrombin Gene Mutation, homozygous	??? possible risk of arterial thrombosis
Prothrombin Gene Mutation, heterozygous + OCP	16
Protein C deficiency, heterozygous	7
Protein C deficiency, homozygous	Severe thrombosis at birth
Protein S deficiency, heterozygous	6
Protein S deficiency, homozygous	Severe thrombosis at birth
Antithrombin deficiency, heterozygous	5
Antithrombin deficiency, homozygous	Thought to be lethal prior to birth
Hyperhomocysteinemia	2–4
Hyperhomocysteinemia combined with Factor V Leiden, heterozygous	20

Pathophysiology

One of the normal functions of the lungs is to filter out small blood clots. This process occurs without any symptoms. However, emboli blocking larger branches of the pulmonary artery provoke a rise in pulmonary arterial pressure causing rapid shallow respiration. The rise in pressure is believed to be due, in part, to reflex vasoconstriction via the sympathetic nerve fibres and also hypoxaemia. Tachypnoea is a reflex response to the activation of vagal innervated luminal stretch receptors and interstitial J receptors within the alveolar and capillary network.

Furthermore, vasoactive substances such as 5-hydroxytryptamine and thromboxane released from activated platelets may enhance vasoconstriction and neurotransmission.

Key point

The effect on haemodynamics will be related to the size of the pulmonary embolus

To explain the variations in pathophysiology and management of pulmonary emboli they will be classified as massive, moderate and minor.

Massive pulmonary embolism

This usually follows an acute obstruction of at least 50% of the pulmonary circulation. An embolus in the main pulmonary trunk or at the bifurcation of the pulmonary artery (saddle embolus) can produce circulatory collapse, i.e. pulseless electrical activity (PEA or electromechanical dissociation) and death. However, an identical clinical picture may arise with lesser degrees of obstruction when there has been previous cardiorespiratory dysfunction. An acute massive pulmonary embolus, without immediate death, elicits the haemodynamic response as described earlier. The acute increase in pulmonary vascular resistance and thus right ventricular afterload causes a sudden rise in end diastolic pressure and hence dilatation of the right ventricle. This may be manifest clinically as an elevated jugular venous pressure and tricuspid regurgitation. The dilated right ventricle and rise in pulmonary arterial pressure cause a marked fall in systemic arterial pressure by the following mechanisms.

- A fall in left ventricular stroke volume. Dilatation of the right ventricle and increased pulmonary artery pressure ensures that the right ventricular stroke work is depressed. This results in delayed emptying of the right ventricle, and hence a fall in left ventricular stroke volume.
- Interventricular septum displacement. The dilated right ventricle and associated increased pressure cause displacement of the interventricular septum into the left ventricular cavity (the reverse Bernheim effect) reducing left ventricular volume.

These processes culminate in a fall in systemic stroke volume, which to some extent is offset by the sympathetic mediated increase in systemic (peripheral) vascular resistance. Thus, the patient with a massive pulmonary embolus can present with the features of 'shock' (see Chapter 9).

Moderate pulmonary emboli

Whilst the pathophysiology is identical to that described above, the effect on pulmonary arterial resistance and hence right ventricular function is minimal. The mechanisms underlying breathlessness have already been described. Infarction of the pulmonary parenchyma and associated pleura induces inflammation, both processes culminating in haemoptysis and pleuritic pain.

Minor emboli

These will often go unnoticed but repeated attacks can result in progressive breathlessness, hyperventilation and possibly effort-induced syncope. If this problem remains undiagnosed pulmonary hypertension will develop leading to hypertrophy and subsequently failure of the right ventricle.

Assessment

The different sites of pulmonary emboli are shown in Fig. 8.7.

Differential diagnosis

Acute circulatory collapse is a cardinal feature of massive pulmonary embolism. The differential diagnosis of this shocked state is considered in detail in Chapter 9. However, specific conditions that warrant mention here in the context of acute breathlessness, hypotension, central chest pain and unconsciousness are acute

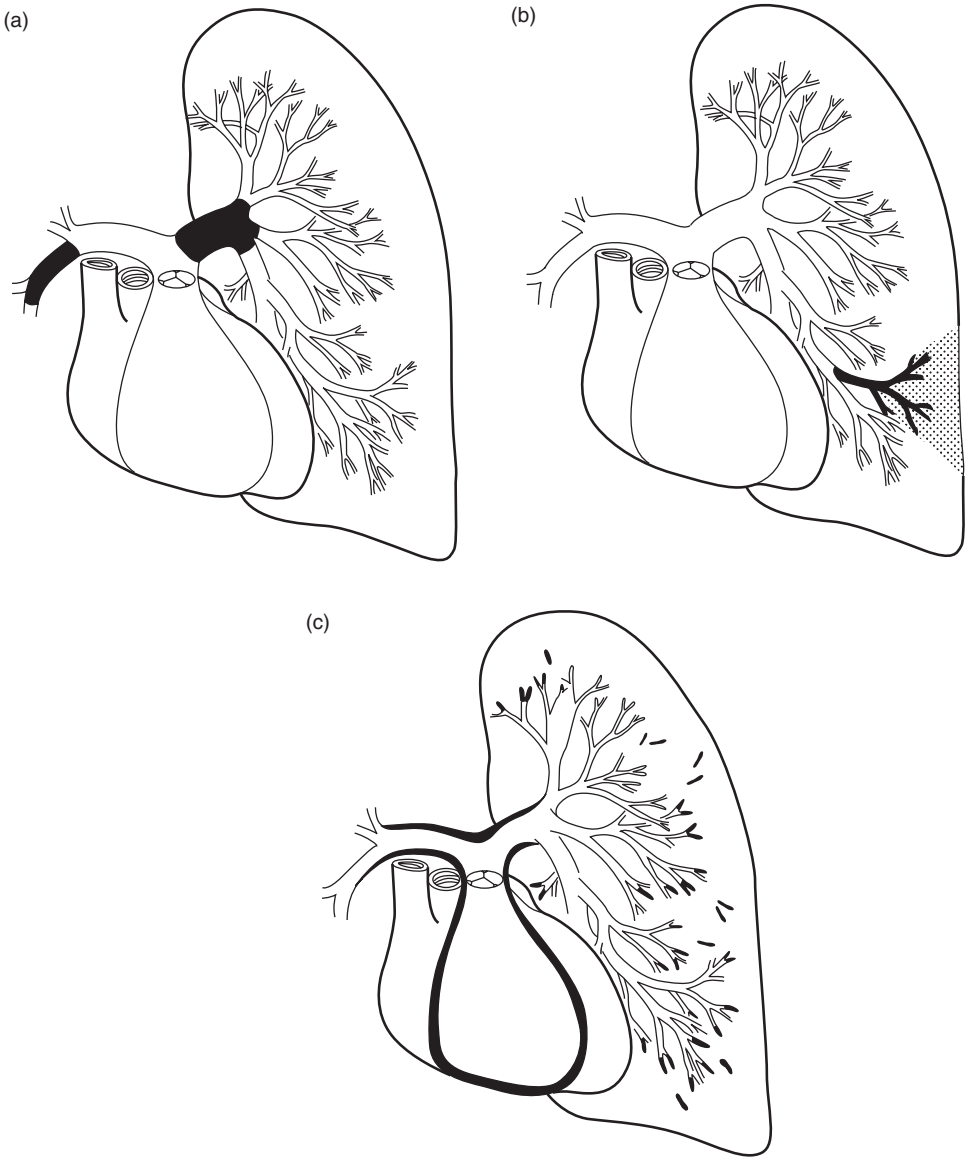

Fig. 8.7 The different sites of pulmonary emboli: (a) main trunk (massive), (b) third division vessels (moderate) and (c) multiple peripheral (minor).

ventricular failure, myocardial infarction and cardiac tamponade. All of these major features are related to diminished cardiac output and hence hypoxaemia and relative hypovolaemia in the acutely ill patient. The differential diagnosis of these conditions can be difficult but there are several key features that can help (Table 8.2).

As with all shocked patients high concentration of inspired oxygen (FiO_2 = 0.85) are required. More comprehensive details regarding the management of the shocked patient are provided in Chapter 9.

Investigations can also help differentiate between these conditions, of which left ventricular failure is the commonest.

Assess the Well's score (page 116) and request D-dimer.

Table 8.2 Key features in the differential diagnosis of breathlessness

Symptoms/signs	PE	LVF	RVF	MI	Tamponade
Breathlessness improves with sitting	No	Yes	No	No	No
Pulmonary oedema	No	Yes	No	No	No
Pulsus paradoxus	Yes	No	No	No	Yes
Raised venous pressure	+++	+	+++	No	+++ (Kussmaul's)
Palpable apex beat	Yes	Yes	Yes	Yes	No
Heart sounds	+ S3/S4	+ S3	+ S3	+ S4	Quiet

PE, pulmonary embolus; LVF, left ventricular failure; RVF, right ventricular failure; MI, myocardial infarction.

Pretest probability of Pulmonary embolus (PE)

Wells score, modified by the Manchester group to include intravenous drug users, provides a pre-test probability:

Table 8.3 Wells score, modified by the Manchester group

Clinical signs of DVT – minimal swelling and pain on palpation of the deep veins	3.0
Other diagnosis less likely	3.0
Intravenous drug user	3.0
HR > 100	1.5
Stasis ≥ 3/7 or operation in <4/52	1.5
History of DVT or PE	1.5
Active cancer or treatment in <6/12	1.0
Haemoptysis	1.0

Low risk 2 or less; moderate risk 2–6; high risk >6.

In pregnancy, confirming the diagnosis can be difficult. Although there is an increased risk of PE the D-dimer is of limited use unless it is negative. Usually, the D-dimer is raised from 6/52 gestation to 3/12 post partum. If a PE is considered obtain a Chest X-ray (less radiation than CT angiography). A perfusion (Q) scan is safe. It is important to balance the risks of delayed treatment (which can be fatal to mother and child) against the risks of the investigations.

This protocol results in <1% missed venous thromboembolic events during follow-up.

Investigations in pregnancy

Objective scoring and D-dimer are less reliable.

- If critically ill arrange a portable echocardiogram otherwise:
  - Chest X-ray to exclude infection and pneumothorax
  - Consider ultrasound Doppler of the legs – no risk to fetus and if positive can treat
  - Consider half dose VQ if normal lungs and Chest X-ray
  - CT pulmonary angiogram if abnormal Chest X-ray or lung disease

  Discuss with mother and specialist

Investigations

- **ECG** changes will occur in approximately 75% of all patients after a massive pulmonary embolus. However, these are often non-specific and T wave inversion in the chest leads (V1 – V3) is the most frequent abnormality. In addition, rhythm disturbances, usually a sinus tachycardia or atrial fibrillation, can occur along with manifestations of acute right heart strain ranging from the classic S1, QIII, TIII pattern to right bundle branch block and voltage criteria of right ventricular hypertrophy. Small complexes, possibly with electrical alternans, can occur in cardiac tamponade.

> **Key point**
>
> **A normal ECG does not exclude either an acute pulmonary embolus or a myocardial infarction**

- **Chest X-ray**. It is usually unhelpful in the diagnosis of acute pulmonary embolus. Occasionally the affected main pulmonary artery may be prominent or there may be loss of lung volume or rarely a 'wedge' shaped defect. However, the chest radiograph will be helpful in the diagnosis of both pulmonary oedema and, to a lesser extent, cardiac tamponade.
- **Arterial blood gases**. Hypoxaemia and acidosis are common after massive or moderate pulmonary emboli. In contrast, a respiratory alkalosis/alkalaemia secondary to hyperventilation is compatible with recurrent small emboli.
- **Plasma D-dimer**. This is a breakdown product of cross-linked fibrin, which is released in thromboembolism. This investigation has become widely used. There are many causes of false positive test results including sepsis. It is generally used to exclude a diagnosis of thromboembolism. The only useful D-dimer is a negative one!
- **Ventilation/perfusion** scans. These can be used to detect pulmonary emboli in patients with normal chest X-rays. They can exclude pulmonary embolism if completely normal but the majority of scans are non-diagnostic. The clinician then has to proceed to further imaging or treat on the basis of clinical suspicion.
- **Echocardiography**. This will show right ventricular abnormalities in 40% of patients and also raised pulmonary arterial pressure.
- **Pulmonary angiography**. This remains the gold standard but very few hospitals have the facilities (Fig. 8.8)
- **Lower limb doppler** studies. They may show a DVT if V/Q scanning is inconclusive.
- **CTPA** (computed tomographic pulmonary angiography). **It is now the recommended imaging modality**.

Specific treatment for acute pulmonary embolism

Most patients with a definitive or suspected diagnosis of pulmonary embolus are treated with a low molecular weight heparin (LMWH) (stops propagation of the embolus/clot and further embolisation from the source thrombus) given subcutaneously. If unfractionated heparin is used, check the activated partial thromboplastin time 6 h after either starting or changing the dose, but be wary of the increased risk of heparin induced thrombocytopenia. Other advantages of LMWH are that there is only one injection per day and there is no loss monitoring unless the patient has renal impairment.

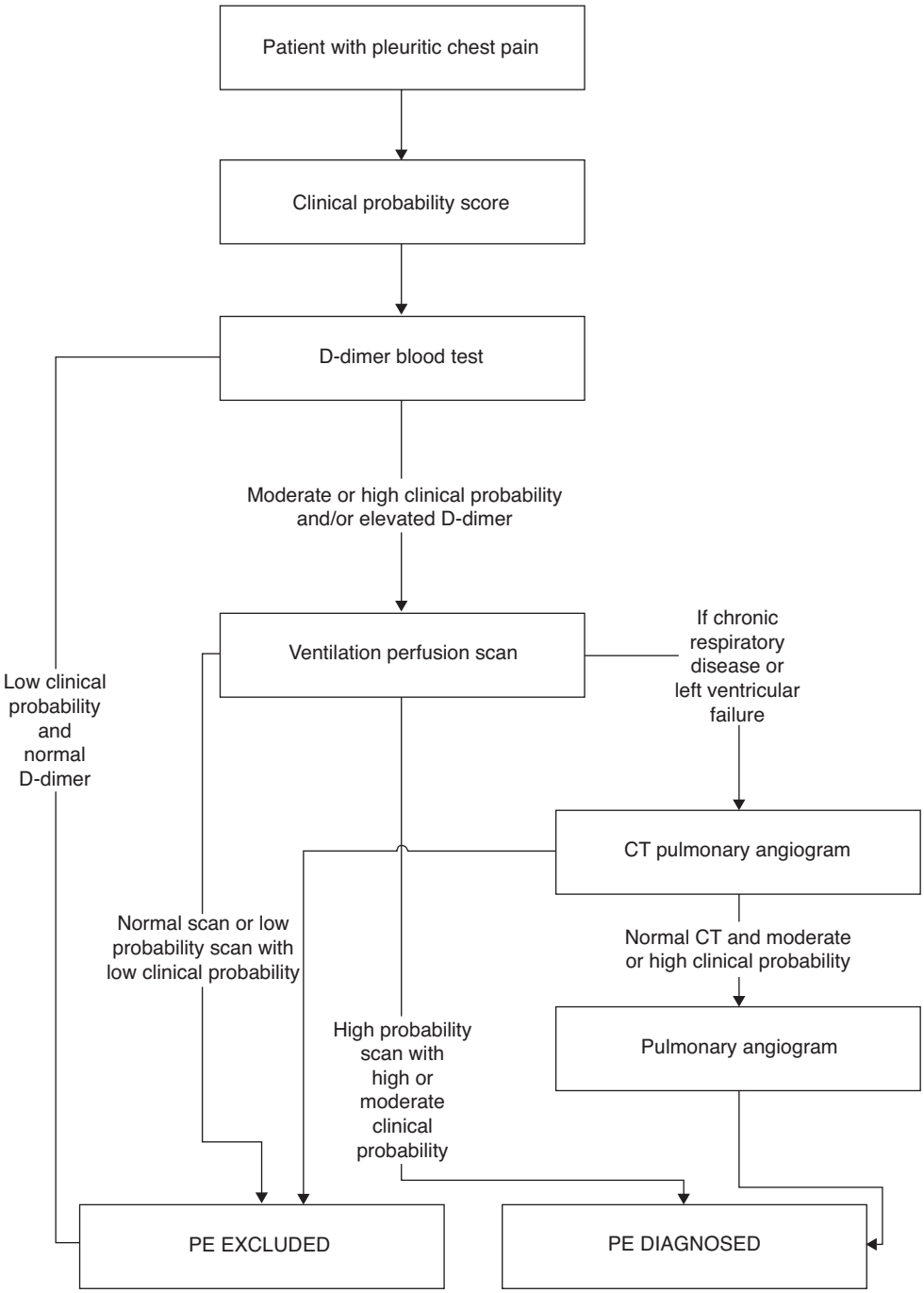

Fig. 8.8 Diagnosis of pulmonary embolism.

Irrespective of the results of investigations, if the clinical suspicion of a pulmonary embolus remains high then the patient should be treated appropriately. If pulseless electrical activity (electromechanical dissociation) results from a massive pulmonary embolus then resuscitation should follow the European and UK guidelines. With the patient who is hypoxaemic and hypotensive, then immediate resuscitation should reduce hypoxaemia and maintain cardiac output. The major decision is medical versus surgical therapy. This will depend primarily on:

- whether the patient has any contraindications to thrombolysis
- local surgical expertise.

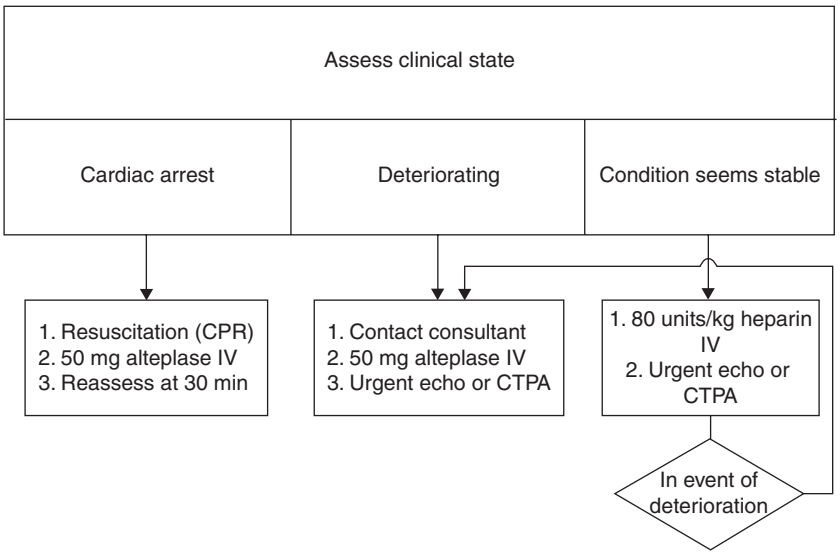

Fig. 8.9 British Thoracic Society protocol for PE thrombolysis.

Comments on Fig. 8.9

In stable patients massive PE is highly likely if:

1 Collapse/hypotension, and
- **Unexplained hypoxaemia and**
- **Engorged neck veins, and**
- **Right ventricular gallop (often)**

2 In patients without haemodynamic compromise where massive PE has been confirmed, IV dose of alteplase is 100 mg in 90 min (i.e. accelerated myocardial infarction regimen)

3 Thrombolysis is followed by unfractionated heparin after 3 h, preferably weight adjusted

4 A few units have facilities for clot fragmentation via pulmonary artery catheter. Elsewhere, contraindications to thrombolysis should be ignored in life-threatening PE (see next box)

5 'Blue light' patients with out-of-hospital cardiac arrest due to PE rarely recover.

Contraindications to thrombolysis are shown in the box.

Contraindications to thrombolysis

Previous haemorrhagic stroke
Any stroke in previous 6 months
Active internal bleeding / active peptic ulceration
Known or suspected aortic dissection
Recent major surgery / head injury / arterial surgery
Pregnancy
Prolonged CPR
Hypertension BP > 180/110
Previous allergic reaction to the thrombolytic agent

Thrombolysis (embolus dissolution), usually in the form of alteplase can be given intravenously as a bolus of 50 mg. This should only be used in a patient who has a massive PE and can be given on clinical grounds alone if cardiac arrest is imminent. Urgent echocardiography, if **immediately** available, can be very useful in confirming your clinical suspicion, by showing a relatively full right ventricle and relatively empty left ventricle). Invasive approaches such as clot fragmentation and 'open heart surgery' can be used if facilities are immediately available.

Time Out 8.8

Take a 5-min break and reflect on the previous section whilst you drink your tea or coffee, and answer the following question.

Why do patients with pulmonary emboli become breathless?

Non-cardiopulmonary causes of breathlessness

The respiratory centre is under the influence of both chemical and neurogenic stimuli. Hypoxaemia, e.g. at high altitude or acidaemia due to diabetic ketoacidosis or salicylate overdose may, therefore, stimulate the respiratory centre in an attempt to provide more oxygen or promote carbon dioxide excretion. Disruption of the integrity of this centre, e.g. by a brain stem haemorrhage, will also result in breathlessness. Other non-pulmonary causes of breathlessness are: any critical illness, shock from any cause especially hypovolaemic, septic or cardiogenic or severe sepsis.

Key point

Be wary of labelling people as 'hysterical hyperventilators' unless underlying pathology has been excluded

SUMMARY

- Breathlessness is a common medical emergency.
- The structured approach will ensure that the immediately life-threatening causes are identified and treated.
- Immediately life-threatening causes of breathlessness are:
  - Airway Obstruction
  - Breathing
    - Acute severe asthma
    - Acute exacerbation of COPD
    - Acute pulmonary oedema
    - Tension pneumothorax
  - Circulation
    - Acute severe left ventricular failure
    - Dysrhythmia
    - Hypovolaemia
    - Pulmonary embolus
    - Cardiac tamponade.
- The pathophysiology of these conditions has been linked to their diagnosis, investigation and management.
- A similar framework has been applied to non-immediately life-threatening conditions, in particular pneumonia and pleural effusion.

Advanced Life Support Group

CHAPTER 9
The patient with shock

OBJECTIVES

After reading this chapter you will be able to understand the:
- definition and causes of shock
- underlying pathophysiology of shock
- importance of oxygen delivery
- structured approach to the shocked patient.

INTRODUCTION

Shock is the result of a series of pathophysiological processes that differ according to the underlying cause. Nevertheless, they culminate in a final common pathway that prevents tissues having enough oxygen to meet their metabolic needs.

Shock can therefore be defined as a clinical syndrome resulting from inadequate delivery, or use, of oxygen by vital organs.

It has many causes, but this chapter will concentrate on those giving rise to inadequate delivery of oxygen to the tissues. The other causes will be mentioned only briefly, but with cross-references to more detailed descriptions elsewhere in this manual.

PATHOPHYSIOLOGY

The amount of oxygen delivered to a particular organ depends on how much blood the heart is pumping out each minute (i.e. the cardiac output) and how much oxygen the blood is carrying:

The oxygen content of blood

Although the majority of oxygen carried in blood is by haemoglobin, we have also mentioned that an additional small volume is physically dissolved in plasma. Therefore, the total volume of oxygen carried in blood at any time is the sum of that carried by the haemoglobin plus that dissolved in the plasma. This total is termed as the **oxygen content**.

The volume of oxygen dissolved in plasma from arterial blood is directly proportional to the PaO_2 and is approximately:

0.003 ml per 100 ml blood per mm Hg PaO_2
or
0.023 ml per 100 ml blood per kPa PaO_2

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It follows that the oxygen content per 100 ml of arterial blood (i.e. the amount associated with the haemoglobin molecule as well as that dissolved in the plasma) is equal to[1]:

(Hb conc × oxygen carrying capacity of Hb × saturation of Hb)	+ (0.003 × PaO_2)
Oxygen content of haemoglobin	**Oxygen content of the plasma**

When the SpO_2 = 100%, each gram of haemoglobin in the arterial system carries 1.34 ml oxygen – a figure known as the **oxygen carrying capacity of haemoglobin.**

Therefore, the oxygen content per 100 ml of arterial blood is:

(Hb conc × 1.34 × saturation of Hb)	+ (0.003 × PaO_2)
Oxygen content of haemoglobin	**Oxygen content of the plasma**

Consider now a normal person who has a PaO_2 of 100 mm Hg with a haemoglobin concentration of 15 g per 100 ml which is 97% saturated with oxygen. In this person, the oxygen content per 100 ml of arterial blood would be equal to:

$$(15 \times 1.34 \times 97\%) + (0.003 \times 100) = 19.5 + 0.3$$
$$= \textbf{19.8 ml oxygen per 100 ml blood}$$

OXYGEN DELIVERY TO THE TISSUES

So far, we have considered only the volume of oxygen in terms of how much oxygen is contained in aliquots of 100 ml of blood, when exposed to varying partial pressures of oxygen. Under normal circumstances, the heart pumps out 5000 ml blood per minute (the cardiac output). Therefore, the total volume of oxygen delivered to the tissues per minute is the product of the cardiac output and the oxygen content:

$$\text{Oxygen delivery } (DO_2) = \text{cardiac output} \times \text{oxygen content}$$
$$\text{In normal circumstances, } DO_2 = 5000 \text{ ml/min} \times 19.8 \text{ ml/100 ml}$$
$$= \textbf{approximately 1000 ml oxygen per minute}$$

Compensatory mechanisms

When a body is under stress it does not immediately fail as several compensatory mechanisms attempt to maintain adequate oxygen delivery to the essential organs.

Oxygen uptake

One mechanism is to increase the respiratory rate in an attempt to take up more oxygen. This is mediated by the sympathetic nervous system. Unfortunately, this does not produce any significant increase in oxygen uptake, because the haemoglobin in blood passing ventilated alveoli is already 97.5% saturated. However, the clinician can help by increasing the inspired concentration of oxygen and ensuring there is adequate ventilation. The slight rise in alveolar oxygen due to the hypocapnia from hyperventilation increases this value by around 1%.

[1] For simplicity, we have just used the values for PaO_2 measured in mm Hg. For those using kPa, you will need to alter the equation in the way indicated above.

Circulatory control

A more effective method of ensuring oxygen delivery to the vital organs is seen with changes in the cardiovascular system. Pressure receptors in the heart and baroreceptors in both the carotid sinus and aortic arch respond to hypovolaemia by triggering a reflex sympathetic response, via control centres in the brain stem. The sympathetic discharge stimulates many tissues in the body, including the adrenal medulla, increasing the release of systemic catecholamines. These combine with the direct sympathetic discharge to prevent, or limit, the fall in cardiac output by positive inotropic and chronotropic effects on the heart and by increasing venous return secondary to venoconstriction.

Furthermore, selective arteriolar and pre-capillary sphincter constriction of non-essential organs (e.g. skin and gut) maintains perfusion of vital organs (e.g. brain and heart). Selective perfusion also leads to a lowering of the hydrostatic pressure in those capillaries serving non-essential organs. This reduces the diffusion of fluid across the capillary membrane into the interstitial space. It also has the effect of increasing the diastolic pressure and thereby reducing the pulse pressure.

Key point

Sympathetic stimulation can give rise to the commonly recognised cardiovascular clinical presentation of patients in advanced shock:
Sweaty and tachycardic – direct sympathetic stimulation
Pale and cool – reduced skin perfusion
Thready pulse – reduced pulse pressure
Ileus – reduced gut perfusion

Any reduction in renal blood flow is detected by the juxtaglomerular apparatus. The resulting increase in renin secretion leads to the formation of angiotensin II and aldosterone. These, together with antidiuretic hormone released from the pituitary, increase the reabsorption of sodium and water by the kidney and reduce urine volume. In addition, the thirst centre of the hypothalamus is stimulated. The overall result is that the circulating volume is increased. Renin, angiotensin II and antidiuretic hormone can also produce generalised vasoconstriction and so help increase the venous return. The body also attempts to enhance the circulating volume by releasing osmotically active substances from the liver. These increase plasma osmotic pressure and so cause interstitial fluid to be drawn into the intravascular space.

Key point

A fall in blood pressure will only take place when no further compensation is possible. It is therefore a late sign in shock. Shock should be identified and resuscitation begun before this point is reached

Autoregulation

Most organs have some capacity to regulate their own blood flow by a process known as autoregulation (see page 59). This enables tissues to compensate for moderate changes in perfusion pressure, by altering local vascular resistance.

However, as the 'shock' state develops, there is paralysis of the smooth muscle in the small blood vessel walls. This allows flow to become pressure dependent and vessels, such as skin arterioles, will begin to distend. This can lead to blood going to non-vital areas at the expense of more clinically important tissues. Autoregulation is further compromised when the vessels become rigid, e.g. with atheroma. As a result, tissue flow will begin to fall at a higher perfusing pressure than normal.

Microcirculatory changes in the late stages of shock lead to stagnation of blood flow, sludging of red cells and a further impairment of tissue perfusion. In addition, the hydrostatic pressure within the capillaries increases, because blood can still perfuse the capillaries but cannot escape. Consequently, further intravascular fluid is lost as it diffuses through the capillary wall into the interstitial space.

Tissue oxygen extraction

At the tissue level, the partial pressure gradient of oxygen is opposite to that found at the alveolar/capillary interface. The capillary PO_2 is approximately 20 mm Hg (2.6 kPa) and cellular PO_2 is only 2–3 mm Hg (<0.4 kPa). Furthermore, local factors also decrease the affinity of haemoglobin for oxygen (shifting the oxygen dissociation curve to the right), allowing O_2 to be released more readily. As mentioned earlier (see Chapter 5), this occurs with an increase in:

- hydrogen ion concentration (i.e. fall in pH)
- $PaCO_2$
- 2,3-DPG
- temperature.

Key point

To help remember these effects, think of the athlete during a race. Active muscles require more oxygen than when they are at rest. With increased metabolism lactic acid, CO_2 and heat are generated. All of these facilitate the release of oxygen from haemoglobin

The total consumption of oxygen per minute (VO_2) for a resting healthy male is 100–160 ml/min/m^2. As the delivery of oxygen (DO_2) is 500–720 ml/min/m^2, the tissues use only 20–25% of the oxygen that is available. This percentage is referred to as the **oxygen extraction ratio**. This low value indicates that body tissues have a tremendous potential to extract more oxygen from the circulating blood.

The total consumption of oxygen per minute is constant throughout a wide range of oxygen delivery in a healthy subject (Fig. 9.1). Under normal circumstances, an increase in oxygen demand is met by increasing the oxygen delivery, usually from a rise in the cardiac output. However, should this not be possible, or inadequate, then VO_2 can be maintained to a limited extent by increasing the oxygen extraction ratio. Should this also fail, then VO_2 will begin to fall, because it is now directly dependent on the delivery of oxygen (Fig. 9.1).

Increasing the oxygen extraction ratio leads to a fall in the venous oxygen saturation. This is difficult to detect clinically early on and its actual value varies from organ to organ. However, it can be measured from a mixed venous sample directly usually by a pulmonary artery catheter (superior venacaval oxygen saturation – $ScVO_2$ is used now as it is easier to obtain and serves similar purpose (normal $ScVO_2$ should be >70%)). Under normal circumstances, this is approximately 75%, with values below 70% indicating that global delivery of oxygen is becoming inadequate.

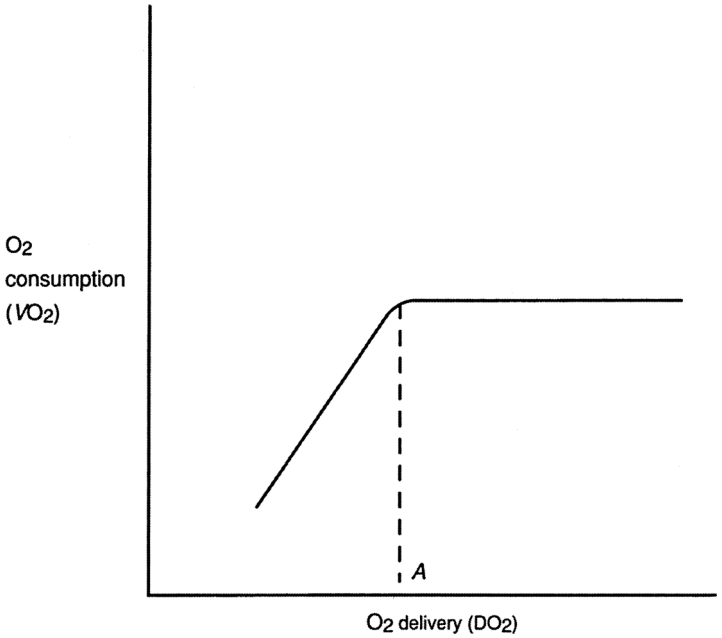

Fig. 9.1 The relationship between oxygen delivery and consumption A is the critical level for DO_2. VO_2 below A depends directly on DO_2.

The amount of anaerobic metabolism increases as the oxygen debt increases. As a result, the plasma lactate level rises in the shocked patient. These levels not only correlate with the severity of the shock, but also allow the body's response to therapy to be assessed.

Resuscitation is completed when tissues return to normal aerobic metabolism and any oxygen debt has been repaid. This is manifested by correction of any metabolic acidosis. Beware that patients may appear adequately resuscitated when simply using clinical vital signs as a measure. Unfortunately, these may not indicate that the body is still trying to compensate for an ongoing lack of tissue oxygenation. If not corrected, this can lead to organ dysfunction and even death in compromised patients. The use of a series of endpoints to measure the shock state, including plasma lactate, is therefore recommended (see later).

Key point

There is a chain of events that delivers oxygen to tissues, where it is used by the cells. Each part has a finite capacity to compensate when one or more links in the chain are defective. When the compensatory capacity is exceeded, shock will develop

Time Out 9.1

a Take a moment to list the five factors that influence the delivery of oxygen to tissues.
b How does the sympathetic nervous system help to compensate for a defect in oxygen delivery?

CAUSES OF SHOCK

Many conditions can lead to an inadequate delivery of oxygen to vital structures of the body (Table 9.1, Fig. 9.2).

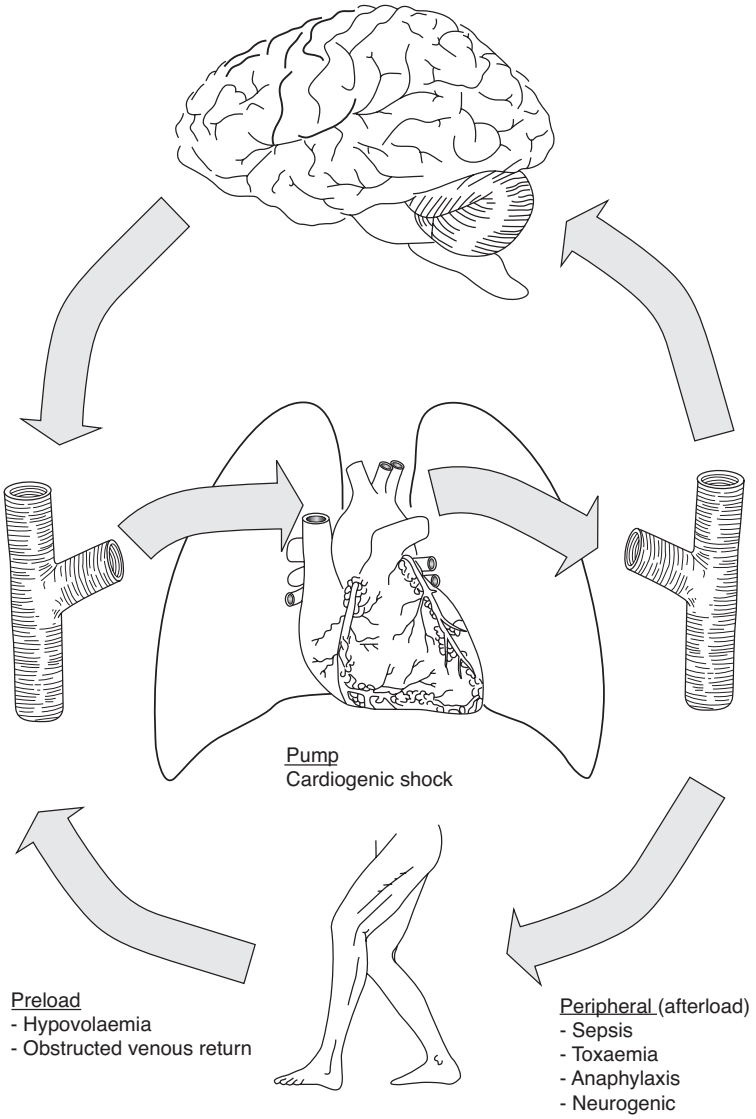

Fig. 9.2 Diagrammatic representations of the causes of shock.

Table 9.1 Mechanisms and causes of inadequate oxygen delivery

Mechanism	Cause
Decrease in oxygen uptake by the lungs	Pneumonia, massive pulmonary embolus, tension pneumothorax, sepsis, anaemia
Reduced venous return	Hypovolaemia, obstructed venous return, sepsis
Impaired cardiac function	Ischaemic heart disease, cardiomyopathy, dysrhythmia, pulmonary emboli, valvular disease (especially severe aortic stenosis and regurgitation), sepsis
Reduced arterial tone	Anaphylaxis, neurogenic shock, sepsis
Impaired organ autoregulation	Sepsis
Decrease oxygen uptake and use by tissues	Poisoning with carbon monoxide, cyanide, sepsis

Decrease in oxygen uptake by the lungs

The airway and pulmonary conditions leading to a fall in oxygen uptake are described in detail in Chapters 5, 8 and 20.

Reduced venous return

Hypovolaemia

True hypovolaemia is associated with loss of either blood or plasma (see next box). Upper gastrointestinal haemorrhage is a common cause in medical patients. In contrast, excessive plasma loss is often seen at the extremes of age with gastroenteritis. However, there may be more than one mechanism involved, e.g. in diabetic ketoacidosis; the fluid loss is related to a combination of hyperventilation, osmotic diuresis, decreased body sodium, vomiting and possibly the precipitating condition.

Causes of hypovolaemic shock

True loss	**Common examples**
Blood loss	Gastrointestinal haemorrhage
	Ruptured aortic aneurysm
Plasma loss	Diarrhoea and vomiting
	Diabetic ketoacidosis
	Osmotic diuresis
	Pancreatitis
	Hyponatraemia and mineralocorticoid deficiency
	Fistula and ostomies
	Burns
Apparent loss	**Common examples**
Venodilators	Nitrates, opiates, intravenous loop diuretics
Hyponatraemia	Glucocorticoid deficiency

Many drugs cause hypotension by reducing preload. Although this effect may be beneficial to patients with left ventricular failure, it can lead to a marked fall in blood pressure, particularly in patients with low blood volumes. The true hypovolaemia and hyponatraemia associated with Addison's adrenal insufficiency are attributed to the deficiency of both mineralo- and gluco-corticoid hormones.

Estimating volume loss and grading shock

The compensatory mechanisms evoked by 'shock' are related to the decline in function of various organs. Respiratory rate, capillary refill (see later), heart rate, blood pressure, urine output and conscious level can be readily measured and so are important indicators of both the grade of shock and the response to treatment. As an approximation, these physiological changes can be used to divide hypovolaemic shock into four categories, depending on the percentage blood loss (Table 9.2). The important features are as given:

- A tachycardia often occurs early due to the sympathetic response.
- In grade II shock, the diastolic blood pressure rises, without any fall in the systolic component, leading to a narrowed pulse pressure. This is due to the compensatory sympathetic nervous system mediated vasoconstriction. Consequently, a narrow pulse pressure with a normal systolic blood pressure is an early sign of shock.

Table 9.2 Categories of hypovolaemic shock

	Category			
	I	II	III	IV
Blood loss (litres)	<0.75	0.75–1.5	1.5–2.0	>2.0
Blood loss (%BV)*	<15%	15–30%	30–40%	>40%
Respiratory rate	14–20	20–30	30–40	>35 or low
Heart rate	<100	>100	>120	140 or low
Systolic BP	Normal	Normal	Decreased	Decreased ++
Diastolic BP	Normal	Raised	Decreased	Decreased ++
Pulse pressure	Normal	Decreased	Decreased	Decreased
Capillary refill	Normal	Delayed	Delayed	Delayed
Skin	Normal	Pale	Pale	Pale/cold
Urine output (ml/h)	>30	20–30	5–15	Negligible
Mental state	Normal	Anxious	Anxious/confused	Confused/drowsy

*% B/V = % of blood volume.

- Tachypnoea can indicate shock as well as underlying respiratory or metabolic pathology.
- Hypotension indicates a loss of at least 30% of the circulating volume.

Limitations to estimations of hypovolaemia

Blindly following the signs in Table 9.2 will lead to gross errors in blood loss estimation – particularly in certain patient types (see box). It is therefore important that management is based on the overall condition of the patient and not isolated physiological parameters.

Pitfalls in assessing blood loss

Elderly
Drugs
Pacemaker
Athlete
Pregnancy
Hypothermia
Compensation
Tissue damage

The **elderly patient** is less able to compensate for acute hypovolaemia as their sympathetic drive is reduced. Consequently the loss of smaller volumes can produce a fall in blood pressure. Reliance only on the blood pressure could therefore lead to an overestimation of blood loss (see Chapter 21).

A variety of **drugs** that are commonly taken can alter the physiological response to blood loss. For example, β blockers will prevent tachycardia and also inhibit the normal sympathetic positive inotropic response. Therefore, after a 15% circulating volume loss, compensatory tachycardia is unlikely to occur in a β-blocked patient. This could lead to an underestimation of the blood loss. It is also

important to remember that the blood pressure falls at lower volumes of blood loss in these patients, by the same mechanisms.

An increasing number of patients have **pacemakers** fitted each year. These devices may only allow the heart to beat at a particular rate, irrespective of the volume loss or sympathetic drive. Therefore they will give rise to the same errors in estimation as β blockers.

The physiological response to training will mean that the **athlete** will have a larger blood volume and a resting bradycardia (about 50 beats/min). The blood volume can increase by 15–20%; thus, it is possible to underestimate blood loss, especially as a compensatory increase in heart rate can mean that the pulse is less than 100 beats/min.

During **pregnancy**, the heart rate progressively increases so that by the third trimester it is 15–20 beats faster than normal. Blood pressure falls by 5–15 mm Hg in the second trimester and returns to normal during the third, as the blood volume increases by 40–50%. Supine hypotension, due to uterine compression of the inferior vena cava, has been discussed earlier (also see Chapter 23).

Hypothermia will reduce the respiratory rate, pulse and blood pressure irrespective of any other cause of shock. Depending on the temperature, hypothermic patients are often resistant to cardiovascular drugs, cardioversion or fluid replacement. The estimation of the fluid requirements of these patients can therefore be very difficult and often invasive monitoring is required (see Chapter 21).

Delays in resuscitation, especially in the young increases the action of the normal **compensatory mechanisms**. This can lead to improvements in respiratory rate, heart rate and blood pressure, thus the clinician may possibly underestimate the volume of blood lost.

The degree of **tissue damage** can have a profound effect on the patient's physiological response. The initial tachycardia can deteriorate into a bradycardia when there is a significant haemorrhage with little tissue damage (e.g. a gastrointestinal bleed). At this stage, the blood pressure also begins to fall. Conversely, when there is marked tissue damage even with a significant haemorrhage, the blood pressure and tachycardia are maintained. Consequently, the degree of blood loss can be over- or underestimated depending on the absence or presence of significant tissue damage.

Obstructed venous return

Blood returning to the heart depends on the pressure gradient created by the high hydrostatic pressure in the peripheral veins and low hydrostatic pressure in the right atrium of the heart. Any reduction in this gradient, e.g. by increasing right atrial pressure, will lead to a fall in venous return to the heart. External compression on the thorax or abdomen can have a similar action in obstructing the venous return. Consequently in the supine position, the gravid uterus can compress the inferior vena cava and impair venous return.

The common causes of obstructed venous return, reducing preload, are shown in the box.

Causes of obstructed venous return

High mean airway pressure (e.g. high PPV)	Daily
Acute asthma	Daily
Pregnancy	Weekly
Massive pulmonary embolus, tension pneumothorax	Monthly
Cardiac tamponade	Annually

Impaired cardiac function

A variety of conditions can adversely influence ventricular function and lead to shock (see next box).

It is important to remember that antiarrhythmic drugs may have a significant negative inotropic effect. The same effect is seen with certain drugs taken as an overdose, e.g. tricyclic antidepressants. Myocardial function can also be impaired by infection (myocarditis), an underlying cardiomyopathy or toxins associated with the systemic inflammatory response syndrome (see later). Cardiac tamponade, in addition to its effect on venous return, impedes ventricular filling.

Cardiogenic shock occurs when around 40% or more of the left ventricle has infarcted. Clinically, it is defined as Class IV on the Killip classification – i.e. a systolic blood pressure of 90 mm Hg or lower, peripheral vasoconstriction, oliguria and pulmonary vascular congestion.

Summary of the cardiac causes of shock

Myocardial	Ventricular failure/ conduction problems	Ischaemia/infarction
		Myocarditis
		Drugs
		Toxins
		Cardiomyopathy
Endocardial	Acute valve lesion	Infective endocarditis
		Papillary muscle rupture
		Aortic root dissection
Epicardial	Acute tamponade	Ventricular wall rupture
		Malignancy
		Post-surgery
	Constrictive pericarditis	Viral
		Tuberculosis
		Radiotherapy

Key point

In cardiogenic shock, the compensatory sympathetic and catecholamine response, i.e. increased heart rate and systemic vascular resistance, only serve to raise the myocardial oxygen demand and exacerbate the degree of myocardial ischaemia

Reduced arterial tone

Anaphylactic shock

Anaphylaxis is an acute reaction to a foreign substance to which the patient has already been sensitised. This leads to an IgE-triggered rapid degranulation of mast cells and basophils (see box). Anaphylactoid reactions have an identical clinical presentation but are not triggered by IgE and do not necessarily require previous exposure. Furthermore, they may not produce a reaction every time.

Common causes of anaphylaxis/anaphylactoid reactions

Anaphylaxis	**Drugs (protein and non-protein) – commonly penicillin or other β lactam drugs, blood products and immunoglobulins**
	Vaccines
	Food – especially nuts, shellfish
	Venoms – especially bees, wasps and hornets
	Parasites
	Chemicals
	Latex
Anaphylactoid	**Complement activation**
	Coagulation/fibrinolysis system activation
	Direct pharmacological release of mediators
	Exercise induced
	Radiological contrast
	Idiopathic

Key point

The most common causes of anaphylactic fatalities are parenteral drugs, bee stings and food-related reactions. Radio-contrast and non-steroidal anti-inflammatory medications are the most common anaphylactoid fatalities

The body's response to these stimuli is to release a collection of mediators from mast cells and basophils that have inflammatory, spasmogenic and chemotactic actions. The inflammatory activators induce vasodilatation and oedema. This leads to a reduction in tissue perfusion as a result of the fall in arterial tone and venous return. The spasmogens cause bronchial smooth muscle contraction, increased mucus production and mucosal oedema. The chemotactic agents attract platelets and white blood cells to the affected area.

In addition, the variety of chemical mediators released cause cardiovascular collapse from one, or more, of the following:

- arrhythmia
- hypovolaemia
- decreased myocardial function
- pulmonary hypertension.

Arrhythmias may result from direct mediator effects, as well as hypoxaemia, hypotension, acidosis, pre-existing cardiac disease and adrenaline given during resuscitation. Hypovolaemia can occur very quickly, with up to 50% of the circulating plasma volume being lost within 10–15 min in severe cases. This is due to a combination of increased vascular permeability, vasodilatation and decreased venous return from raised intrathoracic pressure secondary to bronchospasm and positive pressure ventilation.

The vast majority of serious anaphylactic reactions occur unexpectedly. Over 50% of fatalities occur within the first hour. Seventy five percent of these deaths are due to asphyxia from upper airway obstruction or bronchospasm. The remaining cases die from circulatory failure and hypotension.

Key point

The diagnosis is not difficult when a patient presents with generalised urticaria, wheeze and hypotension following a known stimulus. However, circulatory collapse can occur without preceding warning signs

SEPTICAEMIA, SEPSIS, SEVERE SEPSIS, SEPTIC SHOCK

Septicaemia is an ambiguous term, does not explain the various syndromes associated with infection or the body's response to infection and should, therefore, not be used in clinical practice. This term is also often confused with ***bacteraemia***, which refers to the presence or detection of bacteria in blood where they normally do not exist.

Sepsis, caused by invading microorganisms, is a well-known clinical entity, usually known by its common name: infection. Generally, such patients are not systemically severely ill. Confusion is caused by either not recognising systemic illness consequent upon severe infections or erroneously diagnosing severe sepsis where widespread systemic inflammatory response does not accompany infective illness. There are internationally recognised definitions of sepsis, severe sepsis and septic shock. Sepsis must be distinguished from severe sepsis and septic shock, which in turn must also be distinguished from systemic inflammatory response syndrome as the therapeutic priorities are different in different conditions.

Systemic inflammatory response syndrome (SIRS) is the systemic response to severe inflammation (e.g. pancreatitis) or major trauma. Two or more of the following criteria define SIRS when such inflammation is present:

- respiratory rate of $\geq$20 breaths/min or $PaCO_2$ of $\leq$ 4.25 kPa
- heart rate $\geq$90 beats/min
- core temperature $\geq$38.0°C or $\leq$36.0°C
- white cell count of $\geq$12,000/mm^3 or $\leq$4,000/mm^3.

Sepsis is defined as evidence of SIRS with a known or suspected source of infection. In clinical practice, it is sometimes difficult to distinguish between SIRS and sepsis. It is, therefore, very important that a diligent search is made for the source of infection in these patients. Aggressive treatment with antibiotics and/or drainage (radiological, endocopic, surgical) of the source will help to prevent patients slipping into severe sepsis and/or septic shock and progress to multiple organ dysfunction syndrome (MODS), thus reducing mortality.

Severe sepsis is sepsis accompanied by hypoperfusion and organ dysfunction. Any one of the following organ dysfunctions may be present:

- Cardiovascular: Systolic blood pressure $\leq$90 mm Hg or mean arterial pressure $\leq$70 mm Hg for at least 1 h despite **adequate** volume resuscitation
- Renal: Urine output $\leq$0.5 ml/kg body weight/h (despite **adequate** fluid loading) or acute renal failure (now called *acute renal injury*)
- Pulmonary: PaO_2/FiO_2 ratio of $\leq$33.3–26.5 (kPa) depending on the presence or absence of other organ dysfunction (Normal PaO_2/FiO_2 ratio is $\approx$63.4 for a healthy adult breathing room air.)
- CNS: Acute alteration in mental status, e.g. delirium or a Glasgow Coma Score $<$ 14
- Haematological: Platelet count of $\leq$80,000/mm or a decrease of 50% over 3 days or disseminated intravascular coagulation

- Gastrointestinal: Paralytic ileus, delayed gastric emptying, abnormal liver function tests
- Metabolic: pH $\leq$7.30 or a base deficit $>$5.0 mmol/l and/or a plasma lactate $>$1.5 times the upper limit of normal (usually 2.8–3.0 mmol/l).

Septic shock is severe sepsis with persistent hypoperfusion, despite aggressive and adequate fluid resuscitation. This is usually, but not always, manifested as hypotension. The hypotension of septic shock always requires vasopressors (e.g. norepinephrine) and/or inotropes (dopamine, dobutamine, epinephrine depending on the patients' condition, disease severity and comorbidities).

Mortality increases proportionately from sepsis to severe sepsis to septic shock. Many conditions may mimic sepsis and, in an emergency, any of the following conditions may be mistaken for sepsis:

- acute myocardial infarction
- acute pulmonary embolism
- acute pancreatitis
- fat embolism syndrome
- acute adrenal insufficiency
- acute decompensation of chronic liver disease; mild infections in patients with liver disease
- acute gastrointestinal haemorrhage
- 'overzealous' diuresis or unnecessary and inappropriate use of diuretics
- adverse drug reactions
- transfusion reactions
- procedure-related transient bacteraemia (e.g. urethral instrumentation, removal of an infected central line)
- amniotic fluid embolism
- last stages of malignant diseases, carcinomatosis and mild infections in patients with advanced malignancy.

With sepsis, the circulating endotoxins (inflammatory mediators such as prostaglandins, cytokines and nitric oxide) have a negative inotropic effect, cause vasodilatation and impair energy use at a cellular level; the source is usually Gram negative bacteria. Occasionally, Gram positive bacteria release toxins causing the **toxic shock syndrome**. *Staphylococcus aureus* is the usual organism, although some severe streptococcal infections can have a similar presentation. Much less commonly in the UK, viruses, fungi and protozoa are the sources of the sepsis.

Causes of toxic shock syndrome

Retained tampon
Abscess
Empyema
Surgical wound infection
Osteomyelitis
Cellulitis
Infected burns
Septic abortion

Eventually, septic shock will affect all parts of the circulatory system. Venous return is reduced as pro-inflammatory cytokines increase capillary permeability. Further cellular damage by endotoxins causes the release of proteolytic enzymes. These paralyse precapillary sphincters, enhance capillary leakage and increase

hypovolaemia. The resultant loss of fluid and protein causes hypovolaemia which, combined with venodilatation, produces a fall in preload. The reduction in tissue blood flow resulting from decreased perfusion, and the increased viscosity, leads to platelet aggregation and clot formation. At the same time, thromboplastins are activated. Consequently, disseminated intravascular coagulation can result and lead to further falls in tissue perfusion.

Myocardial depression occurs, especially in severe sepsis. This is due to multiple factors including hypoxaemia, acidosis, myocardial oedema and circulating negative inotropes. Tissue autoregulation is disrupted and there is marked peripheral arterial dilatation. Arteriovenous shunts also develop, resulting in maldistribution of blood flow. This either increases the chances of, or exacerbates, tissue ischaemia. In addition to all of these changes, tissue oxygen demand increases, but uptake is impaired. It will not, therefore, be surprising to find that septic shock has an extremely high mortality rate (>50%).

The diagnosis of septic shock can be difficult. In contrast with other causes of shock (except anaphylactic), the physiological features are usually (but not always) high cardiac output and low systemic vascular resistance (Table 9.3). The classic signs are a wide pulse pressure and warm skin (due to the dilated peripheral vessels), agitation, pyrexia and an increased respiratory rate (due to hypoxaemia). The classic features of hypovolaemic shock are manifested later, with peripheral vasoconstriction and a low or normal core temperature. There may also be evidence of disseminated intravascular coagulation. This abnormality often manifests as blood oozing around wounds and cannula sites.

Table 9.3 Haemodynamic variables in shock (adult mean values)

	Left atrial pressure (mm Hg)	Cardiac output (l/min)	Systemic vascular resistance (dyn/s/cm^2)
Normal	10	5	1200
Left ventricular failure	25	2	3000
Haemorrhage	0	3	3000
Sepsis and anaphylaxis	2	12	300

Key points

Maintain a high index of suspicion, because diagnosing septic shock can be difficult

Always check for the non-blanching purpuric rash of meningococcal septicaemia

Consider the diagnosis in any ill patient with an altered conscious level and haemodynamic abnormalities

As described before, the type of septic shock known as the toxic shock syndrome has many potential causes. However, the clinical presentation remains the same:

- temperature 39°C or above
- macular, blanching rash
- hypotension
- evidence of involvement of at least three systems.

The rash can be localised or general and tends to lead to desquamation after one or two weeks in survivors. Common systems that are involved are gastrointestinal

(diarrhoea and vomiting); neurological (confusion, drowsiness); renal (impaired function); muscle (myalgia, high creatine phosphokinase); haematological (leucocytosis, disseminated intravascular coagulation, thrombocytopenia).

Multiorgan dysfunction

Organ dysfunction of more than one organ is called multiple organ dysfunction syndrome (MODS; previously called multiple organ failure). Severe sepsis and septic shock are the commonest causes of MODS in the critically ill.

Neurogenic shock

Neurogenic shock is caused by disruption of the sympathetic nervous system outflow following spinal injures above T6. The higher the lesion, the greater the impairment and the more marked the effect.

In the context of acute medical emergencies, neurogenic shock is rare. Patients who are susceptible to spontaneous cervical vertebral subluxation include those with:

- rheumatoid disease
- Down's syndrome
- ankylosing spondylitis (an inflexible cervical spine that fractures following minimal trauma).

The lack of sympathetic activity results in generalised vasodilatation, bradycardia, loss of temperature control and lack of both the reflex tachycardia and vasoconstriction responses to hypovolaemia. As neurogenic shock leads to a reduction in blood supply to the spinal column, it also gives rise to additional nervous tissue damage.

Clinically, the patient with a high spinal lesion often has a systolic blood pressure of approximately 90 mm Hg, with a heart rate of around 50/min. In addition, the patient has warm and pink skin due to vasodilatation. However, due to an initial pressor response releasing catecholamines into the circulation, the onset of these signs can take from a few minutes to 24 h to develop.

During the initial neurological assessment using the AVPU scale or Glasgow Coma Scale, an asymmetrical weakness may become apparent by a lack of response to peripheral stimulation. These should be noted and a definitive neurological examination performed in the secondary assessment (see Chapter 7). However, these are difficult in the unconscious patient. If in doubt, immobilise the cervical spine and request a neurosurgical/orthopaedic review. Appropriate imaging can then be chosen and interpreted.

Key points

Be wary of the unconscious patient who is admitted following a fall downstairs. The initial neurological features are often falsely attributed to an underlying stroke

Spinal immobilisation must be maintained until specialist advice is obtained if a spinal injury is suspected, from either the mechanism of the injury or the physical signs

PRIMARY ASSESSMENT AND RESUSCITATION

Patients cannot remain permanently in a state of shock; they either improve or die. Shock could be viewed as a momentary pause on the way to death. Its detection depends on certain physical signs that are produced as a result of poor

oxygen delivery. Thus, the treatment of shock necessitates restoring adequate delivery of oxygen, and not simply restoring a normal blood pressure.

Airway

The first priority in any shocked patient is to clear and, if necessary, secure the airway so that high concentrations of inspired oxygen can be given (see Chapter 4). The patient should also be attached to a pulse oximeter and, if intubated, a capnograph.

Breathing

Once the airway has been cleared, adequate ventilation with a high inspired oxygen concentration is required. This is often difficult (e.g. in patients with active haematemesis); therefore, early liaison with an anaesthetist is necessary. Record the respiratory rate and examine for signs of bronchospasm, pulmonary oedema, and tension pneumothorax, and treat as appropriate.

Circulation

Look at the patient noting colour, sweating and distress. Assess the height and character of the jugular venous pulse. Then **feel** the arterial pulse for either a brady or a tachycardia. Is the patient vasodilated with a bounding pulse? Then check the position and character of the apex beat if it is palpable. Finish by **listening** for the presence of extra heart sounds and/or heart murmurs.

Connect the patient to an ECG and blood pressure monitor. Obtain peripheral intravenous access with the largest cannula possible (ideally a 14 or 16 gauge) and take 20 ml of blood for laboratory tests. These include full blood count, urea and electrolytes, glucose, lactate and an arterial blood gas sample. If clinically appropriate, blood should also be taken for cross-match, markers of myocardial damage, amylase, blood cultures and toxicology.

Plasma lactate is a useful measure, particularly in hypovolaemia where it is related to the degree of hypovolaemic shock and risk of death. The time to normalise the plasma lactate level is also a predictor of survival.

Key point

All shocked patients will have a metabolic acidosis. It should be treated by correcting any A, B and C problems and NOT by giving sodium bicarbonate

If a peripheral site is not available in adults, central venous access is advocated. This procedure should **only** be done by experienced staff, because of the potential for damaging the vein and neighbouring structures. Ultrasound guidance is increasingly being used to locate the central veins and facilitate cannulation.

By the end of this assessment, the answers to the following questions should have been ascertained:

- Is shock present?
- If present, what is its likely cause?

Further information from a well-'phrased' history will help in deciding the answers to these questions.

SPECIFIC TYPES OF SHOCK

Hypovolaemia

In the majority of 'medical patients' with hypovolaemic shock, the primary aim is to restore fluid loss from, e.g. vomiting and diarrhoea. Occasionally, however, the primary aim of treatment is to prevent further bleeding if at all possible. Examples of this include the use of a Sengstaken tube for a variceal bleed (see cautionary comments below) or urgent surgery for a ruptured ectopic pregnancy. Often there is no definite source for blood or fluid loss. In these cases, the clinician should devise a management plan based on the likely cause of fluid loss, or the bleeding source, the degree of hypovolaemia and the patients pre-existing medical condition.

General

In grade I shock, a litre of crystalloid is infused and the response monitored. If hypovolaemia is estimated to be grade II or higher, 500 ml intravenous colloid, or another litre of crystalloid, is required. The aim should be to maintain the haematocrit (packed cell volume) at 30–35%, so that oxygen delivery is optimised. Red cell replacement is secondary, becoming more important with progressively larger blood losses.

All fluids need to be warmed before they are given to patients to prevent iatrogenically induced hypothermia. A simple way of achieving this is to store a supply of crystalloids and colloids in a warming cupboard. However, if this method is used, care must be taken to push the fluids rapidly through a wide-bore short cannula (Poiseuille's law) to prevent the fluids cooling down in the giving set. This eliminates the need for warming coils, which increase resistance to flow and thereby slow the rate of fluid administration. 'Level One' rapid fluid infusors, if available, allow rapid infusion of large volumes of warm fluid.

The above management should be modified in hypotensive patients where there is a definite bleeding source that has not been controlled. In these cases, vigorous fluid resuscitation will lead to further bleeding and a worse prognosis. These patients require the source of the bleeding controlled urgently. In the meantime fluid needs to be administered so that the blood pressure is maintained at 20 mm Hg below the baseline. This is known as **hypotensive resuscitation**.

Specific

The source of bleeding in the acutely ill medical patient is often the upper gastrointestinal tract and, as a group, accounts for 1–2% of medical admissions. The specific causes are listed in the box.

Upper gastrointestinal haemorrrhage: causes and frequency

34%	Duodenal ulcer
19%	Gastric ulcer
15%	No lesion identified
11%	Oesophagitis
8%	Gastroduodenitis
5%	Malignancy (upper gastrointestinal tract)
4%	Varices
4%	Others

In addition to the general management principles described earlier, the clinician should ensure **early** liaison with surgical colleagues. Immediately after resuscitation has started, inform the surgical gastroenterology team of the clinical problem and request a review. Combined medical and surgical management is the ideal. The decision to operate is usually based on continuing haemorrhage and the patient's transfusion needs.

Need for surgery with upper gastrointestinal tract bleed

Six units of blood in patients aged less than 65 years, unless there is a history of non-steroidal anti-inflammatory drugs (NSAIDs) use or comorbid pathology
Four units of blood in patients greater than 65 years of age or those less than 65 years with a history of NSAID use or comorbid pathology

If oesophageal varices are suspected, based on the presence of chronic liver disease stigmata or from the history, give terlipressin (2 mg bolus IV over 1 min, then 20 mg/kg (1 mg) every 4–8 h, max 120 μg/kg/day). Early liaison with a gastroenterologist is important for endoscopic intervention. Tamponade devices are rarely required and should only be introduced by an appropriately trained individual.

Key point

Bleeding from varices is rare when compared with gastroduodenal inflammation/ulceration, even in alcohol users

Obstructed venous return

Severe bronchospasm and a gravid uterus are common causes of obstructed venous return. Please read pages 90 and 372.

Other causes include cardiac tamponade where the symptoms (see Chapter 6) can be transiently improved by a fluid challenge to assist ventricular filling pressures. If, however, this presents as a pulseless electrical activity, then resuscitation according to the UK and European protocol is required, including pericardiocentesis. Ideally, echocardiography should be used to facilitate pericardiocentesis. If the equipment/skill is unavailable and the patient is deteriorating, then drainage of the pericardium should be done blindly, using ECG control, to gain time, allowing the subsequent insertion of a pericardial drain under more controlled conditions.

Both pulmonary emboli and tension pneumothorax are described in detail in Chapter 8.

Impaired cardiac function

Shock resulting from heart failure is common. The signs are described in detail in Chapter 20 and summarised in the box. When the signs of shock are resulting solely from myocardial damage, there is an 80% mortality. There are often other, more treatable, causes adding to the shock state. It is therefore essential that hypovolaemia, vasovagal reactions, arrhythmias and drug reactions are identified and treated.

Signs of cardiogenic shock

Breathlessness and central cyanosis
Fine bi-basal crackles
Tachycardia
Hypotension
Raised JVP
Third heart sound
Murmurs, e.g. mitral valve regurgitation (due to ventricular dilatation)

The first management priority is to correct hypoxaemia. The use of non-invasive ventilation (CPAP or NIPPV) is increasing. However, there is still debate on which is the optimum method of non-invasive ventilation for these patients. Occasionally, the patient may require intubation so that optimal oxygenation can be achieved. The high cardiac filling pressures also need to be reduced in a controlled fashion. Intravenous nitrates are often used, but with caution, because of the risk of aggravating hypotension, as they lower the systemic vascular resistance. Dopamine and dobutamine may also be required to provide inotropic support and improve the cardiac output. Any dysrhythmia causing haemodynamic compromise must also be treated.

When cardiogenic shock is due to right heart failure, give a fluid challenge of 200 ml of colloid and assess the effect, and repeat according to the clinical response.

It is not unusual to find that a combination of mechanical ventilation, vasodilators, inotropes and fluids is required to increase the cardiac index and the delivery of oxygen. Clearly, these procedures require the facilities available in either coronary care or high dependency or intensive treatment units.

Patients with heart failure are less able to compensate for hypovolaemia, should that coexist. This problem is compounded by the fact that measurement of the central venous pressure (CVP) does not provide an accurate estimate of the left ventricular end diastolic pressure (see the box below). These patients are therefore best managed using a pulmonary artery catheter. This enables both the filling pressure of the left side of the heart and the cardiac output to be estimated and accurate fluid resuscitation provided. Many non-invasive haemodynamic monitoring devices are now also available but need specialist training to use them (as does the pulmonary artery catheter).

CVP monitoring in heart failure

Measures right ventricular pressure affected by:
- **Intravascular volume**
- **Intrathoracic pressure**
- **Right ventricular function**
- **Venous tone**

As a result, the CVP can be raised in:
- **Pulmonary pathology**
- **Positive pressure ventilation**
- **Malposition causing false elevations**

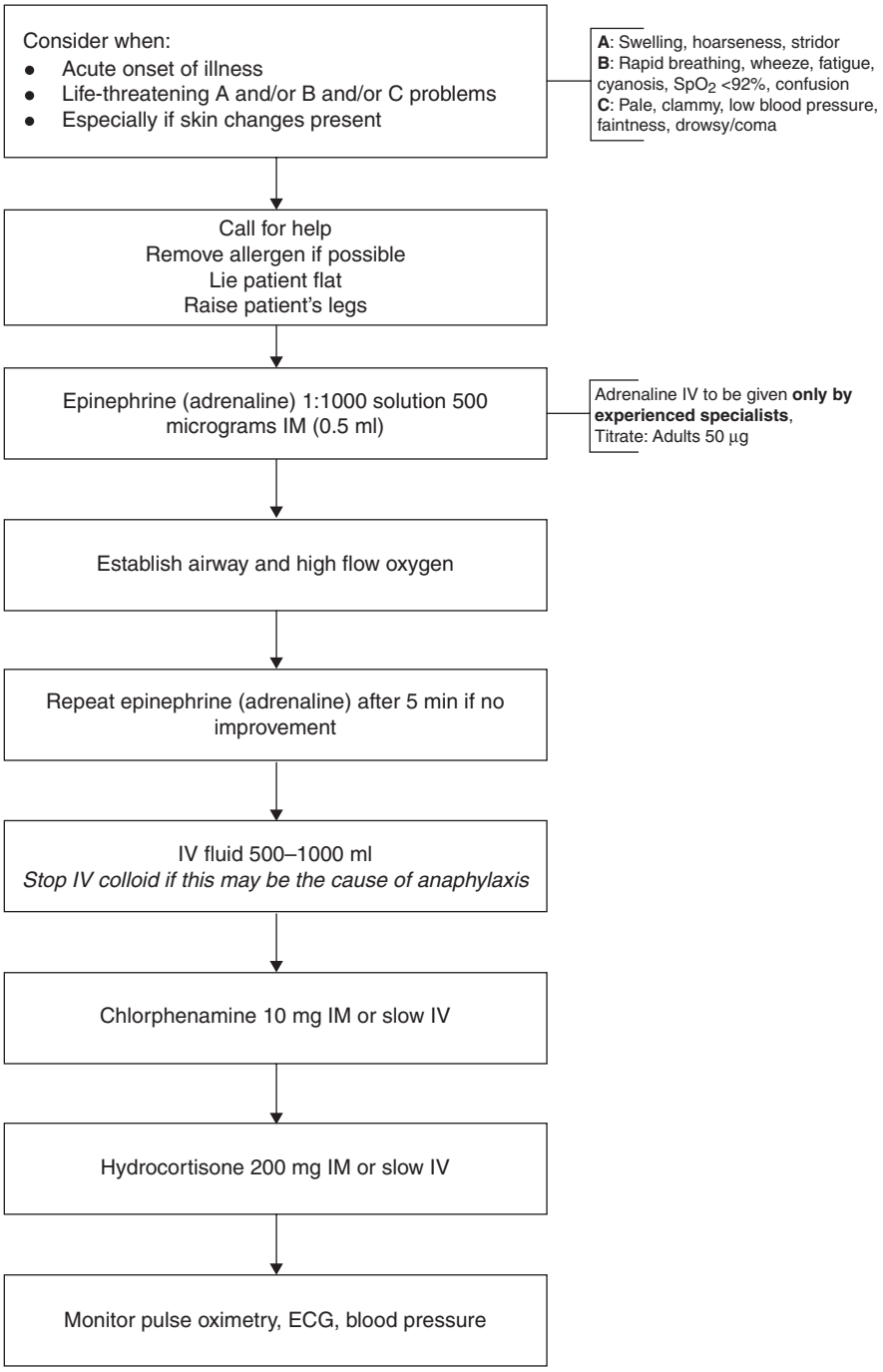

Fig. 9.3 Management of anaphylactic shock (RCUK guidelines 2008) [Reproduced with permission from the Resuscitation Council (UK)].

Anaphylactic shock

The management of anaphylactic shock is dependent on a rapid ABC assessment and resuscitation, considering the diagnosis and preventing any further absorption of the suspected causative agent (Fig. 9.3). Always be wary because airway obstruction, bronchospasm and hypotension can have a delayed, but ultimately sudden, presentation.

If profound shock is judged immediately life-threatening, give cardiopulmonary resuscitation/advanced life support as necessary. Consider slow intravenous (IV) adrenaline (epinephrine) 1:10,000 (i.e. very dilute) solution. This is hazardous

and is recommended only for an experienced practitioner who can also obtain IV access without delay. Note the different strength of adrenaline (epinephrine) that is required for IV use. A crystalloid may be safer than colloid.

Following resuscitation, the patient should be admitted for 8–12 h of monitoring to detect those cases that develop a protracted or biphasic response. The latter is more likely following oral antigen ingestion, or when symptoms started over 30 min after exposure.

Septic shock

Therapeutic priorities are different in different 'stages' of sepsis. Hypotension constitutes a medical emergency. This is a sign of advanced decompensation and will lead to rapid worsening of tissue hypoxaemia, organ dysfunction, organ failure and increase in mortality. A first priority in severe sepsis and/or septic shock, therefore, is to improve tissue oxygenation by supportive measures. These include high concentrations of inspired oxygen, aggressive fluid resuscitation with balanced salt solutions (Hartmann's preferred to 0.9% saline), vasopressors/inotropes as necessary and assessment and management according to the familiar steps of ABC. Some of the supportive measures may not be available in general medical or surgical wards. However, as stated above, severe sepsis and septic shock are emergencies and the resuscitative measures should be started immediately while an appropriate place (a bed in HDU, ICU) for the patient's further care is identified. On no account should the patient be left alone.

Once resuscitative measures have been started, appropriate help should be sought, e.g. an intensivist's opinion either a radiologists or, a surgical opinion for percutaneous or surgically accessible sources of infection or help from microbiologists etc. Initially, after taking appropriate cultures, broad spectrum antibiotics (including those for gram negative bacteria, e.g. aminoglycosides; cover for anaerobes is usually not required) may be started empirically; these should later be changed when microbiological culture results are available. If meningitis is suspected, antibiotics should not be withheld or delayed because appropriate cultures (e.g. cerebrospinal fluid) have not been taken. Blood cultures can usually be taken when gaining venous access.

If septic patients are to survive, the source of infection needs to be identified, treated and removed. When there is a collection of pus, drainage will be required by either surgery or percutaneously under imaging control.

Repeated blood cultures may be required to determine the causative organism. In the meantime, antibiotic therapy should be aimed at the most likely organism. Often a combination of a penicillin, aminoglycoside and metronidazole is used according to the hospital antibiotic policy. If meningococcal septicaemia is suspected, give benzyl penicillin 2.4 g and ceftriaxone 2 g intravenously **immediately**.

The patient will require cardiovascular and respiratory support, as well as intensive monitoring of their fluid and antibiotic regimes. The former aims to maintain a high cardiac index (over 4.5 l/min/m^2), high oxygen delivery (above 600 ml/min/m^2) and tissue perfusion pressure. This usually entails intubating and ventilating the patient with supplemental oxygen, correction of hypovolaemia and the use of inotropes. The response to all vasoactive drugs is unpredictable. It is therefore advisable to start with a low dose and titrate further amounts until the cardiac index is sufficient to allow acceptable tissue perfusion. In adults, this is usually at a level greater than 4.5 l/min/m^2.

The indications for ventilation are no different from those routinely used:

- inability to maintain an airway
- inability to maintain normal PaO_2 and $PaCO_2$

- persistant tachypnoea despite adequate oxygenation and volume replacement
- persistant metabolic acidaemia
- elevated serum lactate.

Noradrenaline is frequently needed for its α-agonist activity that helps counteract some of the profound vasodilatation.

Clinical objectives in treating sepsis

Maximise oxygenation
Improve haemodynamic function
Correct any metabolic derangement
Remove source

These patients are very ill. It should not be assumed that starting resuscitation and antibiotics is the end of their care. Many of these patients deteriorate despite best therapy and care. They need constant monitoring and evaluation. Many will need multiple organ support. These patients behave differently from 'standard' patients on the wards and their needs and priorities are different. They are best cared for in critical care units where their needs will be adequately met. Mortality is high and early recognition and aggressive therapy cannot be overemphasised. Although the survivors of MODS may recover from their critical illness and be discharged from ICU and finally from hospital, their physical and psychological recovery is often slow and prolonged and may take up to 18–24 months after an episode of severe sepsis and MODS (Fig. 9.4).

SSC 'treatment bundles'

Sepsis resuscitation bundle (to be started immediately and completed within 6 h)

Serum lactate measured
Blood cultures obtained prior to antibiotic administration
From the time of presentation, broad-spectrum antibiotics administered within 3 h for admissions to the emergency department and within 1 h for non-emergency department admissions to the intensive care unit (ICU).
In the event of hypotension and/or lactate levels >4 mmol/l (36 mg/dl):
deliver an initial minimum of 20 ml/kg crystalloid (or colloid equivalent)
give vasopressors for hypotension not responding to initial fluid resuscitation to maintain mean arterial pressure ≥65 mm Hg
In the event of persistent arterial hypotension despite volume resuscitation (septic shock) and/or initial lactate >4 mmol/l (36 mg/dl):
achieve central venous pressure of ≥8 mm Hg
achieve central venous oxygen saturation ≥70%*

Sepsis management bundle (to be started immediately and completed within 24 h)

Low-dose steroids administered for septic shock in accordance with a standard ICU policy
Drotrecogin alfa (activated) administered in accordance with a standard ICU policy

Glucose control maintained ≥lower limit of normal but <150 mg/dl (8.3 mmol/l)
For mechanically ventilated patients, inspiratory plateau pressures maintained <30 cm H_2O

*Achieving a mixed venous oxygen saturation of 65% is an acceptable alternative.

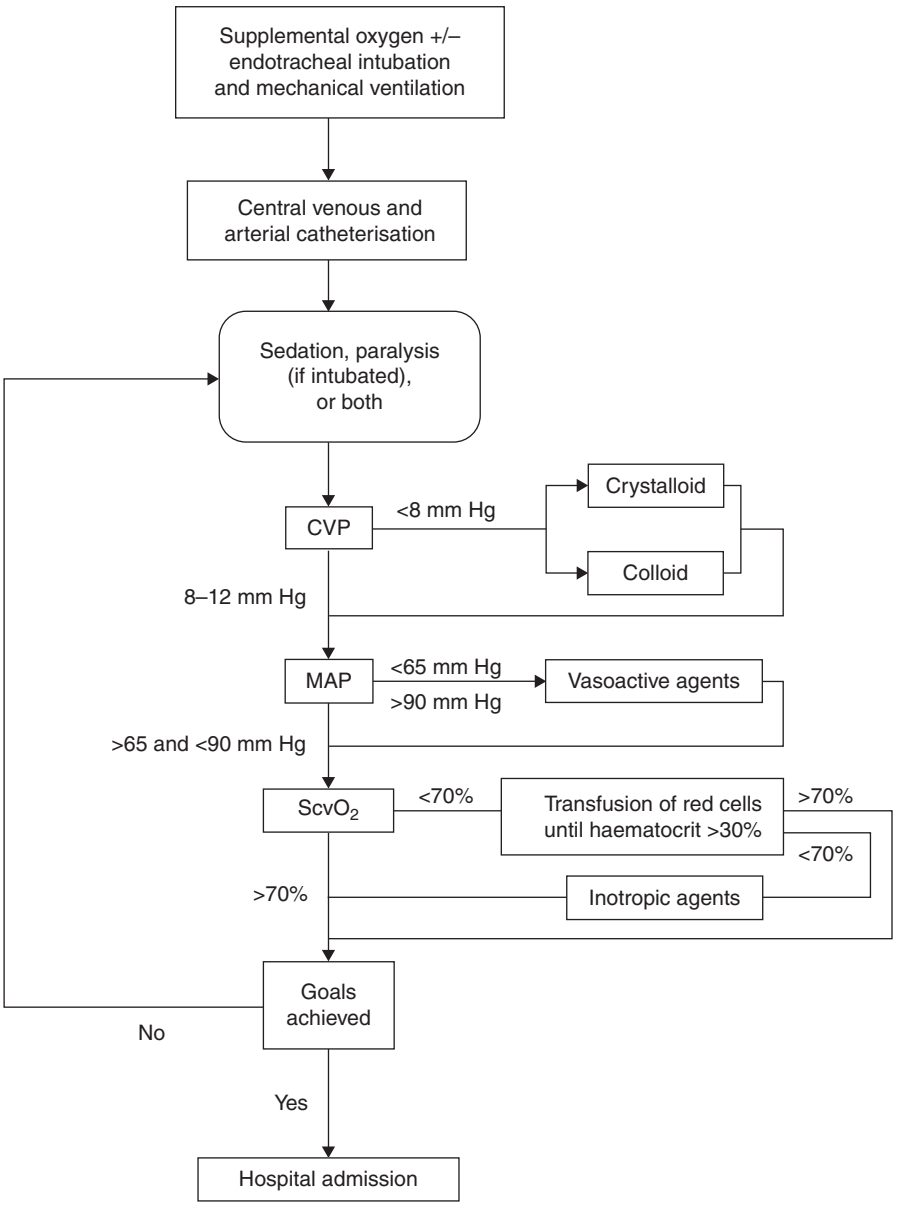

Fig. 9.4 Management of septic shock.

Neurogenic shock

These patients usually require intubation, as the risks of regurgitation and aspiration are increased due to paralytic ileus, a full stomach and an incompetent gastro-oesophageal sphincter.

As close to 100% oxygen as is possible should be given, not least because the damaged spinal cord is very sensitive to hypoxaemia. Always maintain in-line cervical spine immobilisation, by an assistant holding the head during intubation, or by the use of commercially available apparatus.

Be aware that the lack of sympathetic tone decreases the patient's compensatory response to other types of shock. It also enhances the vagal effect produced by stimulation of the pharynx, e.g. during laryngoscopy. This can lead to profound bradycardia, requiring treatment with glycopyrrolate. Atropine can be used, but it produces dry, thick secretions that increase the lung dysfunction. Finally, remember to keep the patient covered by warm sheets and blankets. This not only avoids embarrassment but also prevents heat loss from vasodilatation that occurs after high spinal injuries.

Key point

Intubation is not contraindicated in the presence of cervical spine instability, as long as in-line immobilisation is maintained

Persistent signs or symptoms of shock must not be attributed to the presence of spinal cord injury, particularly if there is evidence of haemorrhage or trauma. Identification and control of any bleeding source is equally relevant in cases of spinal injury, because of the risks of hypoperfusion of the spinal cord. In the presence of an isolated spinal cord injury, a systolic blood pressure of 80–90 mm Hg is initially acceptable and usually achieved with a fluid challenge of 0.5–1 litre. Patients with a bradycardia of less than 50 beats/min should be given atropine 0.5–1 mg intravenously, repeated as necessary until the heart rate is acceptable. If this fails, inotropes and/or pacing may be required. It is important that these patients are neither under- nor overtransfused. The former leads to further spinal injury, the latter leads to pulmonary oedema. A central line is an important way of monitoring the patient's condition.

Early insertion of an arterial line is necessary to provide continuous, accurate blood pressure recordings as well as facilitating repeated arterial blood gas sampling. High doses of methyl prednisolone in the first 24 h after blunt spinal injury are beneficial (see box below). The reason for this improvement is not known, but workers have postulated that it could be due to a decrease in lipid peroxidation, protein degradation, catabolic activity or an increase in impulse conduction by activation of ion pumps.

The early use of methyl prednisolone following blunt spinal injury

30 mg/kg IV over 15 min immediately
Then 5.4 mg/kg/h for 23 h

MONITORING AND ONGOING CARE

The shocked patient's vital signs should be continuously monitored (see next box). Be aware, however, that despite this monitoring it is still possible to miss ongoing tissue hypoxaemia. Consequently, a number of other devices of varying complexity and invasiveness are available (pulmonary capillary wedge

pressure, gastric tonometry, right ventricular diastolic volume index, subcutaneous and muscle oxygenation). However, the risks and practical problems associated with using some of these devices need to be weighed against their potential benefits.

In the shocked patient, coexistent pathology can be present and for those with ischaemic heart disease, the increase in cardiac work and oxygen demand may be critical. Often these patients will require more invasive monitoring and the care of at least a high dependency environment.

Monitored vital signs in hypovolaemic patients

Respiratory rate
Peripheral oxygen saturation
Heart rate
Blood pressure
Pulse pressure
Capillary refill
Chest leads (ECG rhythm and wave form)
Temperature (core and peripheral)
Urinary output
Glasgow Coma score

Time Out 9.2

Take a moment to write down how you would manage a 60-year-old man who presents after a haematemesis. Initial vital signs recorded by the nurse are:

Respiratory rate	28/min
SpO_2	92% (air)
Pulse rate	120/min
Blood pressure	90/60
Pale, sweating and anxious	

SUMMARY

There are many causes of shock, but all lead to inadequate delivery of oxygen to vital tissues. The management goal is to treat hypoxaemia and hypovolaemia, whilst excluding the immediately life-threatening conditions. It is also important to realise that resuscitation, though crucial, only plays a preliminary part in the patient's long-term management. It is therefore important that shocked patients receive multispecialty input from the beginning.

Advanced Life Support Group

CHAPTER 10
The patient with chest pain

OBJECTIVES

After reading this chapter you will be able to:

- identify and treat immediate life-threatening causes of chest pain
- formulate a differential diagnosis for non-immediately life-threatening causes of chest pain
- discuss the investigation and management of other causes of chest pain.

INTRODUCTION

Chest pain has many underlying causes and these range from the immediately life-threatening to the trivial. The nature of the pain (site, severity, radiation and associations) varies with the actual cause, but in clinical practice immediately life-threatening causes (next box) can be difficult to identify rapidly. Therefore, a structured approach to care is advocated, starting with a primary assessment and resuscitation followed by secondary assessment and emergency treatments.

Life-threatening causes of chest pain

Myocardial infarction
Dissecting aortic aneurysm
Massive pulmonary embolus
Tension pneumothorax
Oesophageal rupture

PRIMARY ASSESSMENT AND RESUSCITATION

This concentrates on evaluating and maintaining the ABCs. If the patient is conscious, it is usually also possible to gain key information about their chest pain at the same time.

Airway

Airway patency must be assessed and secured where necessary. If the patient's conscious level is fluctuating, then simple airway adjuncts may be needed. If the airway cannot be maintained despite these measures, then endotracheal intubation may be needed. Tolerance of a Guedel airway suggests the patient's airway is

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currently unprotected from the risk of gastric aspiration (although prompt resuscitation may improve this situation).

Breathing

All patients will require high concentrations of inspired oxygen at 12–15 l/min, via a non-rebreathing mask with reservoir.

The rate, symmetry and effort of respiration should be noted. Palpation in the sternal notch will determine if there is tracheal deviation or tug. After percussing the anterior chest wall for areas of hyper-resonance or dullness, breath sounds should be auscultated and any additional sounds, such as a pleural rub, identified.

Inadequate breathing should be supported – initially by bag–valve–mask ventilation and then by intubation and mechanical ventilation. Pulmonary emboli producing pleuritic chest pain are, in themselves, rarely life-threatening. However, such a symptom should raise the clinician's suspicion of the potential for a larger embolus that may have a significant haemodynamic effect, including pulseless electrical activity (PEA, or previously electromechanical dissociation).

Tension pneumothoraces are a rare cause of chest pain, but are rapidly fatal if ignored. You must be alert to this problem, in particular in patients with pre-existing lung disease. Once the diagnosis has been made, time should not be wasted getting X-rays. An immediate needle thoracocentesis is required. This converts the tension into a simple pneumothorax and allows time for chest drain insertion.

Circulation

Check for the presence of an arterial pulse and assess the rate. The carotid artery is the first choice but radial, carotid and femoral arteries should be palpated to determine their pressure, volume and radio-radial or radio-femoral delay. Check the precordium for the position and character of the apex beat, plus any thrills or heaves. Listen for the presence of normal, altered and added heart sounds, as well as murmurs.

Ideally, all patients should have IV access – the antecubital fossa is usually the easiest site. Monitoring should include SpO_2, pulse, blood pressure and ECG.

Immediate investigations

All patients with non-traumatic chest pain will require an immediate 12-lead ECG, as it can help in differentiating the causes of chest pain (next box).

Key point

A normal ECG does not exclude an acute coronary syndrome of any sort

A diagnosis of myocardial infarct made on ECG should lead to rapid emergency treatment. A 12-lead ECG may be normal during the evolution of myocardial infarct. If pericarditis is present, then the classic concave ST elevation occurs in the leads that lie over the affected area.

ECG features of immediately life-threatening causes of chest pain

Myocardial infarction	**Normal**
	1 mm (01 mV) ST elevation in two of the inferior leads (II, III and aVF)
	1 mm (01 mV) ST elevation in leads 1 and aVL
	2 mm (02 mV) ST elevation in two contiguous anterior chest leads
	New left bundle branch block
	True posterior infarct
Pulmonary emboli	**Normal**
	Sinus tachycardia
	Atrial fibrillation or tachycardia
	Right axis deviation
	Symmetrical T wave inversion in the anterior chest leads
	Right ventricular strain
	Right bundle branch block
Dissecting aortic aneurysm	**Normal**
	Signs of left ventricular hypertrophy and strain due to hypertension
	Acute ischaemic changes, including changes of classical myocardial infarction, when coronary ostia are involved (very rare)

Once intravenous access has been secured, blood should be taken for full blood count, markers of myocardial damage (e.g. troponin I or T), electrolytes and blood glucose. If a dissecting aneurysm is suspected, blood transfusion may be required and, therefore, a sample should be taken for cross-match. Arterial blood gas measurement is also ideally required to exclude any underlying acid–base disturbance, ventilation–perfusion mismatch and inadequate ventilation.

SECONDARY ASSESSMENT

Immediately life-threatening conditions are rare, but it is important that they have been excluded or treated. Attention can then be directed to the secondary assessment, where the crucial exclusions are myocardial infarction, pulmonary embolus, oesophageal rupture, pneumonia and pneumothorax. These last two conditions are usually easily diagnosed radiologically; therefore, the essential management plan is to rule out myocardial infarction and pulmonary embolus. If neither myocardial infarction nor pulmonary embolism seems likely, oesophageal rupture should be considered. Other minor conditions can be investigated, often as an outpatient.

History

Clinical diagnoses are frequently made on the basis of a medical history. The features of chest pain should be assessed in a regular and orderly sequence, paying particular attention to the site, character, radiation, precipitation and relieving factors as well as any other associated symptoms.

A pertinent history can provide invaluable clues as to the differential diagnosis of conditions giving rise to chest pain.

A patient with an acute myocardial infarct and associated ECG changes should be identified immediately and treated appropriately. The remaining patients will have diagnoses ranging from acute coronary syndrome to musculoskeletal pain. While the particular diagnosis in individual patients may take some time to establish, the risks of either myocardial infarction or of later complications can be rapidly assessed by repeating and reviewing the ECGs, taking a focused history and examining the patient. This will allow appropriate decisions about further care to be made.

The most important finding is a history of cardiac-type chest pain, and any patient presenting with such a history requires continuous ECG monitoring in an area where cardiopulmonary resuscitation – especially defibrillation – can be provided immediately. Subsequent investigations by assay of chemical markers of cardiac damage, particularly cardiac troponins, will both help identify patients with cardiac ischaemia presenting with normal ECGs and will help risk stratify all patients, as does pre-discharge exercise testing or nuclear perfusion imaging. Both ECG and clinical findings can predict a high risk of subsequent myocardial infarction and its complications. Dynamic ECG changes, elevation of troponin levels or clinical evidence of left ventricular dysfunction are adverse findings and should prompt consideration of early – pre-discharge – coronary angiography. The approach to clinical risk assessment is shown in Fig. 10.1.

The pain from a dissecting thoracic aortic aneurysm may be severe and poorly responsive to opiates. It usually starts in the centre of the chest, radiates through to the back between the scapulae and may involve the upper limbs. It is often described as tearing, but the nature of the pain may change as the dissection progresses. Dilatation of the aortic root caused by aortic dissection can lead to aortic valve incompetence and regurgitation. Any patient who has evidence of both myocardial infarction and aortic regurgitation should be screened for aortic dissection. Risk factors need to be sought, in particular a family history of ischaemic heart disease, hyperlipidaemia, hypertension, diabetes mellitus, Marfan's syndrome, homocystinuria, procoagulant disorder and a history of cigarette smoking. Oral contraceptive pill use or pregnancy may influence the differential diagnosis, as may the patient's occupation.

Tension pneumothorax can present with progressive dyspnoea, occasionally pleuritic pain, and in extreme cases, a cardiorespiratory arrest. A similar range of presentations may be encountered in patients with pulmonary emboli. It is therefore important to enquire about the history of breathlessness and haemoptysis, as this may help in establishing the correct diagnosis (see Chapter 8).

Oesophageal rupture is a rare cause of severe chest pain, but carries a poor prognosis if not recognised early. Whereas myocardial infarction often has chest pain associated with vomiting, oesophageal rupture has vomiting as an initial symptom, followed by chest pain caused by mediastinitis. The pain is severe and often there are no clinical signs. Be wary of diagnosing a 'functional disorder'.

The clinician should ascertain additional information, so that a well-'phrased' history has been obtained by the end of the secondary assessment.

Examination

The secondary assessment ensures that the physical examination started in the primary assessment is completed in a comprehensive fashion. The blood pressure, pulse pressure and the height and character of the jugular venous pulse should also be recorded. Assessment of the site and character of the apex beat, as well as the presence of normal and additional heart sounds can then be done.

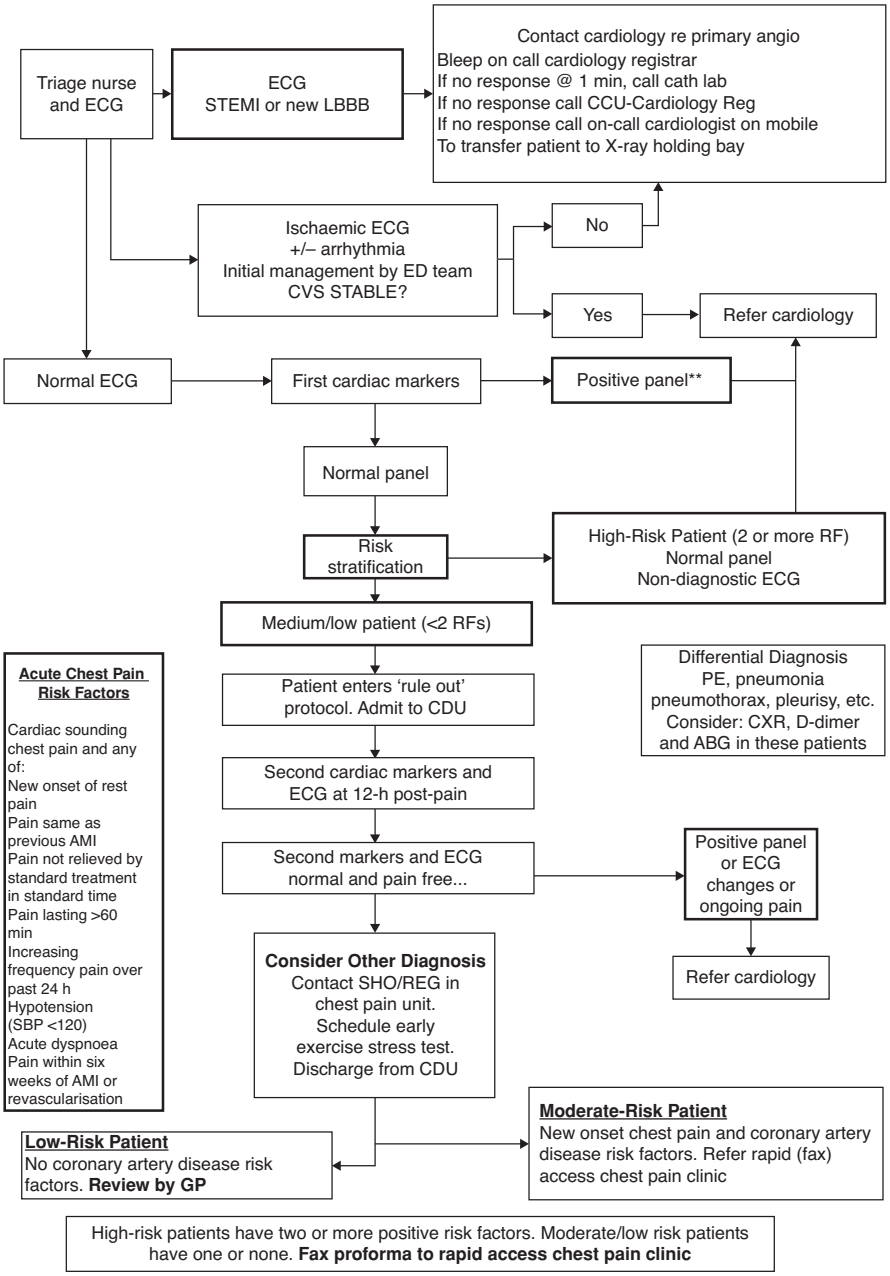

Fig. 10.1 Chest pain assessment.

Occasionally, a blowing early diastolic murmur may be heard when a dissecting thoracic aortic aneurysm has involved the aortic valve. This may be the only initial physical sign.

The clinician should then consider and integrate the facts from the history with the examination findings to pinpoint the cause of the chest pain. The commonest causes of chest pain are listed in Tables 10.1–10.5. For clarity, these are divided into body systems:

- Cardiac chest pain
- Chest pain caused by respiratory disease and oesophageal rupture
- Chest pain caused by gastrointestinal disease
- Chest wall pain
- Functional chest pain

Table 10.1 Clinical features of cardiac chest pain

	Ischaemia	Pericarditis
Site	Retrosternal Deep Arms alone (infrequently)	Surface – according to site of pericarditis
Character	Constricting, band like, heavy	Sharp
Radiation	Left arm > right arm Throat/jaw/teeth (rare)	Left arm > right arm Throat (very rare)
Precipitation (**Stable angina only**)	Exertion Cold winds Anxiety Heavy meals	Deep inspiration Coughing Postural variation – according to site of pericarditis
Relief (**Stable angina only**)	Rest Nitrates (within 1–2 min)	Postural variation
Associated symptoms	Strangling sensation in the throat	Occasionally pleuritic pain (on respiration)
Clinical examination	Limited value Occasionally signs of hyperlipidaemia or atherosclerosis	Pericardial rub

Table 10.2 Clinical features of chest pain caused by respiratory disease and oesophageal rupture

	Pleuritic	Spontaneous pneumothorax	Mediastinitis
Site	Anterior/lateral/posterior	Anterior/lateral/posterior or none	Mid-line Retrosternal
Character	Sharp, stabbing, catching	Sharp, stabbing, catching	Usually stabbing, but may mimic cardiac pain if severe
Radiation	Shoulder tip if basal pleuritis		Neck, back
Precipitation	Coughing Deep inspiration	Coughing Deep inspiration	Coughing Deep inspiration Postural variation
Relief	Shallow breathing Postural variation	Shallow breathing	Postural variation
Associated symptoms	(a) Infective Cough Purulent sputum Prodromal symptoms (b) Embolic Cough Breathless Haemoptysis (see earlier)	Breathless Cough (see earlier re: Tension pneumothorax)	Cough Neck and facial subcutaneous emphysema
Clinical examination	Pleural rub ± signs of consolidation	Hyper-resonant percussion note Reduced breath sounds	Mediastinal friction rub Subcutaneous emphysema

Table 10.3 Clinical features of chest pain caused by gastrointestinal disease

	Peptic ulcer disease	Gastro-oesophageal reflux disease – GORD	Diffuse oesophageal spasm
Site	Epigastric Right hypochondrium	Retrosternal Epigastric	Retrosternal Deep
Character	Postprandial constant/gnawing	Burning	Squeezing Constricting
Radiation	Posteriorly (posterior duodenal ulcer)	Retrosternal Throat	Throat Arms (rare)
Precipitation	Eating Alcohol	Drinking Eating Lying/bending	–
Relief	Acid suppressants Antacids	Standing Antacids Acid suppressants	Antispasmodics Nitrates
Associated symptoms	Nausea Vomiting Waterbrash	Nausea Waterbrash Bronchospasm	Dysphagia
Clinical examination	Epigastric tenderness	Often overweight	Often associated with GORD

Table 10.4 Clinical features of chest wall pain

	Muscular	Cervical spondylosis	Costochondritis	Herpes zoster (shingles)
Site	Intercostal, periscapular, localised	Upper chest, arms (corresponding to nerve root involved)	Costochondral junctions	Chest wall dermatomes
Character	Sharp, stabbing ache	Ache Constant	Ache Sharp	Severe lancing
Radiation	Shoulder	Arms Inframammary	–	Affected nerve root(s)
Precipitation	Coughing Deep inspiration Physical effort involving upper limbs	Movement Physical effort involving upper limbs	Deep inspiration Lying on anterior chest wall	Previous varicella zoster
Relief	Rest Analgesia (NSAIDs) Heat	Rest Analgesia (NSAIDs) Immobilisation	Anti-inflammatory drugs (NSAIDs)	Analgesia Aciclovir Carbamazepine
Associated symptoms	Focal tenderness over muscle mimicked by compression of thoracic cage, movement of muscle group	Paraesthesia (nerve root distribution)	Focal erythema/swelling Tenderness over joint	Erythematous vesicular rash (pain precedes rash)
Clinical examination	Focal tenderness	Restricted neck movement Nerve root signs	Focal erythema/tenderness	Rash

Table 10.5 Clinical features of functional chest pain

Site	Anywhere
	Often left inframammary
Character	Often sharp and stabbing, but may be dull
	Often unrelenting, lasting many hours or even days
Radiation	None, usually
	Arms, infrequently
Precipitation	Anxiety
	Tension
Relief	Reassurance
	Treat underlying cause
Associated symptoms	Palpitations
	Breathless
	Headaches
	Other somatic symptoms
Clinical examination	Unremarkable

EMERGENCY TREATMENT

ST elevation myocardial infarction

In the presence of myocardial infarction, aspirin and vasodilators, i.e. sublingual or intravenous nitrates, will be required. If this combination fails to relieve pain, then opiates should be used. Direct reperfusion with percutaneous coronary intervention is the treatment of choice, if available within 90 min. Pre-treatment with clopidogrel 600 mg and heparin 5000 IE IV can be given, depending on local protocols, along with the aspirin and nitrates.

If coronary intervention is not available, thrombolysis should be started as soon as possible, provided there are no contraindications (next box). Clinical trials have shown that the beneficial effect of thrombolysis is significantly reduced with time, particularly if it is started over 12 h after the onset of chest pain.

Contraindications to thrombolysis

History of gastrointestinal or genitourinary bleeding in the previous two months
Recent major surgery (including dental extraction), trauma, biopsy or head injury
History of intracranial or spinal cord haemorrhage, aneurysm or neoplasm
History of cerebrovascular disease, especially recent events or with any residual disability
Reduced level of consciousness
History of bleeding disorder
Pregnancy or heavy vaginal bleeding (normal menstrual period is not a contraindication)
Traumatic cardiopulmonary resuscitation within the previous 10 days
Non-compressible intra-arterial diagnostic procedures within previous 14 days
Aortic dissection
Acute pancreatitis
Severe liver disease, oesophageal varices

Systolic blood pressure greater than 200 mm Hg or diastolic greater than 110 mm Hg (Thrombolysis may be given if treatment successfully lowers BP to acceptable levels)

The benefits of thrombolysis can be expected to be:

Greatest in patients presenting early – within 4 h, with extensive ST segment changes in many leads, with marked ST segment elevation – more than 5 mm and involving the anterior chest leads – V1 to V6

Least in patients presenting late – approaching 12 h, with limited ST segment changes – in few leads with minor ST segment elevation – 1 or 2 mm and involving the inferior leads – II, III and aVF

The risks of thrombolysis are not time dependant and there is, in particular, a risk of intracerebral haemorrhage of 1%, with half of these patients dying and half of the survivors being disabled. In the elderly, this risk increases to 2.4% in those over 75 years of age. Those at greatest risk are older patients, with hypertension (systolic BP >140 mm Hg or diastolic BP >100 mm Hg) lighter body weight (<67 kg), female and black. Clinical judgements need to be made in each case to optimise patient care. If thrombolytic therapy is contraindicated, aspirin can still be given and affords equal benefit.

Unstable angina and non-ST elevation myocardial infarction (NSTEMI)

In common with all patients with possible cardiac chest pain, patients with unstable angina and NSTEMI should receive aspirin and appropriate analgesia. Antithrombotic therapy should be given to all patients with unstable angina or NSTEMI. Low molecular weight heparin is more effective than unfractionated heparin at reducing the incidence of ischaemic events and the need for revascularisation procedures. The incidence of major bleeding complications is the same for both forms of heparin. Thus, all patients who have chest pain of probable cardiac origin and who are not eligible for fibrinolytic drugs should receive low molecular weight heparin. Patients who have high risk features such as dynamic ECG changes or elevated cardiac troponin levels, are also candidates for additional antiplatelet therapy with glycoprotein IIb/IIIa receptor inhibitors depending on local protocols

Pulmonary embolus

If the diagnosis is suspected, start treatment with heparin (either unfractionated or low molecular weight) immediately, after which embolic, pleuritic pain often melts away within 2–6 h. Thrombolysis is a useful therapeutic adjunct in severe disease (see Chapter 8 and 9).

Dissecting aortic aneurysm

This condition is rare, but be alert to the patient with classical myocardial infarction and aortic regurgitation who may have aortic dissection. When a dissecting aortic aneurysm is discovered, the optimum systolic blood pressure is considered to be 100 mm Hg. This may be achieved by either pharmacological reduction of hypertension or titrated fluid replacement with hypotension. Cardiothoracic advice must be sought urgently.

Tension Pneumothorax

This is considered in detail in Chapter 8.

Oesophageal Rupture

This condition is also rare, and like dissecting aortic aneurysm rarity leads to difficulties in diagnosis which, if delayed, can significantly increase mortality due to the development of mediastinitis. Oesophageal rupture occurs when vomiting takes place against a closed glottis and is often associated with alcohol excess. The sequence of events is that of vomiting then chest pain, rather than the opposite order, which may occur in myocardial infarction. Other causes include therapeutic, and occasionally diagnostic, upper gastrointestinal endoscopy. The patients suffer severe pain that seems out of proportion to other symptoms and signs, especially when initial tests are negative. Hence such patients may be diagnosed as 'functional pain'. Beware! The diagnosis is usually made from the history. The key finding is the presence of air in the mediastinum on chest X-ray, or clinically as surgical emphysema in the subcutaneous tissues of the neck. Arrange for urgent assessment by an intensivist and either an upper gastrointestinal or cardiothoracic surgeon.

DEFINITIVE CARE

After taking a history and examining the patient, the clinician will have either established a diagnosis or postulated a differential diagnosis. The patient with a suspected acute coronary syndrome will be transferred to coronary care, whilst patients with other causes of chest pain will be managed – at least initially – either in another critical care area or on a medical ward. Appropriate investigations will then be required to confirm or refute these conclusions. The choice will depend on which body system(s) is involved.

Investigations

ECG

All patients with chest pain need 12-lead ECGs. Those with possible acute coronary syndromes should have continuous electrocardiograph monitoring, during their initial assessment. Exercise stress testing is a useful tool for risk stratification, both after myocardial infarction and other acute coronary syndromes, where it is used to identify potential candidates for coronary angiography. In addition, it is used to identify patients with ischaemic heart disease who have not shown any other markers following their presentation with chest pain

Imaging

Chest X-ray

A chest X-ray is of limited diagnostic use in patients with angina and myocardial infarction, unless the latter is complicated by heart failure and/or aneurysm of the left ventricle. Similarly, it is usually unremarkable in patients with pericarditis unless there is a coexisting pericardial effusion. A plain chest radiograph may be normal in uncomplicated pleuritis and even with pulmonary emboli. It may, however, show evidence of pulmonary parenchymal infection and either a wedge shaped peripheral defect or hyperlucency associated with pulmonary embolus. Atelectasis is also a manifestation of both of these conditions. A chest X-ray is essential for diagnosing spontaneous, simple pneumothorax, especially when the film is taken in expiration.

In the case of dissecting aneurysm, a chest X-ray may show widening of the mediastinum, deviation of trachea to the right, elevation of right main bronchus and depression of left main bronchus, a pleural cap, the obliteration of the aorticopulmonary window and, more obviously, the aortic knuckle.

The chest X-ray may be normal in a patient with an oesophageal rupture. Other features include air in the mediastinum and/or soft tissues of the neck, and a pleural effusion.

VQ scans

Ventilation perfusion (VQ) scans are rarely used for the immediate diagnosis of pulmonary emboli.

CT Pulmonary Angiography

This is the investigation of choice in patients with suspected pulmonary embolic disease (see Chapter 8).

Echocardiogram

Transthoracic echocardiography is helpful in the diagnosis of pericardial effusion and dissecting aneurysm. However, in the latter, a trans-oesophageal echocardiogram is preferred. CT aortography is an alternative, which may be more readily available, and magnetic resonance imaging has also been used. Transthoracic echocardiography may also help in assessing valvular pathology, pulmonary artery pressures and the presence of thrombus as well as function of the left and right ventricles and atria.

Markers of myocardial damage

These should be requested in all patients with chest pain.

The traditional markers of creatine phosphokinase, aspartate transaminase and lactate dehydrogenase are being superceded by newer tests. Other markers such as the cardiac troponins, Troponin I and T, are used in routine clinical practice. It is important to be aware of your local laboratory protocols and reference ranges.

Endoscopy

Endoscopy is essential for investigating peptic ulcer as well as gastro-oesophageal reflux disease. A normal endoscopy, however, does not exclude this disease and formal pH manometry and a semisolid phase barium swallow may be required to confirm the diagnosis – similarly for diffuse spasm.

Either a barium swallow and/or a CT scan are needed in order to investigate suspected oesophageal perforation. Endoscopy should be avoided unless it is done by the surgeon who is contemplating an operation.

DIAGNOSTIC PITFALLS

- Treat all patients with a good history of cardiac type chest pain as having an acute cardiac syndrome until proven otherwise.
- Remember that elderly patients may have atypical presentations of acute cardiac ischaemia and have fewer ECG changes.
- Diabetic patients may have ‘silent ischaemia’ as may elderly patients.
- Patients with chest pain, irrespective of the cause, will manifest clinical features of anxiety.
- Always reassess every episode of chest pain as though it were the first, even in frequent attenders.
- The absence of deep vein thrombosis does not rule out a diagnosis of pulmonary embolus.
- If the patient’s history suggests a pneumothorax but there is no radiological evidence, re-evaluate the X-ray for evidence of pneumomediastinum.

- A normal chest X-ray and white cell count does not exclude the diagnosis of pulmonary infection.
- Rapid pain relief with nitrates does not point to a diagnosis of angina – diffuse oesophageal spasm responds in an identical fashion.
- Gastro-oesophageal reflux is a common problem and does not indicate that chest pain is due to this cause.

SUMMARY

- Chest pain is a common presentation requiring acute medical admission.
- Most causes of chest pain are not immediately life-threatening.
- Rule out myocardial infarction and pulmonary embolus in patients with a compatible history.

Advanced Life Support Group

CHAPTER 11

The patient with altered conscious level

OBJECTIVES

After reading this chapter you should be able to:

- understand the physiology of the conscious state and how this may be disturbed
- understand how the structured approach can be applied to the unconscious patient
- discuss the initial management of such a patient
- discuss how clinical signs detected in the secondary assessment will influence your diagnosis and subsequent management.

INTRODUCTION

The care of the unconscious patient is a common medical emergency. To understand why patients become unconscious, it is necessary to review briefly the physiology of consciousness, by considering:

- neurophysiology
- cerebral metabolism
- cerebral perfusion
- intracranial pressure.

Neurophysiology

There are two interlinked areas that are of paramount importance in maintaining the conscious state:

- the reticular formation
- the cerebral cortex.

The reticular formation arises in the brain stem, in the midst of a host of neural pathways communicating between the brain and spinal cord and vice versa. It contains the primary centres for cardiovascular and respiratory control, as well as a distinct area called the reticular activating system (RAS). This is crucial for maintaining the conscious state. Neurones in the RAS system pass via the thalamus to synapse in the cortex. There is no specific individual area in the cortex that is responsible for the conscious state, but the coordinated interaction of many cortical areas is required.

It is important to note that the cerebral cortex and the interconnections (including thalamus and hypothalamus) have to be affected bilaterally to affect consciousness. Metabolic disorders, toxins, hypoxaemia and a postictal state are more

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likely to affect conscious level by affecting the cerebral cortices, whereas the RAS is affected by supratentorial pressure, infratentorial pressure or intrinsic brainstem lesions.

Cerebral metabolism

Glucose and oxygen are the essential fuels for cerebral metabolism. The brain, however, only has a small store of glucose and thus its supply is critically dependent on adequate cerebral blood flow. In practice, the brain can only function for approximately 2–3 min in the absence of glucose. If the brain is deprived of both glucose and oxygen, as in cardiorespiratory arrest, then normal energy metabolism can only continue for about 15 s.

Cerebral perfusion

Adequate ventilation and cerebral perfusion are necessary to ensure that the brain is provided with oxygen and glucose. Under normal resting conditions, the brain receives approximately 15–20% of the resting cardiac output. Cerebral blood flow is autoregulated to ensure a constant supply of blood with a mean arterial pressure of 60–160 mm Hg. Within this range, a rise in blood pressure is balanced by intracranial vasoconstriction and, conversely, a fall by vasodilatation. Cerebral blood flow depends not only on the mean arterial pressure, but also on the resistance to blood flow due to intracranial pressure and, to a lesser extent, the central venous pressure.

Cerebral perfusion pressure (CPP) = mean arterial pressure − intracranial pressure

Autoregulation is impaired in conditions like infection and trauma, and, in particular, chronic hypertension. However, under normal circumstances, if cerebral blood flow falls (mean arterial pressure drops below 60 mm Hg) then cerebral ischaemia occurs. In contrast, cerebral oedema and hypertensive encephalopathy may ensue if the mean arterial pressure exceeds 160 mm Hg.

Intracranial pressure

In the adult the volume of the intracranial contents, comprising the brain, cerebrospinal fluid, blood and blood vessels, is fixed by the surrounding rigid skull. These contents produce an intracranial pressure of 6–13 mm Hg. To maintain this normal range, any increase in volume of one of the contents must be balanced by a corresponding decrease in one or more of the others.

The brain is a compliant organ that will mould to accommodate an increase in pressures. Furthermore, cerebrospinal fluid can be displaced into the spinal system and the volume of cerebral venous fluid, in particular within the dural sinuses, can be displaced into the systemic venous circulation. These mechanisms will initially offset any rise in intracranial pressure.

Once these normal compensatory measures are exhausted, any further small increase in intracranial volume will lead to large increases in intracranial pressure, which, in turn, will reduce cerebral perfusion pressure.

Therefore, disruption of one or more of these four mechanisms will result in loss of consciousness.

CAUSES OF COMA

The causes of coma are listed in Tables 11.1–11.3 according to the presence of neck stiffness and/or lateralising signs.

Table 11.1 Causes of coma: no menigism: no focal/lateralising signs

Drug overdose	Daily
Ischaemia/hypoxaemia*	Daily
Hypoglycaemia*	Daily
Cardiac failure	Daily
Respiratory failure	Daily
Alcohol*	Daily
Renal failure	Weekly
Diabetic ketoacidosis	Weekly
Hepatic failure*	Monthly
Hyponatraemia	Monthly
Sepsis	Monthly
Wernicke's encephalopathy	Monthly
Carbon monoxide poisoning	Annually
Hypernatraemia	Annually
Hypothermia	Annually

*These rarely present with focal signs.

Table 11.2 Causes of coma and neck stiffness: no focal/lateralising signs

Bacterial meningitis	Daily
Encephalitis	Weekly
Subarachnoid haemorrhage	Weekly
Cerebral/Cerebellar haemorrhage with extension into subarachnoid space	Monthly
Cerebral malaria	Only in exams

Table 11.3 Causes of coma with focal/lateralising signs

Intracerebral tumour	Daily
Intracerebral haemorrhage	Daily
Intracerebral infarction	Daily
Intracerebral abscess	Monthly

PRIMARY ASSESSMENT AND RESUSCITATION

In the primary assessment, airway, breathing and circulation need to be assessed and managed appropriately. Irrespective of the underlying pathology, every effort should be made to prevent secondary brain damage by identifying and treating hypoxaemia, hypercapnia, hypotension, hypoglycaemia and raised intracranial pressure.

Senior help including an anaesthetist should be sought immediately for all comatose patients, who should be managed – initially – in the resuscitation area.

A – airway and cervical spine

In the patient with altered consciousness, the potential for airway obstruction is high.

Clear and control the airway using the techniques described in Chapter 4. If a patient is comatose (a Glasgow Coma Score of eight or less), the risk of aspiration is increased if the gag reflex is absent, swallowing (even saliva) is uncoordinated and the airway is unprotected. In such cases, the insertion of a cuffed endotracheal tube, by means of rapid sequence induction of anaesthesia should be considered. All patients require high concentrations of inspired oxygen using an appropriate delivery system.

Acute cervical spine problems are rare in acute medicine, but be wary of the patient found unconscious/confused at the bottom of the stairs. Consider a potential cervical spine/cord injury if there is no clear history, and especially if there are external signs of trauma above the clavicle. Seek specialist help to immobilise the cervical spine if you are concerned.

B – breathing

Unconscious patients often have low respiratory rates and if less than 10/min, may need assisted ventilation. Ensure the patient is connected to a pulse oximeter.

Patients in respiratory distress (i.e. respiratory rate greater than 30) may have a life-threatening chest problem. Examination of the chest during the primary assessment is designed specifically to pick up any such chest problems. Give naloxone (100 μg/min IV or 400 μg IM if IV access is not possible) to any patient with signs of opoid toxicity. Intubation and ventilation should be considered if the respiratory rate is less than 10/min and there is inadequate or no response to naloxone.

C – circulation

Shock has to be treated appropriately to prevent secondary brain injury. Once intravenous access is established, blood should be taken for immediate glucose estimation, using a glucometer. Unless hypoglycaemia (<4 mmol/l) can be excluded rapidly and reliably, 50 ml of 50% dextrose should be given intravenously. If intravenous access is difficult, glucagon 1 mg intramuscularly should be given instead.

Thiamine deficiency can occur in any acutely ill patient, especially those with chronic liver disease, folate deficiency, malnutrition, anorexia nervosa and high alcohol intake. Thiamine should be given intravenously (for at least 3 days) especially in patients with Wernicke's encephalopathy.

In addition to the 'routine' blood tests, arterial blood gases must be measured. Patients should be connected to the cardiac monitor and a urinary catheter inserted if the unconsciousness is not quickly reversed.

D – disability

The initial neurological assessment should be a rapid evaluation of the Glasgow Coma Score and pupil size, equality and reaction to light. Check for meningeal irritation – if there are no contraindications. Remember, at this stage, you are looking for conditions which are immediately life-threatening! Consider:

- hypoglycaemia
- antidotes, e.g. naloxone if pinpoint pupils/needle track marks
- antibiotics
- aciclovir
- antiepileptic drugs.

E – exposure

The patient must be fully exposed to allow complete assessment. The temperature must be taken, with a low reading rectal thermometer if necessary. Do not forget

that hypothermia is an important cause of coma and should not be missed. Be wary of inducing hypothermia by fully exposing the patient. The patient's clothes should be searched for useful information such as medical cards, drugs and details of next of kin.

SECONDARY ASSESSMENT

The secondary assessment should only be done once the immediately life-threatening conditions have been treated. A well-'phrased' history should be sought, followed by a complete examination. Further appropriate investigations can be requested.

History

The history is particularly important in this context and must be sought from attending relatives, friends, paramedics and other witnesses. Additional and useful information may be obtained from the hospital notes or the general practitioner.

Examination

A thorough head-to-toe examination of the patient should then take place, looking for evidence of precipitating factors such as head injury, infection, drug use and vascular pathology. In the unconscious patient, particular attention should be paid to the following:

- level of consciousness
- assessment of brain stem function
- focal neurological signs.

Level of consciousness

The Glasgow Coma Scale (GCS, range 3–15) gives a qualitative measurement of the patient's conscious level. The Glasgow Coma Score is the sum of scores in three areas of assessment.

E – best eye opening
V – best verbal response
M – best motor response

Eye opening response

Response	Score
Spontaneous	4
To speech	3
To painful stimuli	2
Nil	1

Verbal response

Response	Score
Orientated	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
Nil	1

Motor response

Response	Score
Obeys commands	6
Localises pain	5
Withdraws from pain	4
Abnormal flexion (decorticate)	3
Abnormal extension (decerebrate)	2
Nil	1

If the patient does not respond to commands, then a painful stimulus is applied by pressure on the supraorbital ridge. If assessment of a verbal response is not possible, e.g.due to an *in situ* endotracheal tube, then this fact should be documented in the patient's notes. The best response of any limb is recorded. If there are differences between limbs, this may suggest a potential neurological lesion. Patients who have a GCS of eight or less are by definition comatose. It is important to recheck the GCS every 15 min.

Assessment of brain stem function

The initial brain stem assessment comprises:

- pupillary response
- eye movements
- corneal response
- respiratory pattern.

Pupillary response

The size, shape and response to light of both pupils should be assessed. An understanding of the common changes in pupillary reflexes is important as this will help to localise lesions (see Table 11.4).

Table 11.4 Pupillary abnormalities

	Pupillary response	Cause
Equal pupils	Small + reactive	Metabolic encephalopathy
		Midbrain herniation
		Senile miosis
	Pinpoint + fixed	Pontine lesion
		Opioids, organophosphates
	Dilated + reactive	Metabolic cause
		Midbrain lesion
		Ecstacy, amphetamines
	Dilated + fixed	Peri ictal
		Hypoxaemia
		Hypothermia
		Anticholinergics
Unequal pupils	Small + reactive	Horner's syndrome
	Small + 'non reactive'	Argyll Robertson (tertiary syphilis)
	Dilated + fixed	Uncal herniation
		IIIrd nerve palsy

Eye movements

Many comatose patients have roving or dysconjugate eye movements; these are common and are of no particular significance.

The oculocephalic response (doll's head/eye movement) provides useful information about the oculomotor and vestibular components of brain stem function. Hold the eyelids open and observe eye movements. Illicit the response by quickly turning the head to the right and then to the left.

- Eyes move in opposite direction to head turn – normal
- Eyes move to one side but not the other – unilateral brain stem lesion – lateral gaze palsy
- Eyes fail to move in any direction – bilateral brain stem lesions

Key point

Do not attempt to elicit the oculocephalic response if cervical spine pathology is suspected

The oculovestibular (caloric) test is a much more potent stimulus to brain stem function than the oculocephalic reflex. It is more time consuming to perform and often unsuitable for emergency assessments. However, it is particularly useful in differentiating psychogenic unresponsiveness from coma.

Having first ensured that the ear drums are intact, the head is inclined at 30° to the trunk and the external auditory canal of one ear is irrigated with ice cold water while the eyes are held open. The following responses may occur:

- Tonic deviation of the eyes towards the irrigated ear in those comatose patients with an intact vestibular component.
- Nystagmus (the quick phase is away from the irrigated side) and vomiting in patients not in coma.
- No movement of the eyes when brain stem function is lost.
- Asymmetry of the oculovestibular response is characteristic of focal brain stem lesion.
- A fixed conjugate gaze due the absence of the fast component indicates cortical damage with an intact brainstem.

Corneal reflexes

They are usually preserved in coma. In the absence of drugs, the loss of this reflex is a very poor prognostic sign.

Respiratory pattern

Alterations in brain stem function produce a variety of respiratory patterns, which may help to localise the lesion. In practice, however, they are of limited value.

- Normal breathing (eupnoeic), e.g. postictal, metabolic coma
- Periodic breathing (Cheyne–Stokes), e.g. lesions in the thalamus and hypothalamus, though there are many non-cerebral causes, including heart failure
- Central neurogenic hyperventilation, e.g. lesions in the midbrain or upper pons
- Slow and irregular breathing (apneustic) lesions in the medulla
- Deep sighing respiration (Kussmaul's) associated with a metabolic acidosis.

Focal neurological signs

The motor system must be assessed for asymmetry of tone, response to pain and deep tendon reflexes. Focal signs with intact brain stem reflexes occur with a focal

hemispheric lesion, whereas focal signs with absent brain stem reflexes are signs of a lesion in the posterior fossa. Assessment of the brain stem reflexes can be very valuable in localising the lesion and selecting patients for immediate further investigation, e.g. CT or MR scanning.

Key point

The absence of papilloedema does not exclude raised intracranial pressure

The fundi must always be examined. The presence of papilloedema indicates raised intracranial pressure. Subhyaloid haemorrhage should be sought as this may indicate subarachnoid haemorrhage or basal skull fracture. Furthermore, changes associated with diabetes mellitus and hypertension also need to be noted.

It cannot be overemphasised that the initial neurological examination is only the beginning. The initial findings are a 'baseline' for comparison with repeated neurological examinations.

Do not forget that specific evidence should also be sought of meningeal irritation (usually neck stiffness, Kernig's sign and Brudzinski's sign). Neck stiffness should not be elicited if cervical spine instability is suspected. It is important to realise that neck stiffness is often absent if the patient's conscious level is depressed.

Investigations

Neuroradiology

Computerised tomography (CT) of the brain is the primary investigation in coma. It is relatively quick and will identify 99% of supratentorial masses, especially when the non-contrast scan is supplemented with one using intravenous contrast. It is important to maintain adequate resuscitation during the scan. Restless or uncooperative patients will need to be electively anaesthetised, intubated and ventilated to get good quality images, avoiding movement artefact.

In comparison with CT scanning, cranial magnetic resonance imaging (MRI) is a more sensitive imaging modality. It not only will demonstrate most cerebral disease processes, but is also the ideal method for imaging posterior fossa lesions. These cannot be seen on CT imaging, because of obtrusive artefacts (e.g. the dense bone of the skull base). CT is the investigation of choice for subarachnoid haemorrhage as it is more sensitive for fresh blood. MRI is, however, highly sensitive for infective/inflammatory changes to cerebral tissue enabling identification of encephalitic disorders at an earlier stage when the CT scan will be normal. A further advantage is that magnetic resonance angiography can be done as a non-invasive, radiation-free technique for the investigation of intra- and extracerebral vascular structures. Unfortunately MRI is a very lengthy process when compared with CT scanning. Furthermore, it is extremely difficult to continue resuscitation in the MRI scanning area, as metal components are not allowed within the area of the 'magnet'. Therefore, for practical reasons, CT is still the initial modality of choice. It is important to realise that the scan must not delay the diagnosis and treatment of conditions like meningitis and encephalitis. These should be treated early with appropriate antibiotics and/or antiviral agents based on a clinical diagnosis before scanning.

Lumbar puncture

A lumbar puncture should not be done in the unconscious patient until a CT scan has excluded a mass lesion. Failure to do this may precipitate central/uncal

herniation as cerebrospinal fluid is drained via the lumbar puncture needle. Furthermore, a diagnosis of subarachnoid haemorrhage on CT will negate the need for lumbar puncture. However, CT scans can miss a small subarachnoid bleed and as this often heralds subsequent catastrophic haemorrhage, a lumbar puncture must be done in any patient who has a clinical history suggestive of subarachnoid haemorrhage and a negative CT scan. Other conditions which may cause coma and neck stiffness are shown in Table 11.2. See further details on lumbar puncture in Chapter 33.

It is important to remember that meningeal irritation may be absent when conscious levels are depressed. The pyrexial unconscious patient without evidence of mass lesion on CT scan will warrant a lumbar puncture. The prognosis is extremely poor for patients with meningitis who are unconscious before treatment is started.

Emergency management

The aims of emergency management are to maintain adequate cerebral metabolism and prevent and/or treat intracranial hypertension whilst a specific diagnosis is made and treatment started.

Maintain cerebral metabolism

The principal metabolic requirements of the brain are oxygen and glucose. Delivery of adequate levels of these substrates must be ensured. The oxygen content of the blood depends on the haemoglobin level and arterial oxygen concentration. The arterial oxygen concentration can be assessed using blood gas analysis and pulse oximetry. Low arterial oxygen tension also has profound effects on cerebral blood flow. When it falls below 50 mm Hg (6.7 kPa), there is a rapid increase in CBF and intracranial blood volume. Supplementary oxygen must be given to prevent this threshold being reached.

The concentration of glucose in the blood must be considered early and the brain must be protected from hypo- or hyperglycaemia. Close control of blood sugar concentrations between 4 and 8 mmol/l offers better preservation of cerebral function in hypoxaemia, cerebral haemorrhage and traumatic brain injury.

Maintain cerebral blood flow

The cerebral blood flow depends on the difference between systemic arterial pressure and intracranial pressure.

Systemic arterial pressure

The aim is to maintain a normal blood pressure, considering the nature of the intracranial pathology and pre-existing medical conditions, e.g. hypertension.

Although autoregulation will endeavour to preserve cerebral perfusion, causes of hypotension, e.g.hypovolaemia and sepsis, must be identified and treated immediately. Conversely, hypertension is often a compensatory response to maintain cerebral perfusion in patients with raised intracranial pressure. Therefore, treat the underlying condition and not the hypertension in patient with raised intracranial pressure. Any reduction in blood pressure will reduce cerebral perfusion pressure resulting in global cerebral infarction.

Intracranial pressure

Several approaches can be taken to keep pressure to acceptable levels:

- $PaCO_2$ should be kept within the normal range. Elevations in $PaCO_2$ will be associated with cerebral vasodilatation and exacerbate the raised intracranial

pressure. In contrast, hyperventilation will not only reduce the arterial carbon dioxide tension and hence reduce cerebral oedema, but also reduce cerebral blood flow, resulting in ischaemia. Keeping the $PaCO_2$ within the range 4.0–4.5 kPa should therefore be the goal. Seek an early liaison with an intensivist/neurosurgeon, maintain a normal $PaCO_2$ and monitor the $PaCO_2$ and intracranial pressure as required.

- Overhydration must be avoided as this may increase cerebral oedema.
- Hyperosmolar fluids must be avoided.
- Diuretics, either loop or osmotic, can be used in certain situations. Cerebral oedema formation is reduced as right atrial pressure is lowered. As the diuresis will produce a negative fluid balance, it is important to avoid jeopardising the circulation. Therefore, they should only be used in consultation with a neurosurgeon, intensivist or neurologist. Mannitol 0.5–1 g/kg is given to patients with signs of cerebral oedema and raised ICP. It is thought to exert its effects initially by increasing circulating blood volume and reducing blood viscosity (improving microcirculatory flow and oxygen delivery) and then reducing brain water by its osmotic action.
- *Corticosteroids*: These are commonly used in less urgent situations. Dexamethasone 4 mg 6 hourly may produce symptomatic relief by reducing tumour associated oedema. They have not been shown to be of benefit in any other situations except in pneumococcal meningitis where dexamethasone (give before the antibiotics) improved outcome. However, beware that dexamethasone reduces the concentration of vancomycin (the antibiotic of choice for penicillin resistant pneumococci).
- *Seizures*: Prolonged seizures are associated with brain damage. Therefore, they should be controlled rapidly. Lorazepam 4 mg intravenously should be used, as it has better ability to control seizures than diazepam. The dose can be repeated. If this is unsuccessful, then phenytoin, 15 mg/kg diluted in 0.9% saline, should be infused over 30 min. Fosphenytoin is a water-soluble pro-drug that is converted into phenytoin by non-specific phosphatases. In comparison with phenytoin, it can be infused faster, causes phlebitis and is soluble in dextrose. The patient should have ECG monitoring, as too rapid an infusion of phenytoin can cause hypotension, bradycardia and asystole. If phenytoin fails to control the seizures, an anaesthetic induction agent should be used and the patient anaesthetised and ventilated as necessary.
- *Temperature control*: Hyperthermia is detrimental to patients with intracerebral pathology. An elevated temperature increases metabolism and therefore substrate requirements, i.e. oxygen and glucose.

Time Out 11.1

Take a 15-min break from reading.

a. List the mechanisms that maintain consciousness.

b. Describe briefly how you would assess brain stem function.

SPECIFIC CONDITIONS

Subarachnoid haemorrhage

Pathophysiology

The causes of subarachnoid haemorrhage are shown in the next box.

Causes of subarachnoid haemorrhage

Intracranial saccular aneurysm
Arteriovenous malformation
Others:
- **extension from intracranial haemorrhage**
- **intracranial venous thrombosis**
- **haemostatic failure**
- **vascular tumour**
- **drug use**

Conditions associated with intracranial saccular aneurysms

Polycystic kidney disease
Aortic stenosis
Infective endocarditis
Coarctation of the aorta
Thyromuscular dysplasia
Others:
- **Marfan's syndrome**
- **Ehler–Danlos syndrome**
- **Pseudoxanthoma elasticum**

The commonest cause is rupture of an intracranial saccular aneurysm. In contrast, only about 5% of patients bleed from arteriovenous malformations. Intracranial saccular aneurysms develop on medium-sized arteries at the base of the brain. The commonest sites are the distal internal carotid/posterior communicating artery and the anterior communicating artery complex. Multiple aneurysms are present in approximately 25% of patients. Aneurysms vary in size from a few millimetres to several centimetres in diameter. Whilst some are undoubtedly congenital, others develop during adult life, possibly as a consequence of atherosclerosis and hypertension. Conditions associated with intracranial saccular aneurysms are shown in the box above. The prevalence of unruptured aneurysms, as derived from prospective autopsy series, is in the region of 3/100 patients.

Assessment

Clinical features

The clinical picture is usually, but not always, dominated by an acute severe occipital headache. There may be a preceding history (usually over 1–2 weeks) of an acute transient severe headache, indicating a sentinel bleed. This can radiate over the head and, around, down into the neck, sometimes as far as the back or legs as blood tracks down the spinal cord. If the haemorrhage is extensive, the patient may become comatosed. If not, consciousness may be either lost transiently or impaired. Vomiting is common. Chemical meningitis (induced by blood) may take several hours to develop and focal signs are rare unless blood has extended into, or emanated from, the cerebral parenchyma. Occasionally, there is an oculomotor nerve palsy from a posterior communicating artery aneurysm. Arteriovenous

malformations may be diagnosed from the history because of recurrent unilateral migrainous headaches or very rarely on examination when an intracranial bruit is heard.

The patients are often irritable, confused and drowsy for several days. Headache may persist for weeks. Fundoscopy may reveal subhyaloid haemorrhages, which are believed to follow a rapid rise in intracranial pressure at the onset of intracranial haemorrhage.

Diagnosis

The diagnosis of subarachnoid haemorrhage is usually made on CT brain scan. However, a negative head CT does not exclude a subarachnoid haemorrhage. In these circumstances, a lumbar puncture (LP) is required. The three-tube test (i.e. a decreasing CSF red blood cell count in three consecutive tubes being indicative of a bloody tap) is unreliable. Only the absence of xanthrochromia, formally assessed by spectrophotometry, can be relied on to exclude subarachnoid haemorrhage following an LP. Xanthochromia refers to the yellowish discolouration of CSF supernatant from breakdown products of haemoglobin (oxyhaemoglobin and bilirubin). After a subarachnoid haemorrhage, red cells are gradually lysed within the CSF.

Released haemoglobin is metabolised to oxyhaemoglobin and bilirubin.. Oxyhaemoglobin can be detected within hours, but bilirubin requires up to 12 h to appear. Timing of an LP is therefore crucial and should be delayed until at least 12 h after the onset of symptoms. However, some neurosurgeons advocate immediate lumbar puncture irrespective of the time of the headache onset as blood may be detected. Thus, timing of the initial LP depends on local policy.

The CSF should be centrifuged and examined promptly so that red cells from a bloody tap do not undergo lysis *in vitro*. Xanthochromia is found in the CSF of all patients with subarachnoid haemorrhages from 12 h to 2 weeks after the haemorrhage, gradually disappearing thereafter.

The reliability of CT and lumbar puncture in the diagnosis of subarachnoid haemorrhage vary with time after the onset of headache as shown:

CT – detection of SAH		LP – presence of bilirubin	
<24 h	98%	1–12 h	Variable
24–48 h	86%	12 h–2 weeks	100%
2–5 days	76%	3 weeks	70%
5 days	58%	4 weeks	40%

The complications of subarachnoid haemorrhage are shown in the next box:

Occasionally, organised blood clot within the subarachnoid space may obstruct cerebrospinal fluid flow, causing acute hydrocephalus. This may lead to a deterioration in the patient's conscious level days or weeks after the haemorrhage. Other causes of neurological deterioration correspond to the complications of subarachnoid haemorrhage shown in the next box. Any change in neurological status is likely to warrant a CT scan to assess the presence of any treatable complication, e.g. hydrocephalus.

Complications of subarachnoid haemorrhage

Local recurrent haemorrhage
Cerebral oedema
Haemorrhage into brain parenchyma
Hydrocephalus
Secondary cerebral infarction due to vasospasm
Epileptic seizures
Hyponatraemia (inappropriate antidiuretic hormone production)
Central/uncal herniation
General hypoxaemia
Pulmonary embolus
Hypertension
Dehydration
Pneumonia/septicaemia
Hyperglycaemia

Management

The aim is to prevent secondary brain injury by following the structured approach. In addition, severe vasospasm may occur and this can be reduced by nimodipine (60 mg orally or NG every 4 h for 3 weeks). Other conditions that can arise include hypertension, cardiac dysrhythmias and neurogenic pulmonary oedema.

Do not forget that haemorrhage into the 4th ventricle can cause a transient rise in blood sugar. This is believed to be due to rapid autonomic nervous system discharge. The blood sugar will rapidly return to normal. Treatment with insulin could be fatal in precipitating hypoglycaemia.

Definitive management

Aneurysms detected on angiography may be treated either by endovascular coiling of the aneurysm with platinum wire coils, craniotomy and clipping of the neck of the aneurysm or, rarely, by stereotactic radiotherapy. The choice depends on individual expertise, the size, position and shape of the aneurysm. Coiling has become increasingly popular, due to its lower morbidity compared with craniotomy.

Outcome

Approximately 25% of patients die within 24 h of their subarachnoid haemorrhage. A further 25% die within the first month as a consequence of either recurrent haemorrhage or vasospasm induced infarction. The remainder survive for longer, but with an increased risk of rebleeding of approximately 2% per year.

Bacterial meningitis

Meningitis is an inflammatory condition of the lining of the brain and the ventricles. Causative organisms will influence the clinical presentation, management and outcome. Bacteria may reach the leptomeninges and produce meningitis in several ways:

- Bacterial seedlings by haematogenous spread.
- Local extension from contiguous extracerebral infections.
- Direct implantation of bacteria into the meninges.

Haematogenous spread

This type of meningitis is usually community acquired. The majority of cases are caused by *Streptococcus pneumoniae* or *meningococcus*. Of the remainder

(approximately 20%), *Listeria monocytogenes*, anaerobic gram negative bacilli (e.g. *Escherichia coli*), *Haemophilus influenzae* and *Staphylococcus aureus* are responsible. Risk factors for *Strep. pneumoniae* meningitis are shown in the box below.

Risk factors for *Strep. pneumoniae* meningitis

Hypogammaglobulinaemia (primary or secondary, e.g. chronic lymphatic leukaemia)
Sickle cell disease
Previous skull trauma with dural fistula

Listeria tends to affect people at the extremes of age, pregnant women and those patients who have prolonged immunosuppression from steroids or alkalating agents, e.g. azathioprine. Contaminated foods, in particular unpasteurised soft cheeses, pâté and poorly refrigerated precooked chicken have been implicated.

Local extension

Local extension from contiguous extracerebral infection (e.g. otitis media or sinusitis). Possible pathways for migration of pathogens from the middle ear to the meninges can be (1) via systemic route in the bloodstream (2) along fascial planes (e.g. posterior fossa) (3) via temporal bone fractures (4) through the oval or round window membranes (e.g. the labyrinths).

Direct implantation

Post-traumatic meningitis can follow trauma to the skull or spine. It often occurs early in the post-traumatic period due to a breach in the meninges; however, it may occur years later. If the infective organism is acquired in the community, then *Strep. pneumoniae* or *H. influenzae* are the likely responsible organisms. In contrast, hospital acquired infections are usually caused by the anaerobic gram negative bacilli (*E. coli, Klebsiella, Enterobacter* and *Pseudomonas* species).

Post-surgical meningitis may follow an operation on the head, neck or spine, or the insertion of cerebrospinal fluid drains and shunts. The majority of the post-surgical meningitides are caused by anaerobic gram negative bacilli (as described earlier).

Key point

It is important to remember that:
- **approximately 2% of meningitides will be culture negative**
- **with recurrent meningitis, cerebrospinal fluid leak, hypogammaglobulinaemia and complement deficiencies should be excluded**

Most infections affecting cerebrospinal fluid drains and shunts are hospital acquired, predominantly caused by coagulase negative staphylococci and *Staph. aureus*. Whilst it is easy to understand how organisms may contaminate the cerebrospinal fluid as a result of trauma or surgery, the precise mode of invasion in spontaneous meningitis is currently unknown.

All organisms induce inflammatory injury to the meninges. This increases the permeability of the blood–brain barrier and raises intracranial pressure due to cerebral oedema. This is due to a combination of:
- interstitial fluid accumulation

- communicating hydrocephalus due to decreased cerebrospinal fluid reabsorption
- cellular swelling
- vasculitis affecting the large vessels traversing the subarachnoid space.

A major consequence of increased intracranial pressure and vascular inflammation is decreased cerebral perfusion and therefore impaired delivery of oxygen and substrates. As the acute inflammatory response affects the pia and arachnoid mater, the cerebrospinal fluid contains numerous neutrophils and fibrin. Therefore, pus accumulates over the surface of the brain, in particular around its base, and can extend over the associated cranial nerves and spinal cord.

The early diagnosis of meningitis is difficult as many of the symptoms are non-specific and include malaise, fever, headache, myalgia and vomiting. As the disease progresses, the picture is dominated by irritability, severe headache and vomiting with the exception of meningococcal infection where diarrhoea is common. The classic non-blanching purpuric rash is common with meningococcal septicaemia, but occurs in only approximately 50% of those with meningococcal meningitis. The precise reason why some patients will develop meningococcal septicaemia rather than meningitis or vice versa is unknown.

Assessment

Clinical signs

The classic triad of fever, headaches and neck stiffness is usually present in 85% of cases. Limitation of neck movement is obvious, but meningitis is unlikely if the patient can shake their head or place their chin on their chest. Meningism is best elicited by passive flexion of the neck when the patient is supine. Confirmatory evidence may be obtained from Kernig's test, where the lower limb is flexed at the hip and the knee gradually extended. Resistance to this manoeuvre by contraction of the hamstrings is indicative of meningeal irritation. In contrast, Brudzinski's test is performed with the patient seated with their legs straight. Flexion of the neck in the presence of meningeal irritation will be associated with flexion of the hips and knees. Marked meningeal irritation can manifest as opisthotonus, i.e. the neck and back fully extended. **Meningism may be absent in patients who are either immunosuppressed or deeply comatosed.** Herpes labialis is commonly seen in **all** forms of bacterial meningitis.

Physical examination must exclude primary sites of infection, in particular otitis media, sinusitis, mastoiditis and pneumonia. Watery rhinorrhoea or otorrhoea should be collected and tested for glucose. Rhinorrhea or otorrhea fluid can also be tested for beta-transferrin, but the results will probably not be available in a useful timeframe, depending on local laboratory resources. Beta-transferrin is a protein that is produced by neuraminidase activity in the brain and is unique to CSF and perilymph fluid. Under these circumstances, a basal skull fracture must be excluded. The other clinical signs in the box below are not always present.

Clinical signs of a basal skull fracture

Bruising over mastoid process – Battle's sign
Otorrhoea – CSF ± blood
Rhinorrhoea – CSF ± blood
Periorbital bruising (panda or racoon eyes)
Subhyaloid haemorrhage

As the disease progresses, cranial nerves may become involved as they cross the inflamed basal meninges – commonly II, III, VI, VII and VIII. Papilloedema means that either cerebral oedema, hydrocephalus, a subdural effusion or empyema is contributing to the development of intracranial hypertension. Headache, vomiting, fever and decreasing level of consciousness usually dominate the clinical picture. Patients with infected shunts may present as described earlier. These devices can become infected in the cranium, the venous circulation or the peritoneal cavity, where they may produce symptoms akin to meningitis, right-sided infective endocarditis and peritonitis, respectively.

Diagnosis

The diagnosis is made clinically and often confirmed by lumbar puncture, provided there are no contraindications. In the majority of patients, the cerebrospinal fluid features of bacterial meningitis, are:

- raised white cell count (>100 white blood cells/ml), the majority of which are neutrophils
- cerebrospinal fluid glucose less than 40 mg/dl
- cerebrospinal fluid protein elevated
- gram staining of the cerebrospinal fluid will reveal organisms in over 50% of cases (see box below).

Gram staining of cerebrospinal fluid in pyogenic bacterial meningitis

Appearance	Probable organism
Gram positive cocci	*Strep. pneumoniae*, *Staph. aureus*
Gram negative cocci	*N. meningitidis*
Gram positive rods	*L. monocytogenes*
Gram negative rods	*H. influenzae*, *Enterobacter*

The causes of a predominant lymphocyte count are listed in the box below:

Meningitis with high cerebrospinal fluid lymphocyte count

Early or partially treated cryogenic bacterial infection
Tuberculosis, leptospirosis, brucellosis, syphilis, *Listeria*
Viral infection
Fungal infection, e.g. *Cryptococcus*
Parameningeal infection – intracerebral abscess or subdural empyema
Neoplastic infiltration

Specific treatment

Antibiotic therapy must be started immediately, as bacterial meningitis progresses rapidly and has a high mortality. The following are appropriate antibiotic agents. You should always refer to local hospital antibiotic policies.

Suspected meningococcal meningitis/septicaemia.
The patient should be given an immediate dose of ceftriaxone 2 g IV or benzylpenicillin 2.4 g qds IV.

Suspected meningitis

In adults, spontaneous meningitis is usually caused by *Strep. pneumoniae* or *Neisseria meningitidis*.

There is an increased risk of *L. monocytogenes* and infections caused by anaerobic gram negative bacilli, e.g. Escherichia coli in patients with hospital acquired meningitis. The recommended first line management is cefotaxime 2 g tds IV.

Remember the benefits of IV dexamethasone (10 mg qds for 4 days) in patients with pneumococcal meningitis (see page 168)

Postsurgical meningitis

This is usually caused by hospital acquired, multiresistant organisms. Often, specific hospital antibiotic policies exist; if not, an initial choice is ceftazidine 2 g every 8 h as this will also provide antipseudomonal cover. Either ceftazidine or ceftriaxone is a good first line antibiotic for patients who have acute infections of either shunts or drains, before they are removed.

Outcome

Mortality rates vary considerably depending on the study and type of organism. The overall mortality ranges from approximately 10% for *N. meningitidis* and *H. influenzae* to over 20% for *L. monocytogenes* and *Strep. pneumoniae meningitis*.

Mortality is much greater in the very young; the elderly, and patients with pre-existing debilitating illnesses. Furthermore, progression from consciousness through to confusion and then coma is associated with an increased mortality. Complications are not uncommon and are listed in the box below.

Complications of meningitis

Raised intracranial pressure
Seizures
Hyponatraemia
Venous sinus thrombosis
Cranial nerve deficit
Hydrocephalus
Cerebral infarcts
Cerebritis and abscess
Subdural effusions and empyema
Ventriculitis
Cerebral oedema

Tuberculous meningitis

There has been an increased incidence of tuberculosis in many parts of the world, related to the human immunodeficiency viral infection. These patients have a high risk of meningeal involvement. Other high risk groups include immigrants from Pakistan, India, Africa and the West Indies, alcoholics, intravenous drug users, immunocompromised patients and those with previous pulmonary tuberculosis.

Pathophysiology

Infection spreads from the primary lesion, or site of chronic infection, through the blood stream to the brain and meninges, where microtubercles are formed.

These rupture and discharge tubercular protein and mycobacteria into the subarachnoid space, inciting an inflammatory response. Many patients develop miliary tuberculosis at this stage because of haematogenous spread. As with bacterial meningitis, the base of brain and associated cranial nerves can be affected. Tuberculous meningitis is also associated with endarteritis. This can produce ischaemia/infarction of superficial cortical areas, internal capsule, basal ganglia and brain stem.

Assessment

Symptoms

The onset is usually subacute with two to eight weeks of non-specific prodromal symptoms, including malaise, irritability, lethargy, headache and vomiting.

Clinical signs

Meningeal irritation and cranial nerve damage (most often involving VI, but also II, IV and VII) are common, as is papilloedema. Raised intracranial pressure usually occurs because of obstruction of cerebrospinal fluid circulation, in particular, through the basal cisterns. The neurological features associated with raised intracranial pressure and hydrocephalus have been described earlier. The development of focal neurological signs, however, does not always imply raised intracranial pressure as these patients are prone to arteritis and hence cerebral infarction. Inappropriate antidiuretic hormone secretion is also common and may precipitate or exacerbate unconsciousness.

British Medical Research Council staging for disease severity is:

Stage I – early non-specific features, including apathy, irritability, headache, fever, anorexia and vomiting without alteration of conscious level.

Stage II – altered conscious level, without delirium or coma, but with minor neurological signs. Signs of meningism are present, with cranial nerve palsies or involuntary movements.

Stage III – advanced state with stupor or coma, severe neurological deficits, seizures, abnormal posturing and/or abnormal movements.

The prognosis is related directly to the clinical stage at diagnosis.

Diagnosis

The cerebrospinal fluid is clear or slightly turbid, with a white cell count less than 500 cells/ml. This is composed of both lymphocytes and neutrophils, in varying proportions. The cerebrospinal fluid glucose is low and the protein concentration is elevated. Tubercle bacilli are rarely seen on cerebrospinal fluid microscopy; however, centrifugation of the sample can increase the diagnostic yield. The most sensitive and specific test uses the polymerase chain reaction (PCR) to detect the *Mycobacterium tuberculosis* genome.

Treatment

This comprises combination chemotherapy with isoniazid (300 mg), rifampicin (600 mg) and pyrazinamide (1500 mg) for 12 months. Streptomycin can also be added for the first two months. Para-aminosalicylic acid should not be used, because it does not enter the cerebrospinal fluid. In view of the numerous complications and morbidity and mortality, specialist advice should be sought early.

Outcome

Prognosis is related to the clinical state at diagnosis. Mortality is still high, at approximately 25%, irrespective of whether patients have coexistent human

immunodeficiency virus. Permanent sequelae occur in approximately 25% of survivors, ranging from cranial nerve deficit (including blindness) to hemiparesis and intellectual impairment.

Summary of the CSF findings in meningitis are shown in Table 11.5.

Encephalitis and viral meningitis

There is considerable geographical variation in the type of virus causing encephalitis. In the UK, however, the commonest diagnosed cause of encephalitis is mumps. Other causes are shown in the box below.

Causes of viral encephalitis

Mumps
Echo virus
Coxsackie virus
Herpes simplex
Herpes zoster
Epstein – Barr virus
Adenovirus
Enterovirus

Many of these infections occur in seasonal peaks or epidemics; e.g. mumps encephalitis is common in the late winter or early spring whilst enterovirus infections occur in summer and early autumn. Other viral infections, in particular herpes simplex encephalitis, are sporadic. Although viral infections affect all age groups, they are most frequent and severe in children, the elderly and those who

Table 11.5 Summary of CSF findings in meningitis

Agent	Opening pressure (mm H_2O)	WBC count (cells/mm^3)	Glucose (mmol/l)	Protein (g/l)	Microbiology
Normal values	80–200	0–5; lymphocytes	60% of blood glucose	0.15–0.40	Negative
Bacterial meningitis	200–300	100–105; >80% PMNs	Very low	< 3	Specific pathogen demonstrated in 60% of gram stains and 80% of cultures
Viral meningitis	90–200	10–2000; lymphocytes	Normal, reduced mumps	< 1.5	Viral isolation, PCR
Tuberculous meningitis	180–300	Up to 4000; lymphocytes	Reduced, 1–4	1–6	Acid-fast bacillus stain, culture, PCR
Aseptic meningitis	90–200	10–300; lymphocytes	Normal	Normal but may be slightly elevated	Negative

PMN, polymorphonuclear lymphocyte; PCR, polymerase chain reaction.

have decreased T-cell immunity, e.g. Hodgkin's disease. Whilst herpes simplex encephalitis affects all age groups, it shows distinct peaks in those patients aged either between 5 and 30 years or greater than 50 years.

Pathophysiology

Most viral infections reach the central nervous system from the primary site of infection, via the blood stream. Nervous system damage is a consequence of direct invasion and immunological reaction. These processes culminate in:

- destruction and phagocytosis of neurones
- inflammatory oedema
- vascular lesions
- demyelination.

Characteristically, viral encephalitides cause lymphocytic infiltration of the meninges. Other features include perivascular cuffing of lymphocytes, plasma cells and histiocytes within the cortex and white matter as well as proliferation of microglia. This results in Neuronal degeneration and demyelination.

Herpes simplex encephalitis has characteristic features, in particular gross oedema, severe haemorrhage and necrotising encephalitis. These features are often asymmetrical and localised to the temporal lobe or, to a lesser extent, the frontal lobe. Demyelination is rare. The unique localisation of herpes simplex encephalitis has not been satisfactorily explained as yet.

Assessment

Clinical features

The symptom profile is similar to that of meningitis, dominated by headache, vomiting, fever and malaise.

Clinical signs

A wide spectrum of clinical signs includes confusion, convulsions, coma, focal neurological signs, features of raised intracranial pressure and psychiatric manifestations.

However, specific symptoms may arise as herpes simplex encephalitis involves primarily the temporal and frontal cortex. These include gustatory and olfactory hallucinations, amnesia, expressive dysphasia, temporal lobe seizures, anosmia and behavioural abnormalities. Cerebral oedema is common with herpes simplex encephalitis and untreated patients usually lapse into coma towards the end of the first week.

Diagnosis

The aim is to demonstrate a specific viral agent, especially herpes simplex, or exclude potentially treatable causes. Provided there are no contraindications, as described earlier, a lumbar puncture is needed. The cerebrospinal fluid pressure is usually increased, especially in herpes simplex encephalitis (related to the intense cerebral oedema) unless it is early in the evolution of the illness. There is often a marked increase in white cells, with lymphocytes and other mononuclear cells predominating. Furthermore, the cerebrospinal fluid may contain erythrocytes or be xanthochromic if there is a haemorrhagic element to the encephalitis, as with herpes simplex. Protein concentration is usually increased in excess of 50 mg/dl, with an increase in the proportion of immunoglobulin G (IgG). A prominent monoclonal IgG band will be seen in the cerebrospinal fluid due to *de novo* synthesis of IgG combined with leakage of IgG from the serum. The cerebrospinal fluid glucose is usually normal.

A specific virus can be found in the majority of patients, from either a throat swab or samples of stool, cerebrospinal fluid and blood. CSF PCR for Herpes Simplex viral DNA is 100% specific and 75–98% sensitive within the first 24–45 h. Magnetic resonance imaging has provided greater detail about the structural damage in patients who have encephalitis. Further supporting evidence may be provided by an EEG that shows irregular activity over the affected area.

Specific treatment

Aciclovir (10 mg/kg every 8 h for 14–21 days), a nucleoside analogue, is an effective treatment for herpes simplex encephalitis. It has the advantage that it is only taken up by infected cells and is therefore non-toxic to normal uninfected cells. If the diagnosis is suspected clinically, then treatment should be started immediately as untreated HSE has a mortality rate of 50–75%. Corticosteroids can be used in an attempt to combat cerebral oedema, but there is no convincing evidence of any benefit.

Often, however, the precise initial diagnosis is in doubt hence patients are treated with both aciclovir and antibiotics until definitive results are available.

Outcome

Neurological sequelae are common in patients following herpes simplex encephalitis and include mental retardation, amnesia, expressive aphasia, hemiparesis, ataxia and recurrent seizures, along with various behavioural and personality disturbances.

Aseptic meningitis

This is a common, rarely fatal condition usually caused by certain viruses. Less commonly it results from nonviral infections (e.g. partially treated bacterial meningitis; TB; fungi and parasites) and many noninfectious causes (e.g. drug reactions; CT disease). It occurs in individuals of all ages, although it is more common in children, especially during summer.

Clinical symptoms vary – typically there is headache and fever which are mild and go without treatment. However some develop full-blown life-threatening meningitis.

Treatment

As encephalitis. Subsequent changes depend on confirmation of the cause.

Cerebral malaria

Malaria remains the most important human parasitic disease globally. *Plasmodium falciparum* is the only malarial parasite that causes cerebral pathology but this is the predominant species in the highly endemic areas of Africa, New Guinea and Haiti.

Infection in man is acquired from either the female anopheles mosquito, which inoculates parasites into the human blood stream, or by transfusion of blood containing the parasite. The parasite, at this stage referred to as a sporozoite, enters hepatic parenchymal cells. *Plasmodium falciparum* does not have a dormant phase within the liver so relapses do not occur. After a period of 6–8 days, mature forms (merozoites) are liberated into the blood stream. Here they attach to and invade circulating erythrocytes. Parasites undergo many morphological changes in the erythrocytes to eventually produce shizonts containing daughter erythrocytic merozoites.

These are liberated by red cell lysis and immediately invade uninfected erythrocytes, resulting in a cycle of invasion and multiplication. The intraerythrocytic division is relatively regular, as is red cell lysis and merozoite release. These processes and the inflammatory components they provoke (e.g. cytokines) are responsible for the regular attacks of fever that occur at approximately the same time of day for the duration of the infection.

Pathophysiology

Plasmodium falciparum, in contrast to the other three forms of human malaria (*P. ovale, P. malariae, P. vivax*), affects the brain as well as other tissues. This unique difference is attributed to the fact the mature parasites adhere to specific endothelial receptors, in particular, on the venule. As a consequence, partial occlusion of small vessels occurs, which reduces perfusion. The resulting tissue anoxia and damage is exacerbated by red cells impacting on the parasites adhering to the vascular endothelium. Consequently areas of the brain will be deprived of oxygen and appropriate substrates, in particular glucose.

Assessment

Clinical features

Prodromal symptoms often predominate and include malaise, headache, myalgia, anorexia and mild fever. These are present for several days before the first rigor. This typically starts with the patient feeling cold and apprehensive. Shivering rapidly evolves into a rigor lasting for 1 h associated with vomiting, throbbing headache, palpitations, breathlessness and fainting. Culminating in a drenching sweat. The whole episode lasts for approximately 8–12 h, after which the exhausted patient sleeps. A high irregular continuous fever is not uncommon in a patient with falciparum malaria. In addition, generalised seizures, confusion, delirium, irritability and loss of consciousness may occur. Mild meningism can be present but neck stiffness, photophobia and papilloedema are rare. A conjugate gaze palsy is common, but pupillary, corneal and oculocephalic reflexes are normal. Muscle tone is increased symmetrically, knee reflexes are generally brisk and both plantar responses are extensor. Furthermore, extensor posturing is common and can be associated with sustained gaze. Other clinical features are shown in the box.

Non-neurological features associated with cerebral malaria

Anaemia
Spontaneous bleeding from the gastrointestinal tract
Jaundice
Hypoglycaemia
Shock
Oliguria
Acute renal failure
Pulmonary oedema

Diagnosis

Malaria should be considered in the differential diagnosis of any acute febrile illness until it can be excluded by definite lack of exposure, repeated examination of blood smears or following a therapeutic trial of antimalarial chemotherapy.

Examination of at least three thick and thin blood films is required to exclude the diagnosis.

Other techniques include:

- rapid diagnosis tests which use a finger prick blood sample to give results within 15 min. These tests are ideal in areas where microscope facilities and/or diagnostic expertise are limited.
- polymerase chain reaction which detects either DNA or mRNA from particular plasmodium species.

These tests have lower specificities and sensitivities. The gold standard remains thick and thin film examination.

Key point

Do not dismiss the possibility of malaria in patients who have taken prophylactic drugs, as protection is never complete

Absence of parasites in peripheral blood smears may indicate partial antimalarial treatment or sequestration in deep vascular beds. Treatment must be started and the diagnosis may be made on bone marrow aspirate.

If there are no contraindications, lumbar puncture must be done because it is important to exclude other treatable encephalopathies. The cerebrospinal fluid will show approximately 15 lymphocytes/ml, with increased protein and normal glucose, unless the patient is hypoglycaemic.

Specific therapy

Quinine is the drug of choice. This should be given by an intravenous infusion to patients who are seriously ill or unable to swallow tablets.

- Loading dose 20 mg/kg of quinine salt diluted in 5% dextrose over 4 h.
- Maintenance dose 10 mg/kg of quinine salt given over 4 h by intravenous infusion every 8–12 h until the patient can swallow tablets to complete a 7-day course. If patients require more than 48 h of parenteral therapy, the maintenance dose should be halved to 5 mg/kg.

Note that quinine can induce hypoglycaemia as a result of islet cell stimulation. This may be combined with hypoglycaemia due to extensive hyperparasitaemia. Thus, regular monitoring of blood glucose is necessary. Contraindications to quinine therapy are shown in the box below.

Contraindications to quinine therapy

Hypersensitivity to quinine
Concurrent use of cimetidine, amiodarone or digoxin
Therapeutic administration of mefloquine within the previous 14 days
Resistant *Plasmodium falciparum*

Side effects from quinine are rare and usually follow rapid intravenous injection (see box below). Thus, careful monitoring of the infusion speed is required.

Side effects following quinine administration

Cardiovascular	**Sinus arrest, junctional rhythms, arteriovenous block, ventricular tachycardia/fibrillation, sudden death, prolongation of QT interval**
Neurological	**Visual disturbances, partial deafness, headache, tinnitus, myopathy**
Haematological	**Thrombocytopenia, haemolytic anaemia**
Endocrine	**Hypoglycaemia**

Any of the following regimes are appropriate for patients who are not seriously ill and can swallow tablets:
- quinine 600 mg three times a day for 7 days
- fansidar–three tablets as a single dose
- doxycycline 200 mg daily for at least 7 days.

Key point

Tetracyclines, sulfadoxine and pyrimethamine are contraindicated in pregnancy

The first line treatment for uncomplicated chloraquine resistant falciparum malaria (WHO recommended) is artemether combination regime (artemisinin 10–12 mg/kg total does over 3–5 days) with e.g. mefloquine. Avoid combinations with drugs that have a reduced effect due to resistance such as chloraquine or sulfadoxine-pyrimethamine.

Outcome

Mortality is approximately 10%, but varies according to the medical facilities available. Severe falciparum malaria can occur with the following conditions:
- impaired acquired immunity
- post splenectomy
- pregnancy
- immunosuppression.

Complications such as retinal haemorrhage, renal failure, hypoglycaemia, haemoglobinuria, metabolic acidaemia and pulmonary oedema carry a poor prognosis.

Intracranial abscess

These can be extradural, subdural or intracerebral. Occasionally, subdural and intracerebral abscesses may rupture into the subarachnoid space, resulting in meningitis.

Pathophysiology

Intracerebral abscess

These occur as a consequence of middle ear infection, frontal sinusitis and penetrating trauma to the head. Other causes include sepsis related to infective endocarditis, lung abscess and bronchiectasis. As most abscesses are related to disease affecting either the middle ear or sinuses, they tend to be found in the temporal lobes, cerebellum or frontal lobes. Not surprisingly, sepsis can be associated with multiple intracerebral abscesses.

Large intracerebral abscesses may rupture into the ventricular system producing ventriculitis.

Many of the organisms involved are described earlier and also under the section entitled subdural abscess/empyema.

Subdural abscess

This is often a sequel to an infection in the paranasal sinuses or middle ear. Other causes include meningitis and sepsis related to cyanotic congenital heart disease and lung abscesses. Penetrating trauma and intracranial surgery can also be implicated. Subdural abscesses may be extensive with pus extending over the surface of the brain. The most common organisms include *Strep. pneumoniae*, *Strept. milleri*, *Strept. pyogenes*, *Staph. aureus* and *Bacteroides* species along with *H. influenzae*.

Extradural abscess

As the dura mater is tightly adherent to the periosteum of the skull, epidural collections of pus are usually localised. They are related to either infections within the mastoid and nasal sinuses or focal osteomyelitis of the skull. Occasionally, infection may spread intracranially as described previously. This is more likely to occur in penetrating trauma to the skull or rarely following craniotomy. Common organisms include *Streptococcus, Staph. aureus,* Enterobacteriaceae, *Bacteroides* and many anaerobic species.

Assessment

Clinical features

The clinical features will depend on the number, site and extent of the lesions, as well as the impact on surrounding structures.

- *Intracerebral* abscess can present as headache, vomiting, impaired consciousness, hemiparesis and seizures. There may also be features to suggest either a pulmonary or cardiac primary focus of infection.
- *Subdural* abscess is often associated with severe headache, pyrexia, confusion, seizures and coma. A contralateral hemiparesis can be present. There may be evidence of either mastoiditis, frontal sinusitis or a scalp infection.
- *Extradural* abscess is often difficult to diagnose clinically, but may present with a localised headache in association with mastoiditis and sinusitis.

Diagnosis

This is usually made on either CT scan or magnetic resonance imaging. Lumbar puncture is rarely needed and is contraindicated when raised intracranial pressure is present. Usually the results are non-specific, consisting of an elevated protein level, pleocytosis with variable neutrophil count, a normal glucose level and sterile cultures. A lumbar puncture is mostly of value to rule out other disease processes, especially bacterial meningitis

Specific management

Neurosurgical opinion is necessary. Most supratentorial abscesses can be aspirated via a burr hole but subdural collections usually require evacuation through a craniotomy. Small abscesses are usually treated with antibiotics.

Outcome

The mortality is 10–20%. However, one third of survivors will have persistent epilepsy, in particular, as a sequel to temporal lobe or subdural abscesses.

Intracranial haematoma

Classification is identical to 'intracranial abscess', i.e. extradural, subdural and intracerebral.

Intracerebral haematoma

Occasionally, spontaneous haemorrhage can produce a haematoma within the substance of the brain. This is the result of rupture of small arteries affected by lipohyaline degeneration (Charcot–Bouchard aneurysm). It typically occurs in patients with hypertension and occurs at well-defined sites – basal ganglia, pons, cerebellum and subcortical white matter. The site and extent of the lesion will determine the clinical findings. One such condition which deserves mention is a cerebellar haematoma, because surgical treatment can be life saving. The patient presents with acute occipital headache, dizziness, truncal ataxia and rapid reduction in consciousness.

Subdural haematoma

This may be acute in patients who are overanticoagulated or chronic in the elderly, epileptic or alcoholic. A chronic subdural haematoma is usually associated with a trivial injury that may go unnoticed by the patient. Haemorrhage is due to rupture of the small veins crossing the subdural space with blood forming a localised collection between the dura and arachnoid mater.

Absorption of fluid from the adjacent arachnoid space causes expansion of the blood clot. The onset of symptoms is insidious, with headache, often mental changes, drowsiness and vomiting. There may be mild hemiplegia, but raised intracranial pressure is not initially prominent.

Extradural haematoma

This usually follows a tear to the middle meningeal artery following a fracture in the temporoparietal region. In a third of cases there is a triad of symptoms: unconscious for a short time recovery (the lucid interval, where confusion is common) and then comat, minutes or hours later as the extradural haematoma raises the intracranial pressure. The other two thirds of patients are either unconscious from the initial impact or do not have an initial period of unconsciousness.

Diagnosis

In all cases, the diagnosis is confirmed by either a CT scan or magnetic resonance imaging. An extradural haematoma is 'egg-shaped' and a subdural haematoma is 'saucer-shaped' on CT scan.

Specific management

For all of these conditions, urgent neurosurgical consultation is required. It is important to realise that patients with such lesions can deteriorate very quickly. Regular monitoring is necessary, as is prevention of secondary brain injury.

Prognosis

The outcome for extradural haematoma is good provided there is no underlying brain damage and the haematoma is evacuated rapidly (less than 4 hours).

For subdural haematoma and intracranial haematoma the prognosis is worse as there is invariably primary brain damage. Factors affecting survival are:

- Age $\geq$60 years
- Presenting GCS $\leq$6 (poor conscious level), significant comorbidity on coagulation/antiplatelet therapy

- Dominant hemisphere, deep-seated clot
- Clot >50 cm^3

Intracranial tumours

These may be benign or malignant, primary or secondary. The clinical effects are related to the site and extent of the lesion(s) as well as the impact on neighbouring structures. An in-depth discussion on the different types of tumour is beyond the scope of this text. Furthermore, this will not influence the initial management.

Pathophysiology

The effect of any intracranial neoplastic lesion depends on the following:

- type of tumour
- growth rate
- site
- extent
- capacity to incite oedema formation in the adjacent brain tissue
- effect on neighbouring structures
- potential to obstruct flow of cerebrospinal fluid and blood.

The precise effects that these will have on intracranial pressure have been explained earlier.

Assessment

Clinical features

Patients with intracerebral tumours tend to present with either epilepsy, or focal neurological signs, or raised intracranial pressure or a combination of these features. Late onset epilepsy (patients over 25 years) should always raise the suspicion of an intracranial tumour. Focal neurological deficits will obviously be related to the site and extent of the tumour, as well as its effect on adjacent structures. The effects of raised intracranial pressure have been discussed earlier. The progressive development of clinical signs is the most significant factor in the diagnosis of intracerebral tumours.

Investigations

Imaging, either CT scan or MRI, is the investigation of choice.

Specific management

Dexamethasone (4 mg tds) can reduce oedema surrounding the tumour(s). Neurosurgical consultation is required.

Time Out 11.2

Check your knowledge acquisition by answering the following questions.

a. What is the commonest site for a saccular aneurysm?
b. List the two common causes of subarachnoid haemorrhage.
c. How long does it take for xanthochromia to develop?
d. What is the mortality in the first 24 h after a subarachnoid haemorrhage?
e. List the two common bacteria that cause spontaneous meningitis.
f. Which categories of patients are 'high risk' for TB meningitis (a clue – six major groups)?
g. List the characteristic features of herpes simplex encephalitis.
h. In which patients would you consider a diagnosis of cerebral malaria?
i. List the non-neurological features of *Plasmodium falciparum* infection.

j. List any differences between abscesses in the extradural, subdural and intracerebral locations
k. List any differences between haematomata in the extradural, subdural and intracerebral locations.

SUMMARY

- The patient with altered conscious level is a common medical problem.
- Prevent secondary brain injury by ensuring appropriate provision of supplemental oxygen and glucose.
- It is important that CT scanning is the critical first investigation providing all immediately life-threatening problems have been treated and hypoglycaemia excluded. If either meningitis or encephalitis is suspected treatment should be given before investigations are done.
- Late onset epilepsy may indicate an intracranial tumour.
- Early liaison with specialist colleagues in microbiology, neurology or neurosurgery is important.

Advanced Life Support Group

CHAPTER 12
The 'collapsed' patient

OBJECTIVES

After reading this chapter you will be able to:

- describe the structured approach to the collapsed patient
- understand the pathophysiology of collapse
- discuss the causes and initial investigation of transient loss of consciousness
- describe some of the common conditions that present as 'collapse'.

INTRODUCTION

A wide variety of medical conditions present to hospital as 'collapse'. This term has different meaning to different groups of medical professionals and patients. To some it refers to any patient who has been found on the floor, or less responsive than normal. To others it refers to a transient loss of consciousness with return to pre-existing neurological function (more correctly called syncope). To yet others it may include near-syncope or dizziness. Regardless of the cause – which may not be known even after investigation – the same structured approach is applicable.

PRIMARY ASSESSMENT AND RESUSCITATION

An overview of this has been described in Chapter 3. Specific details relating to the collapsed patient will now be considered.

A – airway (and cervical spine)

Assess and clear the airway as described in Chapter 4. Patients with syncope are unlikely to have airway problems. Those with other causes of collapse especially stroke and epilepsy have significant potential for airway compromise due to position, loss of or increase in muscle tone, loss of protective reflexes and inability to swallow.

Trauma due to the collapse may compromise the airway due to bleeding or loose teeth. Although cervical spine injury is unusual in patients falling to the floor from their own height, it does occur, especially in the elderly and those with rheumatoid arthritis and ankylosing spondylitis. Remember the potential for this condition in patients found collapsed at the bottom of the stairs. Consider and treat for potential cervical spine injury if there is no clear history, especially in patients with signs of injury above the clavicles. Give supplementary oxygen (12–15 l/min via non-rebreathing mask with reservoir bag) titrated to oxygen saturation in all acutely ill patients (see Chapter 3).

B – breathing

Examine for evidence of a condition that may have either caused the collapse (e.g. pulmonary oedema) or more likely be a consequence (e.g. aspiration pneumonia in an unconscious patient).

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C – circulation

Seek features of shock and treat with intra-venous fluids for hypovolaemia. Institute cardiac rhythm monitoring and treat life-threatening arrhythmias according to UK and European resuscitation council guidelines. Other causes of shock and collapse, e.g. anaphylaxis are considered in Chapter 9.

D – disability

The aim of this brief examination is to identify and begin treatment for immediately life-threatening conditions such as hypoglycaemia, status epilepticus and meningitis. Assess:

- pupil size and reaction
- conscious level using Glasgow Coma Scale (GCS)
- for evidence of meningism
- for signs of seizure activity
- bedside glucose.

As neurological signs may develop and change, monitor GCS, pupillary response and glucose.

E – exposure

Quickly identify signs that may point to a life-threatening cause of collapse. Look for non-blanching purpuric rash, cutaneous opioid patches, evidence of illicit drug use, recent surgical scars, signs of deliberate self harm. Check the temperature. Remember that a patient who has collapsed may have spent several hours on the floor. Hence also consider the risk of rhabdomyolysis and pressure sores.

STROKE/TIA

Introduction

Stroke is a syndrome characterised by an acute onset of focal (at times global) loss of neurological function lasting more than 24 h (or causing earlier death) due to cerebrovascular disease. Therefore, it is a clinical diagnosis. Traditionally, a transient ischaemic attack (TIA) has been defined as a neurological deficit caused by focal brain ischaemia that completely resolves within 24 h. Most have a much shorter duration than this, and with increased use of magnetic resonance imaging (MRI) it has become clear that over half of the patients who present with TIA have evidence of infarction in the corresponding territory.

TRANSIENT ISCHAEMIC ATTACK

Score to predict of early stroke within 7 days (after TIA):

A – Age $\geq$ 60 years = 1 point
B – BP (SBP $\geq$ 140; DBP $\geq$ 90; Both) all = 1 point
C – Clinical sign – unilateral weakness 2 pts; speech 1 pt; other 0 pt
D2 – Duration $\geq$ 1 h = 2 pt; 10–59 min = 1 pt; < 10 min 0 pt
–Diabetes mellitus (1 point)

With TIA, it is important to assess the patient's risk of a subsequent stroke. One way is the National Institute of Health scoring system described in the next box. Those with a score of 3 or less can be referred to the TIA clinic within 2 weeks.

Patients scoring 4 or more or those in atrial fibrillation should be admitted for specialist assessment. This score can then be used to predict the risk of stroke.

A–D2 score	Risk of stroke within 2 days
6–7	8 fold
4–5	4 fold
<4	1 fold

Treatment includes one or more of the following:
- anti-platelet therapy
- anticoagulation
- endarterectomy.

If a vascular stenosis is detected and treated early (<2/7 ideally) – the chance of stroke is reduced by 30%.

If the patient is already on aspirin give dipyridamole. This combined tablet is called asasantin. Remember that dipyridamole cannot be used if the patient has either severe coronary artery disease or contra indications for aspirin use.

Stroke

Acute stroke affects about 2/1000 population per annum. This incidence increases steeply with increasing age (20/1000 in those over 85). Stroke is more common in men.

Mortality is high (20% within 30 days after a first stroke), as is prevalence of disability in survivors (about one third are dependent on others at one year). The risk of stroke after a first TIA is approximately 10% in the first 3 months – half of such strokes occurring in the first 48 h. The risk factors that make stroke more likely within a short interval are:
- age over 60
- diabetes mellitus
- weakness or speech impairment during the episode
- duration longer than 10 min
- occurrence despite aspirin
- probable cardiac embolic source.

Pathophysiology

The types of stroke are listed in the box

Types and causes of stroke

Cerebral infarction	**80%**
(large vessel disease 50%)	
(small vessel disease [lacunar] 25%)	
(cardiogenic embolism 20%)	
Primary intracerebral haemorrhage	**10%**
(hypertension 50%)	
Subarachnoid haemorrhage	**5%**
Unknown	**5%**

Atherosclerosis of the major vessels supplying the brain can precipitate a stroke by causing either embolisation from atherosclerotic plaques or major vessel occlusion. Small vessel disease, with occlusion of small penetrating arterioles, leads to small infarcts in the subcortical white matter, internal capsule and basal ganglia (lacunar infarcts). Atrial fibrillation, valvular heart disease, recent myocardial infarction and ventricular aneurysm can cause embolic strokes.

Intracerebral haemorrhage usually follows the sudden rupture of microaneurysms caused by hypertensive vascular disease, characteristically in the basal ganglia, brain stem and cerebellum.

Subarachnoid haemorrhage is commonly caused by a rupture of an aneurysm arising on one of the arteries at the base of the brain, but it may arise from an arteriovenous malformation. (See Chapters 11 and 14 for further details.)

The distinction between strokes in the internal carotid (anterior circulation) territory and those in the vertebrobasilar (posterior circulation) territory is not always easy on clinical grounds. Dysphasia or visual spatial apraxia indicates definite carotid distribution. In contrast, simultaneous bilateral weakness or sensory loss, cortical blindness, diplopia, vertigo, ataxia and dysphagia suggest vertebrobasilar distribution.

Lacunar strokes tend not to affect conscious level or cognitive function. They may cause pure motor stroke, pure sensory stroke, sensorimotor stroke, ataxic hemiparesis and, rarely, movement disorders such as hemiballismus or hemichorea.

Primary assessment and immediate treatment

In a patient presenting with stroke, it is essential to follow the structured approach previously described to optimise oxygenation and cerebral perfusion, and to limit secondary cerebral damage. Specific problems in the stroke patient include:

A – Airway may not be maintained. Clear and secure if necessary.

B – Respiratory drive may be depressed.

C – Cardiovascular compromise may have precipitated the stroke. Treat hypotension and tachycardias/bradycardias appropriately.

D – Check glucose. Hypoglycaemia may present with focal signs or depressed conscious level. Look for evidence of seizure activity.

E – Check temperature. Both hypothermia and hyperthermia may complicate stroke.

Secondary assessment and emergency treatment

Phrased history

An account of the onset of symptoms must be obtained from the patient or relative. A rapid onset of a focal neurological deficit is characteristic of a stroke (minutes or hours). A history of sudden onset of a severe headache associated with neck stiffness suggests subarachnoid haemorrhage. In contrast, a slower onset of symptoms is more likely to indicate other diagnoses, e.g. intracranial tumour or chronic subdural haematoma.

Note any vascular risk factors including history of transient ischaemic attacks, hypertension, atrial fibrillation, ischaemic heart disease, cigarette smoking, diabetes mellitus, hyperlipidaemia and family history.

Examination

A full neurological assessment will help localise the lesion and record the degree of disability.

One way of recognising stroke in the emergency room is shown in the next box:

ROSIER (Recognition of stroke in the ER) score

History	
LOC	**−1**
Convulsion	**−1**
Examination	
Face, arm, leg	**+1 for each affected**
Speech defect	**+1**
Visual field defect	**+1**

Negative score – consider stroke mimic

For scores over zero: PPV for stroke – 90%; NPV for stroke 88%

Examination of the fundi may reveal changes of raised intracranial pressure or show evidence of previously undiagnosed hypertension or diabetes mellitus.

The cardiovascular examination should assess:

- heart rhythm (in particular atrial fibrillation)
- blood pressure, taken in both arms (subclavian steal, aortic dissection)
- the presence of any valvular heart disease
- reduced carotid blood flow (pulse pressure/bruit)
- peripheral pulses.

Listen carefully at the lung bases. Patients with swallowing difficulty are at risk of aspiration.

Look for evidence of development of pressure sores or rhabdomyolysis in patients who may have been on the floor for some time.

Investigations

CT scan

This is essential to establish whether the underlying pathology is infarction or haemorrhage and to exclude possible cerebral tumour or subdural haematoma.

Key point

It is not possible to distinguish cerebral infarction from cerebral haemorrhage on clinical grounds

CT scanning has several advantages over MRI in the acute stage and is the current method of choice as it is:

- more widely available
- cheaper
- more sensitive at identifying haemorrhage in the early stages
- easier to monitor the patient during a CT scan.

However, there are disadvantages to CT scanning.

- It will not identify an infarction in the first few (possibly up to 48) hours after the onset of symptoms.
- It has limited ability to show vascular lesions in the brain stem and cerebellum and small ischaemic infarcts deep in the cerebral hemispheres.

MRI modalities such as diffusion weighted imaging can detect ischaemia within minutes of onset.

Other investigations

- ECG is essential to demonstrate rhythm disturbance, particularly atrial fibrillation, and evidence of ischaemic, hypertensive or valvular heart disease.
- Chest X-ray may reveal features consistent with a potential cardiogenic source of emboli as well as evidence of aspiration.
- Full blood count and clotting will exclude polycythaemia, thrombocytosis and clotting disorders.
- Plasma viscosity/ erythrocyte sedimentation rate as a screen for infection, vasculitis.
- Blood glucose, to exclude hypoglycaemia and diabetes mellitus.
- Urea and electrolytes, to identify:
  - electrolyte disturbances in patients on diuretics
  - evidence of renal impairment in patients with hypertension
  - hyponatraemia (a rare cause of focal neurological deficit).
- Fasting lipids, in all but the very elderly (although levels in acute phase of a stroke may not reflect premorbid profile).
- Syphilis serology, to identify meningovascular syphilis as a treatable, but rare, cause of cerebral ischaemia.

Management

The immediately life-threatening problems will have been identified and treated as part of the primary assessment. As soon as the CT has shown that it is not intracranial haemorrhage the stroke team should be informed and thrombolysis started.

Other important considerations at this stage include the following:

Antiplatelet therapy: In acute stroke, aspirin should be started as soon as the CT scan confirms there is no cerebral bleed. A starting dose of 150–300 mg daily should be given and continued until decisions have been made about secondary prevention. Depending on local policy other agents such as clopidogrel may be used instead, especially in patients where there are contraindications to aspirin.

Anticoagulant therapy: There is no evidence at present to support the use of anticoagulants in **acute** stroke, even for patients in atrial fibrillation. However, all patients in atrial fibrillation will require anticoagulation, provided there are no contraindications.

Thrombolysis: Intravenous thrombolytic therapy is a potentially effective treatment of acute thrombotic stroke, to provide early reperfusion of ischaemic cerebral tissue and limitation of infarct size. However, all thrombolytic drugs need to be given early after the onset of symptoms (probably within 3 h) and involve a risk of cerebral haemorrhage. Results of trials so far suggest an increase in the proportion of patients making a good recovery by six months, but a substantial increase in cerebral haemorrhage within the first two weeks.

The National Institute for Neurological Disorders (NINDS) has set down time goals for the management of acute stroke in the context of thrombolysis as follows:

- Clinical assessment within 10 min of Emergency Department (ED) arrival.
- CT scan performed within 25 min of ED arrival.
- CT scan interpreted within 45 min of ED arrival.
- Initiation of fibrinolytic therapy (if indicated) within 1 h of ED arrival and 3 h of onset.
- Door-to-admission time of 3 h.

In view of the potential risks and service implications, the most recent advice from the Intercollegiate Stroke Working Party (Royal College of Physicians, 2006) is that hospitals offering thrombolysis for acute stroke, outside a clinical trial, should have registered with the UK Safe Implementation of Thrombolysis in Stroke Monitoring Study programme. The recommendations on the use of thrombolysis in acute stroke have been produced by the National Institute of Clinical Excellence in July 2008.

Cardiovascular: Hypotension must be corrected (hypovolaemic hypotension may occur due to inadequate oral fluid intake). Hypertension must be managed cautiously. Some elevation in blood pressure is often seen with an acute stroke. Too drastic a reduction in blood pressure may reduce cerebral blood flow in the area around the infarct, causing extension of the stroke. Mild to moderate elevations in blood pressure do not require treatment unless they are maintained for several days after the acute event. If the diastolic blood pressure is persistently above 120 mm Hg, the blood pressure must be lowered cautiously, using oral agents, e.g. sublingual nifedipine. Avoid intramuscular preparations which may cause precipitous falls in blood pressure.

Respiratory: Patients with swallowing difficulty are at risk of bronchopulmonary aspiration. Monitor carefully for evidence of aspiration in the early stages and treat accordingly. An early review by the speech and language therapy team is beneficial.

Metabolic: Blood sugar must be maintained within normal limits. Hyperglycaemia is harmful. Monitor fluid and electrolyte balance, in view of probable inadequate oral intake in the early stages.

Surgery: Neurosurgery may need to be considered for some cases of intracerebral haemorrhage. Evacuation of a cerebellar haematoma may be life saving and result in good long-term recovery. Evacuation of supratentorial haematomas may also be life saving but survivors usually have greater disability.

Other investigations and treatments will need to be considered later to reduce the risk of recurrent stroke, but these are beyond the scope of this text.

Summary

- Stroke is common.
- Mortality is high.
- Prevalence of dependency in survivors is high.
- The history and clinical assessment provide the diagnosis in the majority of patients, but a CT brain scan is necessary to exclude other pathology and to distinguish between thrombosis and haemorrhage.
- In the acute phase, the main aim is to optimise cerebral oxygen supply and ensure normal glucose, fluid and electrolyte balance.

- Aspirin is of benefit and should be started once cerebral thrombosis has been confirmed.
- Early thrombolysis in appropriate cases is essential to minimise further brain injury.

TRANSIENT COLLAPSE

Collapse is common. It accounts for about 3% of visits to the emergency department and between 1 and 6% of general medical admissions. Collapse may be an isolated episode or a recurrent problem.

Recurrent collapse is important because it:

- is common
- is disabling
- may cause serious injury to the patient or to others
- can indicate life-threatening underlying pathology.

Certain factors increase the likelihood of a serious cause for syncope, warranting hospital admission. These include abnormal ECG, shortness of breath in the history of the presenting complaint, systolic blood pressure less than 90 mm Hg and haematocrit less than 30%.

Cerebral function can be disturbed by interruption of blood supply, epilepsy or metabolic factors. Some causes are listed in the next box. Whatever the cause, the initial approach to the patient should be a primary assessment and appropriate resuscitation before moving on to the secondary assessment and a more detailed history.

The prevalence of the various causes depends on the population studied. Some recently published pooled data are summarised in the box. However, carotid sinus hypersensitivity may cause up to 47% of syncope in the elderly.

Causes of recurrent collapse – prevalence

Vasovagal syncope	**18%**
Arrhythmia	**14%**
Epilepsy	**10%**
Postural hypotension	**8%**
Situational syncope	**5%**
Organic heart disease	**4%**
Medications	**3%**
Psychiatric	**2%**
Carotid sinus hypersensitivity	**1%**
Unknown	**35%**

Pathophysiology

The causes and associated reasons for a transient disturbance of consciousness are summarised in the following box.

Causes of transient disturbances of consciousness

Reduction in cerebral blood flow:		
Generalised cerebral hypoperfusion (syncope)		
(i) Cardiac:		
Reduced cardiac output	**Myocardial ischaemia**	**Daily**
	Hypovolaemia	**Daily**
	Aortic stenosis	**Monthly**
	Hypertrophic obstructive cardiomyopathy (HOCM)	**Annually**
	Pulmonary hypertension	**Annually**
Reduced ventricular filling	**Arrhythmia**	**Daily**
	Pulmonary embolism	**Weekly**
	Atrial myxoma	**Only in exams**
(ii) Reflex mediated:		
Vasovagal		**Daily**
Situational (micturition, cough)		**Weekly**
Carotid sinus hypersensitivity		**Weekly**
(iii) Postural hypotension		**Daily**
Localised vascular disease		
Vertebrobasilar		**Weekly**
Transient ischaemic attack		**Annually**
Basilar artery migraine		**Daily**
Epilepsy		
Metabolic disturbances/drugs:		
Hypoxaemia		**Daily**
Hypoglycaemia		**Daily**
Hyperventilation		**Only in exams**

Syncope

Syncope is defined as a transient loss of consciousness associated with an acute reduction in cerebral blood flow. Although cerebral autoregulation compensates for minor changes in blood pressure, more severe reductions will cause a fall in cerebral perfusion pressure. This will lead to a loss of consciousness.

Syncope is the most common cause of recurrent loss of consciousness. The other main differential diagnosis to consider is epilepsy. The distinction is usually clear from the history. However, seizures can sometimes be precipitated by cerebral hypoperfusion due to a primary cardiac problem. It is not unusual to see abnormal jerking movements at the onset of loss of conciousness due to an arrhythymia that causes decreased cerebral perfusion (especially VT) and at the start of a witnessed cardiac arrest.

Postural hypotension is common – especially in the elderly – due to a combination of reduced baroreceptor sensitivity, excessive venous pooling and autonomic dysfunction. It is often exacerbated by drugs and dehydration.

In vasovagal syncope, venous pooling in the upright posture reduces venous return resulting in increased sympathetic activity. In response to the vigorous

contraction of the underfilled ventricles, stimulation of ventricular mechanoreceptors initiates a brain stem reflex. This causes profound hypotension due to a combination of vagal stimulation (causing bradycardia) and withdrawal of sympathetic stimulation (causing vasodilatation) – the Bezold–Jarisch reflex.

Situational syncope occurs when the parasympathetic nervous system is activated by a trigger such as micturition or coughing.

It is important to remember ectopic pregnancy as a cause of syncope in women of child bearing age. A normal pregnancy can also cause syncope (see Chapter 23 for further details). In UK emergency medicine practice it is considered negligent not to exclude this diagnosis.

Localised vascular disease

Any disorder of the cerebral blood vessels can result in reduced cerebral perfusion. Syncope in isolation is not typically a feature of transient ischaemic episodes. Loss of consciousness does not usually occur with a stroke in the carotid artery territory. A brain stem vascular episode may result in impaired consciousness but other symptoms usually occur, e.g. vertigo, diplopia and ataxia.

Metabolic causes

Metabolic causes of transient loss of consciousness are uncommon. Hypoglycaemia must not be forgotten. It is usually due to overtreatment of diabetes mellitus but may occur in other situations, e.g. cirrhosis, Addison's disease (adrenocortical failure), postgastrectomy and insulinoma.

Hyperventilation can cause a respiratory alkalosis, which rarely predisposes to syncope.

Chronic catecholamine oversecretion with phaeochromocytoma can be associated with postural hypotension.

Drugs

Drugs can cause collapse by:

- interfering with cardiac conduction (e.g. digoxin, β blockers, calcium channel blockers, amiodarone)
- causing postural hypotension (e.g. diuretics, antihypertensives, antidepressants, levodopa preparations).
- altering conscious level – alcohol
- disturbing electrolyte balance (diuretics, B2 agonists, glucocorticoids).

Assessment

The paroxysmal nature of the problem means that you are likely to see the patient between episodes of collapse, when primary assessment is likely to reveal no major problems. Secondary assessment with a careful history and physical examination will provide the diagnosis in the majority of patients.

History

It is important to obtain a history from the patient, and also a witness if available. The circumstances of the collapse may be relevant, e.g. cough or micturition syncope.

Vasovagal syncope is usually associated with a hot environment or stressful, emotional situations. Collapse associated with head turning may indicate carotid sinus hypersensitivity. Episodes associated with exertion suggest

mechanical limitation of cardiac output (aortic stenosis, Hypertrophic Obstructive Cardiomyopathy (HOCM)) or an exercise induced arrhythmia. Symptoms on prolonged standing suggest postural hypotension or vasovagal syncope.

Ask specifically about cardiovascular symptoms (palpitations, chest pain, breathlessness) and neurological symptoms (headache, weakness/parasthesiae, autonomic dysfunction). **The importance of an accurate drug history cannot be over-emphasised**. A family history of syncope or sudden death may be relevant. It is also important to establish the occupation, hobbies and driving status of the patient to enable you to advise them appropriately prior to discharge. Be aware of the DVLA guidelines on driving after episodes of collapse.

The distinction between epilepsy and syncope can be difficult. A witnessed tonic–clonic convulsion associated with tongue biting and incontinence is obviously helpful in making a diagnosis, but the story may not always be so clear.

- A patient with syncope usually reports symptoms of light-headedness, nausea, sweating or blurring of vision before consciousness is lost. In contrast, a generalised tonic–clonic seizure usually has minimal prodromal symptoms.
- In syncope the duration of unconsciousness is shorter than epilepsy (seconds versus minutes) and recovery is more rapid without the usual drowsy confused postictal period.
- Brief twitching may be seen with an episode of syncope but this is usually very transient.
- Pallor may be seen before the collapse. This is common with syncope, although it may be seen with epilepsy.

Key point

The distinction between epilepsy and syncope is important. A careful history from the patient and witnesses will clarify the situation in the majority of cases

Examination

Assess the pulse rate, rhythm and character. Measure the lying and standing blood pressure. A fall in systolic blood pressure of 20 mm Hg after 2 min standing is significant. Remember that postural hypotension may indicate serious pathology – it is found in hypovolaemia and in some patients with peritoneal irritation. Examine the precordium for evidence of structural heart disease, especially aortic stenosis or other causes of outflow obstruction. Listen for carotid bruits.

A thorough neurological assessment is essential. Look for patterns of signs including upper motor neurone features extrapyramidal pathology, cerebellar features, brain stem signs and evidence of peripheral neuropathy.

Remember to look for injuries relating to the collapse. If the skin is broken check the tetanus status.

Actively seek evidence of tongue biting and incontinence (patients may deny the latter).

Many physicians only examine the patient on the bed. After collapse it is important to ensure the patient is still able to walk: fractured pubic rami often do not cause symptoms until the patient is weight bearing.

Investigations

Further investigations will be guided by the history and clinical findings.

(a) Cardiological
- 12-lead ECG is needed for all patients with recurrent collapse looking for evidence of ischaemia, left ventricular hypertrophy or conduction abnormalities.
- 24-h ECG monitoring may be useful if there is a suspicion of paroxysmal rhythm disturbances, even though 12-lead ECG may be normal.
- Echocardiography is invaluable if either left ventricular outflow obstruction is suspected or left ventricular function is impaired and in patients with pulmonary hypertension.
- Exercise testing may be useful when collapse is associated with exertion (providing left ventricular outflow obstruction has been excluded), as it may reveal ischaemia, hypotension, an arrhythmia and also hypoxaemia.
- Specialist referral for either left and/or right heart catheterisation.

(b) Neurological
- CT/MRI scanning is rarely required unless there are focal neurological signs or there has been a witnessed seizure.
- Electroencephalogram is of little value in the assessment of patients with recurrent collapse. It may be helpful in confirming a diagnosis of epilepsy, when this is suspected clinically, but it is not indicated routinely in the assessment of syncope.
- Carotid or transcranial doppler ultrasonography is rarely helpful. It should only be considered in the presence of bruits, a palpaple discrepancy between carotid pulses or when the history suggests either carotid or vertebrobasilar insufficiency.

(c) Laboratory tests
- Laboratory tests have a poor yield unless there is clinical suspicion of an abnormality. However, it is worth checking glucose, urea and electrolytes, and haemoglobin.
- Rarely the clinical features may indicate either Addison's disease (adrenocortical failure) or phaeochromocytoma; therefore, a short Synacthen® test or 24-h urine collection for catecholamines, respectively, may be needed.

(d) Other investigations
- Carotid sinus massage. This is contraindicated:
  - **(i)** in the presence of carotid bruits or cerebrovascular disease
  - **(ii)** if there is a history of ventricular arrhythmias or recent myocardial infarction.

Providing there are no contraindications, place the patient in the supine position and monitor ECG and blood pressure. The right carotid artery is massaged longitudinally, with the neck slightly extended, for a maximum of 5 s. If the response is negative there should be a 30-s interval before the left carotid artery is massaged (maximum 5 s).

> **Key point**
>
> **Bilateral carotid massage must never be attempted at the same time.**

A positive cardioinhibitory response is defined as a sinus pause of 3 s or more. A positive vasodepressor response is defined as a fall in systolic blood pressure of more than 50 mm Hg.

- Tilt testing is useful in the further assessment of unexplained recurrent syncope after exclusion of other cardiac causes including arrhythmias. Briefly:
  - **(i)** Baseline pulse and blood pressure recordings are measured with the patient lying supine for 30 min.
  - **(ii)** The patient is tilted to 60–75° for up to 45 min and asked to report any symptoms.
  - **(iii)** A positive result is a cardioinhibitory response and/or a vasodepressor response in association with symptoms.
  - **(iv)** If a positive response occurs, the patient is immediately returned to the horizontal position.

  Other measures may be used to increase the sensitivity of the test.

Key point

With both carotid sinus massage and tilt testing full resuscitation facilities must be available immediately

SPECIFIC CONDITIONS

Status epilepticus

Status epilepticus is defined as either a single seizure lasting for 30 min or repeated seizures between which there is incomplete recovery of consciousness. However, seizures lasting more than 5 min can indicate impending status epilepticus. This may be prevented by immediate treatment.

Key point

Generalised convulsive status epilepticus is a common and serious medical emergency. There is a significant risk of permanent brain damage and death from cardiorespiratory failure (5–10% mortality in those admitted to intensive care units)

Primary assessment and resuscitation – specific summary for epilepsy management

A – Maintain patency/initially with nasopharyngeal airway
Give oxygen ($FiO_2 = 0.85$)
Do not attempt to insert oral airway/intubate while jaw is clenched
Early liaison with anaesthetist

B – Pulse oximeter. Respiratory rate. Occasionally respiration may need to be assisted

C – Establish IV access
Monitor ECG

D – IV benzodiazepine to terminate seizure. Lorazepam is probably better than diazepam as it lasts longer, has a lower incidence of cardio-respiratory side effects and has the same speed of onset
In the absence of IV benzodiazepine access, rectal diazepam may be used.
Check glucose-immediate bedside test and laboratory test

IV thiamine (250 mg over 10 min) especially if there is a history of chronic alcohol abuse

Look for evidence of head injury

E – Check temperature

Look for purpura (meningococcal septicaemia)

Respiratory depression and hypotension may occur after IV diazemuls or lorazepam. The dose of lorazepam is 0.1 mg/kg, max 4 mg by slow IV injection (2 mg/min), that of diazepam 10 mg at a rate of 2.5 mg/30 s. Both can be repeated if necessary. If control is not achieved, phenytoin 15 mg/kg IV should be given with ECG monitoring (reduce dose if patient is previously on phenytoin). The infusion rate should not exceed 50 mg/min because of the risk of cardiac arrhythmias. Further doses up to a total of 30 mg/kg may be given if seizures persist. Then maintenance doses of 100 mg IV should be given every 6–8 h. Phenytoin has the advantage of suppressing seizures without causing cortical or respiratory depression. An alternative is fosphenytoin (18 mg/kg phenytoin equivalent IV up to 150 mg/min). If seizures continue, the patient should be anaesthetised and ventilated.

Cerebral function monitoring is very useful in this situation. Anaesthesia and ventilation should continue until 12–24 h after the last seizure.

Secondary assessment

A history from a relative is important. Are there any symptoms to suggest tumour, meningitis or head injury? Ask about alcohol consumption. If the patient is a known epileptic, ask about current drug regime, compliance or any recent changes in drug therapy.

Physical examination includes a careful neurological assessment, looking particularly for evidence of meningeal irritation, raised intracranial pressure and focal neurological deficits.

Arrhythmia

Bradycardia

Bradycardia may be diagnosed on 24-h ECG monitoring but it is important to document associated symptoms. Review the patient's medications and stop those which may cause bradycardia.

In the presence of sino-atrial node disease, pacing may be considered if pauses greater than 3 s are documented.

With atrioventicular node dysfunction, pacing should be considered for second-degree or third-degree heart block, in the absence of a reversible cause (drugs or ischaemia).

Tachycardia

Supraventricular tachycardias, including atrial fibrillation, often cause palpitations and dizziness but rarely present with syncope. Ventricular tachycardia is more likely to cause syncope. The Wolff–Parkinson–White syndrome and the prolonged QT syndrome should be considered in patients with recurrent syncope. The type of tachycardia will determine the treatment. This comprises anti-arrhythmic drug therapy, occasionally an anti-tachycardia pacemaker/defibrillator or radio-ablation. Transient rhythm abnormalities are increasingly common with increasing age, e.g. short runs of atrial fibrillation and sinus bradycardia occur at night. Do not treat unless there is clear evidence that these arrhythmias are associated with symptoms or predispose to further pathology, e.g. paroxysmal atrial fibrillation and stroke.

Vasovagal syncope

The mechanism of collapse in vasovagal syncope and the assessment of patients by tilt testing has been described. Treatment is not always satisfactory. β blockers may be used to inhibit the initial sympathetic activation in vasovagal syncope. With a positive cardioinhibitory response to tilt testing, disopyramide may be useful (to block the vagal outflow) or dual chamber pacing may be necessary. With a predominant vasodepressor response, ephedrine, dihydroergotamine or fludrocortisone have been tried with variable success.

Carotid sinus hypersensitivity

Hypersensitivity of the carotid artery baroreceptors can cause bradycardia and/or vasodilatation due to vagal activation. The patient complains of dizziness or syncope associated with head turning or the wearing of a tight collar. Diagnosis is by carotid sinus massage as described previously. A positive cardioinhibitory response to this technique responds well to cardiac pacing. As with vasovagal syncope, a vasodepressor response is more difficult to treat.

Postural hypotension

Postural hypotension is associated with:

- hypovolaemia (dehydration, haemorrhage, diuretics)
- drugs (nitrates, levodopa preparations)
- autonomic failure (diabetes mellitus, Parkinson's disease, old age).

It is difficult to treat patients who have postural hypotension. Attempt to correct intravascular volume and rationalise the drug therapy as much as possible. Patients should be advised to stand up slowly and to avoid prolonged standing. Graduated elastic stockings may reduce venous pooling. Fludrocortisone increases salt and water retention and is occasionally helpful. Midadrine, an alpha agonist, can be used orally to increase systemic vascular resistance, but should only be used on the advice of a cardiologist.

Left ventricular outflow obstruction

Advanced aortic stenosis may cause exertional dizziness and syncope because cardiac output is reduced. Such symptoms indicate urgent assessment with a view to aortic valve replacement.

HOCM is associated with restricted cardiac output during stress. Treatment is with negatively inotropic drugs (β blockers, verapamil) to reduce the outflow tract gradient. Dual chamber pacing or surgery may be needed in more advanced cases.

Time Out 12.1

a Define 'stroke'.
b Describe your immediate management of a patient with a suspected transient ischaemic attack.

SUMMARY

Recurrent collapse is common.

- It can be associated with life-threatening underlying pathology and can cause serious injury.

- History and physical examination provide a likely diagnosis in the majority of patients.
- Further investigation will be guided by clinical judgment and by the frequency and severity of the symptoms.

Following stroke:

- Early CT scanning will identify patients suitable for thrombolysis.
- A normal CT brain scan excludes a cerebral bleed.

In transient collapse:

- loss of consciousness is an uncommon feature of transient ischaemic attack
- prodromal symptoms of light headedness, nausea and sweating suggest syncope rather than epilepsy as a cause of collapse
- a sinus pause of 3 s or more with carotid sinus massage is significant.

Advanced Life Support Group

CHAPTER 13
The overdose patient

OBJECTIVES

After reading this chapter you will be able to:

- describe how the structured approach can be applied to patients who have taken an overdose
- discuss the diagnostic clues that may be available in the primary assessment
- understand the indications and contraindications of measures to minimise drug absorption
- describe specific treatment strategies for drugs commonly taken as an overdose

INTRODUCTION

The management of overdose patients is a challenging aspect of emergency medicine. The diagnosis can be difficult in the absence of a clear history, so medical staff should have a high index of suspicion when assessing patients who may have taken a drug overdose. This is especially important in patients presenting with reduced conscious level. Furthermore, many patients are reluctant to co-operate during their initial assessment. The potentially significant effects of substances taken may not be immediately obvious, but may require emergency intervention to limit morbidity and mortality.

The majority of cases are a result of deliberate self-harm. Remember that 'overdose' is a description of an action, not a definitive diagnosis. Overdoses with multiple different medications is no longer a rarity. Be aware of those drugs with high lethality in overdose and narrow therapeutic windows (see below) and be sure to enquire by proper, forensic history taking about the likelihood of the patient having taken any other medications, as well as the apparent one.

Accidental overdose is also fairly common, especially in children and in recreational drug users. Accidental chronic overdose can also occur in elderly patients on multiple medications and in patients with long-standing health problems such as chronic renal failure. The presentation of these patients is variable and may include unusual behaviour, decreased conscious level, fits or cardiac arrhythmias.

Whatever the presentation, medical care should follow the structured approach described in Chapter 3 – with primary assessment and resuscitation preceding secondary assessment, emergency treatment and definitive care. Psychiatric assessment is often necessary in this group of patients and should take place as soon as is reasonably possible.

Acute Medical Emergencies; The Practical Approach, 2nd edition.
Edited by Terence Wardle, Peter Driscoll, and Sue Wieteska.

PRIMARY ASSESSMENT AND RESUSCITATION

A – airway

An appropriate answer to the question, 'Are you all right?' will allow the examining doctor to establish that the patient has a patent airway with reasonable laryngeal function, is conscious with adequate cerebral perfusion, and has sufficient respiratory function to speak. A patient who fails to respond to this question should prompt more detailed airway assessment as to the need for supporting airway adjuncts, as described in Chapter 3. Remember that a patient with a reduced conscious level is likely to have impaired protective airway reflexes and is therefore at risk of regurgitation and aspiration. As always with the unconscious patient, consider the risk of spinal column injury, avoid unnecessary movement of the neck and consider formal cervical spine immobilisation if you feel it is indicated.

B – breathing

Since a number of agents taken in overdose can produce respiratory depression, it is very important to look for adequate breathing. The rate, depth and work of breathing should be assessed. If there are any signs of ventilatory inadequacy, breathing should be supported by the use of a bag–valve–mask device attached to high concentrations of inspired oxygen; consider reversing the cause of the inadequate ventilation (e.g. naloxone in opiate overdose). Even in the patient who appears to be breathing adequately, high concentrations of inspired oxygen should be given until it is deemed unnecessary. Remember that adequate oxygen saturation on pulse oximetry does not guarantee adequate ventilation and carbon dioxide retention may be present with normal oxygen saturation. Arterial blood gases should be measured if there is any concern about the patient's ventilation.

Remember that unexplained tachypnoea may reflect a metabolic acidosis resulting from the overdose, e.g. following salicylates.

C – circulation

Pulse rate, adequacy of peripheral perfusion, cardiac rhythm and blood pressure should be assessed. Inadequate circulation in the overdose patient is generally caused by hypotension or cardiac arrhythmia.

Hypotension is usually caused by a relative hypovolaemia secondary to peripheral vasodilation, often compounded by poor intake during a period of reduced consciousness or the diuretic effect of alcohol taken in association with the overdose. This responds well to fluid resuscitation.

The cause of cardiac arrhythmias differs from those seen in ischaemic heart disease, as they are likely to be due to drug toxicity, and therefore require a different approach in their management. Arrhythmias are often surprisingly well tolerated in the overdose patient and specific anti-arrhythmic drug treatment should be avoided, if possible. Treatment should initially be directed at minimising the effects of the drug likely to be provoking the arrhythmia, in particular correcting abnormalities in electrolytes, calcium and magnesium in the bloodstream. If anti-arrhythmic treatment becomes necessary, cardioversion should be considered as an alternative to medical treatment to avoid potential drug interactions and side effects.

Intravenous access should be established at this stage, providing a route for fluid resuscitation, emergency medication and an opportunity to take blood samples for relevant investigations.

D – disability

Assess conscious level using either the AVPU or Glasgow Coma Scale (see Chapter 3) and measure the pupillary size and response to light. These latter observations can be helpful in establishing a diagnosis if the agent that has been taken is unknown. Although the Glasgow Coma Scale has not been validated for poisoned patients, it remains the most useful objective measure of conscious level.

Many drugs, such as paracetamol and alcohol, can cause rapid hypoglycaemia, so a bedside glucose level should be measured, followed by a formal laboratory sample.

E – exposure

Full exposure is necessary, looking for signs of injury, rashes and possible needle track marks. It is very important to assess temperature at this stage, since a number of drugs can alter thermoregulatory mechanisms, e.g. phenothiazines. Once patients have been fully exposed and the required examination has been completed, cover immediately, as many will lose heat rapidly in this situation.

By the end of the primary assessment, the minimum essential monitoring should include pulse oximetry and ECG monitoring. The respiratory rate, pulse, blood pressure, Glasgow Coma Score, temperature and glucose concentration should have been documented. These observations need to be repeated on a regular basis, to monitor the patient's condition and response to treatment.

Diagnostic clues from the primary assessment may provide a pointer towards the specific drug or drugs ingested and therefore guide specific management. These are listed in Table 13.1.

LETHALITY ASSESSMENT

At the end of the primary assessment, it is important to assess the potential lethality of the overdose. This requires knowledge of the substance, the time it was taken and the dose. Corroborative evidence may need to be sought from other sources, such as family members, friends or paramedic staff. If the nature of the overdose is unknown then a high potential lethality should be assumed.

The UK National Poisons Information Service (NPIS) provides the online TOXBASE database, which provides evidenced-based advice on diagnosis, treatment and management of patients who have been poisoned. This is supported by a second tier consultant-led information service for more complex clinical advice. TOXBASE is available to all health care workers, usually via a registered hospital department or general practice and should be the first point of contact for poisons information. The NPIS has six regional poisons centres which can also be accessed by phone 24 h a day for advice consistent with that available on TOXBASE.

IMMEDIATE MANAGEMENT

Problems identified in the primary assessment should be treated in the standard way. In addition, there are techniques available to reduce absorption of ingested drugs from the gastrointestinal tract and to increase elimination of drugs that have already been absorbed.

Table 13.1 Diagnostic clues from the primary assessment

	Sign	Drug
B	Tachypnoea	Aspirin, ethylene glycol
	Bradypnoea	Opioids
		CNS depressants
C	Tachycardia	Antidepressants
		Sympathomimetics
		Amphetamines
		Cocaine
	Bradycardia	β blockers
		Digoxin
		Clonidine
	Hypertension	Amphetamines
		Cocaine
D	Small pupils	Opioids
		Cholinesterase inhibitors
	Large pupils	Tricyclic antidepressants
		Anticholinergics
		Antihistamines
		Ephedrine
		Amphetamines
		Cocaine
	Coma	Barbiturates
		Tricyclic antidepressants
		Opioids
		Benzodiazepines
		Ethanol
E	Hypothermia	Tricyclic antidepressants
		Barbiturates
		Phenothiazines
	Hyperthermia	Amphetamines
		Cocaine

Reducing absorption

Methods to minimise drug absorption have been used liberally in the past. Recent evidence shows that such techniques are of limited value and only have a role in a minority of patients. It should be noted that they should never be used as a punitive measure, as their use does not deter patients from further episodes of overdose and they expose the patient to the risk of side effects without likely therapeutic benefit. These measures should be used on the advice of Toxbase or NPIS.

Activated charcoal

Charcoal works by absorbing ingested drugs onto its large surface area. It is the treatment of choice when efforts to decrease the systemic absorption of drugs in the overdose patient are indicated. Its dose is 50 g (1 g/kg in children). However, it is usually limited to patients presenting within 1 h of taking a life-threatening overdose. This time period may be increased for certain drugs which prolong gastric emptying, most commonly aspirin and tricyclic antidepressants (Table 13.2).

Table 13.2 Drugs which delay gastric emptying

Drug	Minimum dose	Maximum time since ingestion
Paracetamol	12 g	4 h
Theophyllines	>2.5 g	4 h
Tricyclic antidepressants	>750 mg	8 h
Aspirin	15 g	12 h

Charcoal will only absorb 10% of its own weight of a drug, so in large overdose repeated doses of charcoal can be considered. Compliance with taking charcoal is generally low, due to its unpleasant black appearance and taste. The use of a naso-gastric or oro-gastric tube may help those patients unwilling to drink charcoal. Consider endotracheal intubation, to prevent aspiration either in unconscious patients, or in those who have recently taken an overdose that will render them unconscious.

Certain agents are not absorbed by charcoal; these include metal salts (iron, lithium), alcohols, acids/alkalis, solvents, hydrocarbons, cyanide and fluoride.

Whole bowel irrigation

Polyethylene glycol (e.g. Kleen Prep) is given either orally or via a naso-gastric tube, at a rate of 2 l/h, until the resulting watery diarrhoea becomes clear. It should not be used in the presence of a paralytic ileus or suspected mechanical obstruction. It is indicated after ingestion of large volumes of agents not absorbed by charcoal, body packers and significant ingestion of sustained release or enteric coated formulations.

Gastric lavage

Gastric lavage has largely been superceded by the use of activated charcoal. Delayed gastric lavage has little value and may propel drugs further along the gastrointestinal tract. It may be considered for those drugs not absorbed by charcoal, such as iron and lithium. It is absolutely contraindicated after ingestion of caustic agents (Table 13.3). If the patient's airway is compromised and gastric lavage is thought to be of value, the airway should be managed by a doctor with anaesthetic training and will usually require insertion of a cuffed endotracheal tube.

Table 13.3 Contraindications to gastric lavage

Corrosive agents	Acid
	Alkali
	Bleach
Petroleum derivatives	Petrol
	Paraffin
	White spirits
	Turpentine substitute
	Kerosene

Therapeutic emesis

Centrally acting emetics such as ipecacuanha have been used historically to promote active vomiting. The symptoms produced can often cloud an already complicated clinical picture. There is no evidence that it decreases absorption and its use in emergency practice is now obsolete.

Increasing elimination

Adequate cardiovascular and fluid resuscitation of the patient in the primary assessment will maximise the body's normal routes for excretion of toxins via the liver and kidneys. Measures to increase elimination of life-threatening overdoses are specific to the drugs ingested and include therapeutic diuresis, alkalinisation, chelation, haemoperfusion and haemodialysis. Such treatments should only be used on the advice of specialists.

Multiple dose activated charcoal

Fifty grams can be given every 4 h for large ingestions of drugs that have a significant entero-hepatic circulation, such as carbamazepine, theophylline, phenobarbitone, quinine, dapsone, digoxin and salicylate.

Urinary alkalinisation

This can be used for large ingestions of aspirin, phenobarbitone, chlorpropramide, mecoprop and phenoxyacetate herbicides. Urinary alkalinisation can be achieved by giving intravenous sodium bicarbonate (1 litre of 1.26% over 3 h). Renal function and plasma potassium must be checked first, as it is difficult to produce alkaline urine in the presence of hypokalaemia. Potassium levels may fall during the diuresis, so they should be checked regularly and replaced with 20–40 mmol of potassium in each litre of sodium bicarbonate if needed. The therapeutic goal is a blood gas base excess reading of +8 and urinary pH $>$ 7.5.

Extracorporeal methods

These include charcoal haemoperfusion, haemofiltration and haemodialysis. They are only indicated in a limited number of situations and are usually done in an HDU or ITU setting. However, when clearly indicated/advised by the Poisons Information Centre, these treatments are life-saving and should be started immediately.

SECONDARY ASSESSMENT

As in all other emergency presentations, the secondary assessment involves taking as full a history as possible. A full examination is also necessary, and should include examining for physical evidence of self-harm and injury such as cutting, needle marks and signs of head injury. Some symptoms and signs elicited in the secondary assessment may provide clues to specific types of overdose. These are listed in Table 13.4.

Appropriate investigations should be ordered, based on the findings in the secondary assessment. A 12-lead ECG should be recorded in all patients with any kind of rhythm disturbance or with potential cardiac sequelae from their overdose, in particular looking carefully for signs of conduction abnormalities that may precede frank rhythm disturbance. A chest X-ray is necessary in unconscious patients, those with possible aspiration and those with abnormal findings on respiratory examination.

Table 13.4 Clues to possible overdose from secondary assessment

Pulmonary oedema	Salicylate
	Ethylene glycol
	Opiate
	Organophosphates
	Paraquat
Hypoglycaemia	Insulin
	Oral hypoglycaemic
	Ethanol
	Paracetamol
	Salicylate
Hyperglycaemia	Salbutamol
	Theophylline
Hypokalaemia	Salbutamol
	Theophylline
	Salicylates
Metabolic acidosis	Salicylates
	Paracetamol
	Ethanol
	Ethylene glycol
	Tricyclics
Raised osmolality	Ethanol
	Methanol
	Ethylene glycol
Prolonged prothrombin time	Salicylates
	Paracetamol

Blood investigations depend on the drugs ingested. They are likely to include full blood count, glucose, urea and electrolytes, renal and liver function, coagulation screen and serum osmolality. Arterial blood gases will help to quantify any respiratory compromise and also indicate an acid–base disturbance. Toxicology screening is generally limited by local resources to paracetamol and salicylate estimation, but blood and urine may be saved for further testing, depending on the services available.

Commercially available urinary dip tests can be used to indicate the presence of certain drugs used recreationally such as benzodiazepines, opioids, cocaine, amphetamines and cannabinoids. It is worth remembering that some of these chemicals remain present in the urine for several weeks. 'Recreational' drug use is common. As with all investigations, treat the patient, not the result. If the patient's condition does not clearly fit the 'toxidrome/toxic drug syndrome' produced by the drug that has been shown to be present search for another cause of the patient's medical emergency. In particular, for example the patient with altered consciousness may have head trauma, stroke or intracranial infection.

EMERGENCY TREATMENT OF SPECIFIC OVERDOSES

In addition to the general management described previously, certain poisons require specific antidotes. Some of the more commonly used antidotes are listed in Table 13.5.

Table 13.5 Specific measures in overdose

Drug	Treatment
Paracetamol	*N*-Acetyl cysteine
Opioids	Naloxone
Tricyclic antidepressants	Sodium bicarbonate
Digoxin	Specific Fab antibodies
Ethylene glycol	Ethanol, fomepizol, haemodialysis
Iron	Desferrioxamine
Methanol	Ethanol
Cyanide	100% oxygen, amyl nitrite, sodium thiosulphate, high dose vitamin B_{12}
Organophosphates	Atropine, pralidoxime
β blockers	Glucagon, dobutamine, atropine, isoprenaline, temporary pacing
Aspirin	Dose dependent: diuresis, alkaline diuresis, haemodialysis

Paracetamol, tricyclic antidepressant and opioid overdoses are common. These drugs will be discussed in more detail, due to their, risk of high morbidity and mortality and because of the potential benefit from correct management.

Paracetamol

Paracetamol overdose is very common in the UK, with a risk of high morbidity and mortality. With correct treatment, most patients will make a full recovery with no long-term sequelae.

Paracetamol is normally metabolised in the liver to form non-toxic metabolites which are excreted in the urine. If the normal pathways of metabolism are saturated, the excess paracetamol will form toxic metabolites which are rapidly conjugated with glutathione and excreted. Accumulation of the toxic metabolites results in hepatocellular damage and liver necrosis.

The antidote to paracetamol overdose is *N*-acetylcysteine (NAC), which works by preventing toxic metabolite formation, increasing availability of glutathione and acting as a glutathione analogue. It also has vasodilatory, anti-inflammatory and anti-oxidant effects, which limit morbidity and mortality once hepatotoxicty is established.

Certain patient groups are at greater risk of morbidity in paracetamol overdose, either from glutathione deficiency (alcoholism, AIDS, anorexia nervosa, malnutrition) or enhanced enzyme activity, which increases toxic metabolite formation (patients on rifampicin, barbiturates, anti-convulsants).

Important factors in the assessment of the patient are: amount taken, time taken and whether the overdose was staggered over a period of time. Significant overdose is considered to occur if the patient has ingested a total of at least 12 g or 150 mg/kg of paracetamol. This level is reduced to 75 mg/kg in the at-risk patient groups described above.

The serum paracetamol level is valuable to diagnosis and management, as the patient may exhibit non-specific symptoms or be asymptomatic for up to 24 h following ingestion. The level should be taken 4 h after single ingestion and plotted on the paracetamol treatment graph to determine need for NAC therapy. The level should not be taken until 4 h have elapsed from ingestion, as the result is likely to

be inaccurate and can lead to under-treatment of the patient. Paracetamol levels are also extremely difficult to interpret in cases of staggered overdose or when 24 h have elapsed since the time of ingestion. In these cases, markers of hepatic and renal damage such as prothrombin time, creatinine and electrolytes, venous bicarbonate and arterial blood gases (and, of course, expert advice), should guide management.

In situations where there is strong clinical suspicion of significant overdose or the paracetamol level will not be available within 8 h of ingestion, start NAC therapy until either paracetamol levels are available or other markers of toxicity are available to guide further management. Although NAC is most effective if started early, it should not be started without clear clinical indication as it can cause an anaphylactoid reaction in susceptible patients. The safe management of patients with paracetamol overdose can be complicated and is beyond the scope of this chapter. Detailed advice relating to individual patients, in particular those who have taken a staggered overdose over a period of time, should be obtained from the NPIS phone lines or from TOXBASE.

Tricyclic antidepressant drugs

The toxicity of tricyclic antidepressant (TCA) drugs is mainly due to anticholinergic effects and a quinidine-like effect on the myocardium (blocking of fast sodium channels). The majority of life-threatening problems occur in the first 6 h following ingestion and are principally due to seizures and arrhythmias. The patient may show signs of agitation, tachycardia, dilated pupils and ataxia. This may progress rapidly to drowsiness or coma, increased tone, hyperreflexia, hypotension, and respiratory depression. The ECG may show tachycardia and, in severe poisoning, bizarre rhythms may be present. Widening of the QRS complex indicates a greater risk of seizures and arrhythmias.

These patients are often under-resuscitated and may need intensive care. Assisted ventilation may be needed to correct hypoxaemia and hypercapnia. Blood should be taken for urea and electrolytes, glucose and arterial blood gases. A 12-lead ECG is needed to look for conduction disturbance and the patient must be monitored for a rhythm disturbance.

If the patient has possibly taken a significant overdose (>1 g of TCA in an adult), they must be placed in a high dependency treatment area on a cardiac monitor until at least 6 h have passed from the latest possible time of ingestion, even if their initial ECG and physical examination signs are normal. If the initial ECG or GCS is abnormal, they should remain on a cardiac monitor for at least 12 h after their ECG has returned to normal.

Cardiac arrhythmias should be treated by correction of hypoxaemia, electrolyte disturbance and acidosis, rather than by drugs wherever possible. The exception to this, however, is sodium bicarbonate which is of particular value in these patients. Alkalinisation alters the binding of TCAs to the myocardium and the sodium load counteracts the sodium channel blockade, both of which are cardioprotective. Even in the absence of acidosis, treat adults with arrhythmias or ECG abnormalities with 50 mmol sodium bicarbonate intravenously. (500 ml of 1.26% contains 75 mmol sodium bicarbonate and 50 ml of 8.4% contains 50 mmol sodium bicarbonate), but remember the stronger concentration as it is very irritant to veins and can cause skin necrosis if extravasation occurs. Further doses may be required depending on the clinical response. The patient with significant physiological disturbance should have their pH maintained above the upper range of normal, at 7.5–7.55, by means of manipulation of hyperventilation, where possible, and further doses of sodium bicarbonate.

Convulsions should be treated in the usual fashion with intravenous benzodiazepines. If seizures persist, the patient may need to be intubated, paralysed and ventilated. Further anti-convulsants need to be administered. **Phenytoin should not be given** in TCA overdose as it blocks sodium channels and can increase the risk of arrhythmias.

If the patient is thought to have taken a combined TCA and benzodiazepine overdose, **flumazenil should not be used** to reverse the benzodiazepine component. In combined overdose, the benzodiazepine is protective and reversal may precipitate seizures or cardiac arrest, which may be refractory to treatment.

Opioids

Acute opioid poisoning is commonly accidental in intravenous drug users, either from diamorphine (heroin) or from methadone. These patients often present with pinpoint pupils and profound respiratory depression, or even respiratory arrest. Fresh and old venepuncture marks, track marks and thrombosed superficial veins may give a clue to the diagnosis.

Immediate management should be aimed to ensure a patent airway and oxygenation. This can usually be achieved on a short-term basis with bag valve mask ventilation and high concentrations of inspired oxygen. Endotracheal intubation may be required. Be careful not to fall into the trap of letting an apnoeic patient become more and more cyanosed while hunting for venous access to give naloxone. Ensure adequate oxygenation and ventilation first.

Naloxone is a specific opioid antagonist and should be given intravenously as a therapeutic trial as soon as safely possible in suspected opioid poisoning. The naloxone should be titrated in small aliquots against the patient's response, as a large bolus runs the risk of rapid complete reversal. In chronic users, this will precipitate an acute withdrawal syndrome (AWS) or 'cold turkey' characterised by profound agitation, nausea, abdominal pain and diarrhoea. This can result in an angry, uncooperative, potentially violent and often slightly confused patient taking their own discharge from the resuscitation room. The therapeutic effect of naloxone is much shorter than most of the opioids taken recreationally, due to more rapid redistribution of naloxone away from the receptor sites. Therefore, the patient is at considerable risk of relapsing into life-threatening respiratory depression shortly after leaving the hospital.

To avoid precipitating AWS, titrate naloxone at a rate of 0.1 mg/min, aiming for a respiratory rate of greater than 10 and a GCS of 13–14/15. The patient may still be drowsy at this level of reversal, but should maintain their own airway and self ventilate adequately. An infusion of naloxone may be indicated in some patients, usually at a dose of 2/3 of the initial dose per hour. The patient should be carefully monitored until the opioid is fully reversed. Avoid using subcutaneous and intra muscular routes if possible as absorption from these sites is unpredictable. However, if intravenous access is impossible and the patient is collapsed, doses of 0.8 mg subcutaneously or 0.4 mg intramuscularly can be given and the patient observed carefully for at least 2 h.

MENTAL HEALTH ASSESSMENT

It is essential that all patients who have taken an intentional drug overdose undergo mental health assessment. This is important for two reasons. Firstly, it is important to make a risk assessment as soon as reasonably possible to determine the patient's risk of immediate further self-harm. This risk assessment can help to direct the care each patient requires. This may include the need for one-to-one

psychiatric nursing for those patients at risk of further suicide attempts. A number of factors can help to indicate the seriousness of the individual's intent for self-harm and some of these are summarised in the next box. Secondly, the patient's need for ongoing psychiatric support needs to be decided.

Factors defining intent in deliberate self-harm

Patient's perception of lethality
Evidence of premeditation
Measures to prevent discovery
Social circumstances
Evidence of depression
Evidence of psychosis

A common error in the management of deliberate self-harming patients is to delay mental health assessment until all medical treatment is completed. This can put vulnerable patients at considerable risk, by delaying recognition and appropriate management of severe mental illness. In most cases, the medical and psychiatric care can be conducted in parallel, e.g. the psychiatric liaison nurse can perform a risk assessment while medics await blood results. Similarly, moderate alcohol consumption is not a contraindication to mental health assessment. The exceptions to this are grossly intoxicated patients, those who are critically ill or have reduced conscious level.

All doctors involved in the medical management of overdose patients should be trained in basic mental health risk assessment and be able to recognise when psychiatric referral is appropriate. Many emergency departments have a mental health liaison team, consisting of specially trained psychiatric nurses who work as independent practitioners and are able to assess, manage and discharge patients with mental health problems. These practitioners' skill and experience can lend invaluable support to emergency medical teams.

DEFINITIVE CARE

Some patients may require admission to the medical wards or to an emergency department short stay ward, either for active management or for a short period of observation. However, a large number will be fit for medical discharge after immediate assessment. It should be remembered that many of the effects of drug overdose are delayed, e.g. nephrotoxic renal failure due to either paracetamol or aspirin. It is important to anticipate such complications and where possible take measures to prevent them. Unfortunately this is not always possible if there is a prolonged time between ingestion and presentation to hospital.

SUMMARY

- The structured approach to the seriously ill patient should be used when dealing with patients who have taken overdoses.
- The potential lethality of the overdose must be assessed at the end of the primary assessment.

- If indicated, measures should be taken to stop absorption and increase excretion of the ingested compound.
- Specific treatment may be indicated once the substance ingested has been identified.
- A mental health assessment must be made in all patients to determine the risk of further self-harm and need for psychiatric support.

Advanced Life Support Group

CHAPTER 14
The patient with a headache

OBJECTIVES

After reading this chapter you should be able to:
- understand the causes of headache
- describe a classification of headache that will be useful in clinical practice
- discuss the initial management of a patient with headache
- describe how clinical signs detected in the secondary assessment influence diagnosis and subsequent management.

INTRODUCTION

Patients presenting with a headache of acute onset account for less than 2.5% of new emergency attendances. Of these, only 15% will have a serious cause for their headache. Therefore, the aim is to identify the relatively small group of high risk patients.

PATHOPHYSIOLOGY

Pain sensitive structures that can cause a headache include:
- dura
- arteries
- venous sinuses
- paranasal sinuses
- eyes
- tympanic membranes
- cervical spine.

These are innervated by somatic afferents from the V, VII, IX and X cranial nerves (linked via the spinal tract of the trigeminal nerve) and the upper three cervical nerve roots. Pain will occur if there is traction, inflammation or distension of these structures, in particular, the dura, blood vessels and nerves. A throbbing headache is non-specific because it is common to many intracranial conditions. Similarly the site of pain is non-specific, but it can provide clues to underlying pathology as outlined below.

• Frontal	Ipsilateral forehead and eye pain, referred via the trigeminal nerve, can indicate a lesion in the anterior or middle cranial fossa. Bifrontal headache can be a presenting feature of acute hydrocephalus secondary, e.g. to either a supra- or infratentorial lesion. The pain is attributed to vascular distortion following dilatation of the lateral ventricles.

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- Frontotemporal — Unilateral pain is common with sinusitis and dental problems. In addition, orbital cellulitis, glaucoma, and cavernous sinus thrombosis have a similar presentation.
- Occipital — Posterior fossa or upper cervical spine pathology (referred via the upper three cervical nerve roots) can present with occipital pain.

In contrast, the distribution of pain can be more specific.

- Trigeminal — Neuralgia is restricted to the distribution of the trigeminal nerve. The searing paroxysms of intense pain are usually unilateral and confined to one division. Occasionally, two or all three divisions are involved. This specific distribution of pain is attributed to distortion of the blood vessels supplying the trigeminal nerve.
- Somatic afferent — Postherpetic neuralgia is secondary to inflammation and will occur in the distribution of the affected nerve, i.e. the V, VII, IX and X cranial nerves.

CLINICAL ASSESSMENT

A useful way to categorise patients presenting with a headache is shown in the next box.

Clinical classification of headache

Headache with altered Glasgow Coma Score and/or focal neurological signs
Headache with papilloedema but no focal neurological signs
Headache with fever but no focal neurological signs
Headache with extracranial signs
Headache with no abnormal signs

This classification will form the framework of the remaining sections in this chapter. It is important to note that some conditions occur in more than one category – reflecting their diverse manifestations. The structure of the initial assessment and the above classification is designed to ensure early detection and management of an immediately life-threatening problem, i.e. headache with **altered Glasgow Coma Score** and/or **focal neurological signs**. The remaining, non-immediately life-threatening causes will be identified in the secondary assessment.

HEADACHE WITH ALTERED GLASGOW COMA SCORE AND/OR FOCAL NEUROLOGICAL SIGNS

After assessing 'D' the following will have been identified:
- a reduction in the Glasgow Coma Score
- the presence of lateralising signs
- pupillary abnormalities
- meningeal irritation.

Table 14.1 Causes of headache with altered Glasgow Coma Score and/or focal neurological signs

Vascular	Stroke	Daily
	Subarachnoid haemorrhage	Weekly
	Chronic subdural haematoma	Monthly
Infective	Meningitis	Daily
	Encephalitis	Monthly
	Cerebral abscess	Monthly
	Subdural empyema	Annually
	Cerebral malaria	Annually
Neoplastic	Secondary intracerebral tumour	Weekly
	Primary intracerebral tumour	Monthly

Although the specific diagnosis is often unknown at this stage, the patient should receive optimum oxygenation and appropriate control of both blood pressure and serum glucose.

An immediately life-threatening event either causing or following a headache will be identified in the **primary assessment**. Such conditions are listed in Table 14.1.

> **Key point**
>
> **Remember that the goal of initial management is to prevent secondary brain injury**

Key management issues

- The mode of onset of symptoms will help distinguish different conditions, e.g.:
  - acute onset = vascular
  - subacute onset = infective
  - chronic onset = neoplastic.
- If the patient is febrile, take blood cultures and start appropriate antibiotic therapy to cover bacterial meningitis. If there is a history of foreign travel to relevant areas, request thick and thin films to exclude malaria. Subsequent investigations will include imaging, either CT or MR. This should precede lumbar puncture (see Chapter 11).
- Further management should be discussed with appropriate clinicians, i.e. neurologist, microbiologist, neurosurgeon and/or infectious disease physician.

Specific management of the conditions shown in Table 14.1 is considered in the unconscious patient (Chapter 11).

> **Key point**
>
> **Emphasis should be placed on seeking meningeal irritation, fever, reduced conscious level, focal neurological features and skin rash**

The primary assessment will detect any changes in the Glasgow Coma Score, pupillary response and lateralising signs. However, physical signs can change.

Table 14.2 Key features of the assessment of a patient with a headache

Characteristics	New onset
	Acute onset
	Progressive
	Wakens from sleep
	Worst ever
Associated symptoms	Photophobia
	Neck stiffness
	Fever
	Altered mental state
	Neurological dysfunction
Examination findings	Temperature
	Meningeal irritation
	Abnormal neurological signs
	Rash

Thus, the secondary assessment facilitates re-evaluation, combined with obtaining further information and a more comprehensive examination. The relevant secondary assessment features are summarised in Table 14.2.

Most patients presenting with a headache will not have an immediately life-threatening condition. Thus, in the **secondary assessment** the doctor has time to take a full history. A new headache, or one different from normal, can indicate intracranial pathology.

Key point

Over one third of patients with major subarachnoid haemorrhage will have suffered a minor (sentinel) bleed in the preceding hours or days

It is important to elicit the frequency of headache and what the patient was doing at the onset of the pain, e.g. a headache that wakes a patient from sleep suggests significant pathology. Furthermore, headaches that become progressively more severe, or chronic ones that are different from usual, may be caused by raised intracranial pressure.

An important part of this assessment is to exclude raised intracranial pressure. Features that would indicate this diagnosis are listed in the box below.

Headache with features suggestive of raised intracranial pressure

Worse on waking
Aggravated by coughing, vomiting, straining, standing and sitting
Relieved by lying down
Papilloedema and neurological signs (these are late signs)

Key point

It is important to realise that the classic early morning headache of raised intracranial pressure is uncommon

Headache exacerbated by changes in posture or associated with nausea, vomiting or ataxia requires further investigation, especially cranial imaging, when neurological signs are detected.

Specific information should also be sought regarding photophobia, neck stiffness, altered mental function, neurological dysfunction and the presence of a fever or skin rash. These features may be transient.

HEADACHE WITH PAPILLOEDEMA BUT NO FOCAL NEUROLOGICAL SIGNS

The mechanism of cerebrospinal fluid production and the relationship to intracranial pressure is discussed in Chapter 11.

It is also important to remember the pathophysiology of raised intracranial pressure. The brain is contained within a rigid skull with little room for expansion. There are four ways to disturb the normal cerebral homeostasis:

- increasing the pressure in the arteries
- adding to the intracranial contents, e.g. tumour or oedema
- obstructing cerebrospinal fluid drainage
- preventing venous drainage, e.g. congestion.

In the context of headache, the relevant causes are listed in Table 14.3.

Key point

Papilloedema:
is usually bilateral and causes minimal interference with vision
is associated with hypertension due to optic nerve vascular damage and cerebral oedema

Key management issues

- Antihypertensive therapy is required if the diastolic blood pressure is greater than 120 mm Hg and retinal haemorrhages are present.
- CT is warranted, especially if the blood pressure is normal.
- The CT scan result will guide further management, e.g. dexamethasone for tumour associated oedema and neurosurgical referral for evacuation of haematoma.
- Further management will follow discussion with appropriate clinicians, especially a neurologist or neurosurgeon.

Table 14.3 Causes of headache and papilloedema, but with no focal or neurological signs

Arterial	Accelerated hypertension/arterial dilatation
Intracranial	Mass lesions, e.g. tumour, haematoma
	Cerebrospinal fluid accumulation
	Cerebral oedema
	Benign intracranial hypertension
Venous	Obstruction to outflow, i.e. sinus thrombosis
	Congestion

Table 14.4 Causes of headache with fever but no focal neurological signs

Intracranial		Meningitis	Daily
		Subarachnoid haemorrhage	Weekly
		Encephalitis	Monthly
Extracranial	– focal	Acute sinusitis	Daily
	– systemic	Viral illness	Daily
		Malaria	Annually
		Typhoid	Annually

- Although neurological signs may be absent at presentation, they can develop as the condition progresses. For example, hypertension can lead to a stroke; a host of focal features can be associated with either an intracerebral tumour (depending on the site and extent) or venous sinus thrombosis. In addition, they can all present as epilepsy.

HEADACHE WITH FEVER BUT NO FOCAL NEUROLOGICAL SIGNS

This is a common mode of presentation and the major conditions are listed in Table 14.4. However, headaches and fever are common to many infectious diseases. One particularly useful differentiating feature is the presence of neck stiffness.

Key point

Do not assess neck stiffness in patients with potential cervical spine instability, e.g. rheumatoid disease, ankylosing spondylitis, Down's syndrome and trauma

Neck stiffness is a non-specific sign that should be assessed with the patient flat, your hands supporting the occipital region and by feeling for increased tone while:

1. gently rotating the head (as if the patient is saying no)
2. slowly lifting the head off the bed. During this manoeuvre also watch for hip and knee flexion. This response, referred to as Brudzinski's sign, indicates meningeal irritation. The latter will also produce a positive Kernig's sign, i.e. whilst the patient is lying flat with one leg flexed at both the hip and knee, resistance is experienced when trying to extend the knee. Repeat on the other limb. A bilateral response indicates meningeal irritation., A positive Kernig's sign can occur with a radiculopathy but here other symptoms and signs of nerve root irritation will be found.

Neck stiffness can be elicited in the following conditions:

- Meningeal irritation
  - Infective – Commonly bacterial or viral
  - Chemical – Subarachnoid haemorrhage
- Cervical spondylosis
- Parkinsonism
- Myalgia, e.g. as a prodromal feature of a viral illness
- Pharyngitis
- Cervical lymphadenopathy.

Other features from the history and examination will provide clues to the underlying diagnosis.

Key point

In a patient with neck stiffness:
Kernig's sign usually indicates meningeal irritation
discomfort only on forward flexion suggests pharyngitis and/or cervical lymphadenopathy

If meningeal irritation is present, a lumbar puncture is necessary after a CT scan, to exclude either meningitis or subarachnoid haemorrhage. Similarly, cerebrospinal fluid is required to establish a diagnosis of encephalitis. In contrast, if there is a history of foreign travel, further details and investigations are required to exclude relevant infectious diseases, especially malaria and typhoid.

Time Out 14.1

During your 5-min break answer the following questions.
a List the conditions that can present as 'headache with fever but no focal signs'.
b List the diagnostic signs of a radiculopathy.

HEADACHE WITH EXTRACRANIAL SIGNS

Many conditions can present with headache and extracranial signs; some examples are listed in Table 14.5.

Acute sinusitis

This acute infection commonly causes frontal and/or maxillary sinusitis. However, it may extend to involve the ethmoid and sphenoid sinuses. In contrast, isolated infection in these areas is rare. Sinusitis is usually secondary to either the common cold or influenza and both streptococci and staphylococci are involved. On occasions, anaerobes can be present when maxillary sinusitis is associated with a dental apical abscess.

Patients usually relate an initial history of an upper respiratory tract infection. This can be followed by headache and facial pain (which is often supraorbital (frontal sinusitis) and infraorbital (maxillary sinusitis)). The pain is often worse in the morning and exacerbated by head movements or stooping. Nasal obstruction is invariably present. The clinical signs are listed in the box.

Table 14.5 Causes of headache with pericranial signs

Acute sinusitis	Daily
Cervical spondylosis	Daily
Giant cell arteritis	Monthly
Acute glaucoma	Annually

Clinical signs of sinusitis

Pyrexia
Tenderness over the affected sinus
Oedema of the upper eyelid

Key point

Swelling of the cheek:
is very rare in maxillary sinusitis
is commonly of dental origin
from antral pathology usually implies a carcinoma

The treatment comprises:
- analgesia
- antibiotics
- nasal decongestants.

Most patients with acute sinusitis will recover completely. However, liaison with an ear, nose, and throat (ENT) specialist is required when either chronic infection or complications may occur (see Table 14.6).

Further investigations are often needed and the results will dictate referral to the relevant specialist colleague.

Cervical spondylosis

This is a common condition caused by intervertebral disc degeneration producing two main effects:
- *Annulus bulging,* which elevates the periosteum from adjacent vertebral bodies, resulting in osteophyte formation.
- *Disc space narrowing* causes malalignment of posterior facet joints, which develop hypertrophic osteoarthritic changes, and ligament folding, and disruption as the vertebral bodies become closer.

These chronic degenerative changes are referred to as spondylosis and, in the cervical spine, occur commonly at the C4/5, C5/6 and C6/7 interspaces. The combination of disc space narrowing, posterior facet joint malalignment and ligament folding results in either anterior or posterior displacement of one vertebral body

Table 14.6 Indications for ENT referral in patients with acute sinusitis

Potential for chronic infection	Poor drainage
	Virulent infection
	Dental infection
	Immunocompromised patient
Complications	Laryngitis
	Pneumonia
	Orbital cellulitis/abscess
	Meningitis
	Cerebral abscess
	Osteomyelitis
	Cavernous sinus thrombosis

on another. Any of these effects, either individually or combined, can cause compression of the spinal cord (producing a myelopathy) or adjacent nerve roots (radiculopathy).

Another feature of this degenerative condition is headache. This is thought to arise not only from posterior facet joints and the associated ligaments, but also from osteophytes, which may irritate the C2 nerve root and branches of the greater occipital nerve. The pain classically involves one or both sides of the neck, extending to the occiput or even the temporal and frontal areas. It is often aggravated by movement and is worse in the morning after the neck has been inappropriately positioned on, or inadequately supported by, pillows.

Clinical examination usually reveals restriction of neck movements, especially lateral flexion and rotation.

Key point

Always check for signs of a myelopathy or radiculopathy

The headache will usually respond to antiinflammatory drugs, but local infiltration with lignocaine and hydrocortisone may be required. It is best to leave this type of treatment to the 'pain specialist'.

Despite the extensive degenerative changes and associated neurology, the cervical spine is usually stable and acute cord compression is rare (the exception is an acute disc prolapse). Assessment for spinal surgery is advocated, as it may be possible to prevent further neurological compromise.

Giant cell arteritis (cranial arteritis, temporal arteritis and granulomatous arteritis)

This condition predominantly affects large/medium sized arteries. It is rare before the age of 50 and commonly affects those aged between 65 and 75 years.

Giant cell arteritis classically involves the branches of the arteries originating from the aortic arch in a patchy distribution. Microscopically, the affected vessels show infiltration with lymphocytes, macrophages, histiocytes and multinucleate giant cells. These result in intimal fibrosis and thickening producing narrowing or occlusion of the vessel lumen.

Giant cell arteritis also affects the extracranial branches of the vertebral artery in 70–100% of cases. It commonly presents with neurological deficits due to vertebro-basilar insufficiency (VBI) which may present as abnormal gait, dizziness, vertigo, vomiting and slurred speech.

Tip: Beware VBI involvement can occur without the symptoms of temporal arteritis.

The onset of arteritis may be acute, but the symptoms are often present for many months before the diagnosis is made. Thus, a high degree of suspicion is needed. Most patients have clinical features related to the arteries involved, i.e. mainly those originating from the aortic arch. Therefore, symptoms and signs related to the head and neck are common.

Presentation

- Usually > 50 year
- Female > male

- Insidious onset over weeks
- Often associated with jaw claudication on chewing (very specific sign) and diplopia
- Risk of blindness if not treated.

Headache is a frequent presentation, localised to the superficial temporal or occipital arteries, and described as throbbing and worse at night. On examination, these vessels can be tender, red, firm and pulseless. In addition, increased scalp sensitivity may predominate with complaints like, 'It is painful to comb my hair'. Visual problems (see next box) are associated with involvement of the following:

- ciliary artery producing an ischaemic optic neuropathy
- posterior cerebral artery leading to hemianopia
- vessels supplying the III, IV and VI cranial nerves resulting in ophthalmoplegia.

Visual problems associated with giant cell arteritis

Blurred vision
Visual hallucinations
Amaurosis fugax
Loss of vision – Transient
– Permanent
Hemianopia
Ophthalmoplegia

Although the head and neck vessels are commonly affected, arteritis can be widespread (see next box). However, these features are rare. In contrast, constitutional symptoms are common and include weight loss, fever, malaise and a low-grade anaemia (often a normocytic normochromic picture). Furthermore, polymyalgia rheumatica is present in approximately 50% of patients who have giant cell arteritis.

Other manifestations of giant cell arteritis

Intermittent claudication
Peripheral neuropathy
Myocardial ischaemia/infarction
Gut ischaemia/infarction
Stroke
Aortic arch syndrome

Diagnosis

The diagnosis is confirmed, in approximately 75% of cases, by biopsy of an affected vessel, usually the temporal artery. However, the histology changes may be patchy. Other laboratory investigations yield non-specific results, which reflect the inflammatory response, e.g. elevated ESR and CRP. The vessels can be imaged by Colour flow Doppler or MR angiography which can distinguish VBI from giant cell arteritis from atherosclerotic disease.

Key point

A normal ESR and CRP do not exclude the diagnosis of giant cell arteritis

Treatment

The lack of readily available supportive laboratory data means that the diagnosis relies on the clinician's skills. Despite the many potential modes of presentation, treatment should be started as soon as the condition is suspected, because of the profound morbidity and mortality. Do not wait for a biopsy to confirm the diagnosis. This can be done at a later stage, if required, as the histological changes persist for approximately 14 days. Prednisolone 60 mg daily, in divided doses, is an effective treatment. This is reduced slowly, according to the patient's response, to achieve a maintenance dose of 10 mg after one year. Occasionally, 'pulse' intravenous methylprednisolone is used to treat ocular involvement. Early liaison with specialist colleagues is necessary when patients present with clinical signs or when giant cell arteritis has entered into the differential diagnosis. This is very important as correct diagnosis and treatment will reduce morbidity and prevent inappropriate chronic therapy with corticosteroids (and their related side effects).

Acute glaucoma

Acute closed angle glaucoma results from raised intraocular pressure. It commonly occurs in patients who are over 50 years. Under normal circumstances, aqueous humour circulates from the capillaries of the iris and ciliary muscle in the posterior chamber (between the iris and the lens) to reach the anterior chamber (between the iris and the cornea) (Fig. 14.1).

With raised intraocular pressure, the iris root protrudes into the back of the cornea and closes the canal of Schlemm preventing drainage of aqueous (Fig. 14.2). The precise cause of glaucoma is unknown, but patients who have diabetes mellitus or an affected first-degree relative are at increased risk. Furthermore, it is more common in individuals who are long sighted (hypermetropia). The shorter eyeball has a shallow anterior chamber, hence a very narrow drainage angle, which is more readily obstructed. The sudden rise in intraocular pressure causes vascular insufficiency, which can lead to ischaemia of the optic nerve and retina, if left untreated.

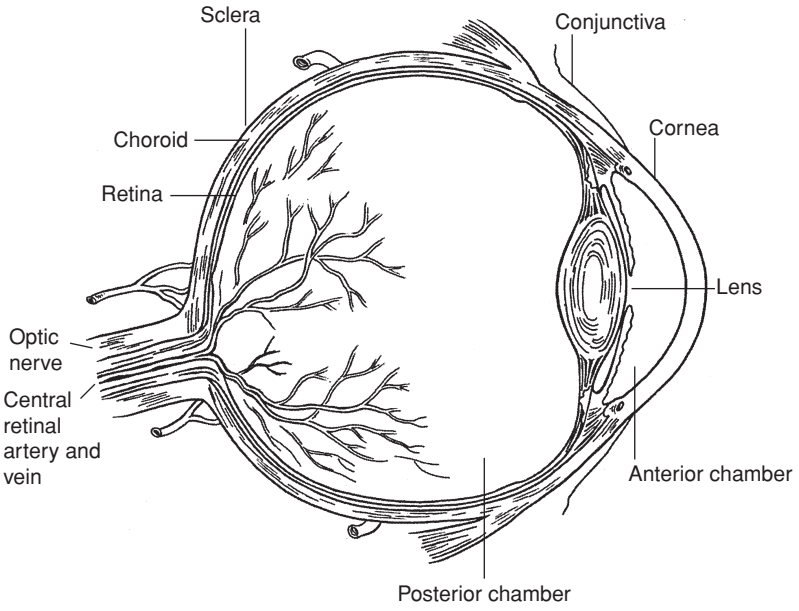

Fig. 14.1 The chambers of the eye.

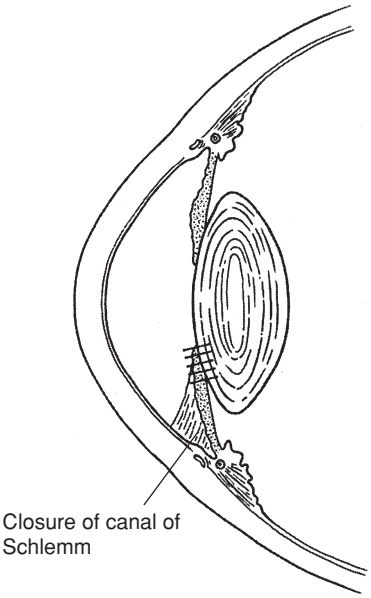

Fig. 14.2 The mechanism of acute closed angle glaucoma.

Acute glaucoma usually presents with severe pain in, and around, the eye. Visual changes include blurring, photophobia and marked impairment of acuity to such an extent that only light can be perceived. In addition, the patient experiences either nausea or vomiting.

The affected eye is red (ciliary congestion) with a cloudy cornea (oedema) and a mid-dilated, oval pupil that is unreactive to light. Urgent referral to an ophthalmologist is required.

Key points

Subacute glaucoma occurs with mild, transient episodes of intraocular hypertension. The patient may describe 'coloured haloes around lights' especially at night. This should be regarded as a danger signal indicating an imminent acute attack

Coloured haloes around lights can also occur as:

- **light diffuses through an early cataract**
- **a scintillating aura associated with migraine**

Time Out 14.2

Assess your knowledge by answering the following questions.

a What is the differential diagnosis in a patient presenting with a headache with extracranial signs?

b List the major complications associated with each condition.

HEADACHE WITH NO ABNORMAL SIGNS

In most patients presenting with an acute headache, no abnormal signs are detected (Table 14.7).

Table 14.7 Causes of headache with no abnormal signs

Tension	Daily
Migraine	Daily
Drugs	Daily
Toxins	Daily
Subarachnoid haemorrhage	Weekly
Giant cell arteritis	Monthly
Cluster headache	Annually
Coital migraine/cephalgia	Annually
Hyponatraemia	Annually

Tension type headache

This is common and also referred to as muscle contraction headache. Patients are usually aged 20–40 years, predominantly female (female/male = 3:1) and may describe either an acute or chronic history.

Acute

After an acute onset, the pain rapidly increases in severity over a few hours. The patient often appears pale and anxious with tachycardia, photophobia and neck stiffness (attributed to muscle spasm). These latter two clinical features mandate lumbar puncture to exclude meningitis. The main treatment is to reassure the patient, provide adequate analgesia and help to sort the emotional problems that are invariably associated.

Chronic

This common problem is the classic presentation of a tension type headache. The features are listed in Table 14.8. Patients can seek medical advice at any stage. Therefore, a comprehensive history is important to exclude other conditions, identify patient concerns (usually a brain tumour) and possible therapeutic avenues. Clinical examination provides some reassurance, but referral for

Table 14.8 Features of tension type headache

Site	Diffuse, but commonly at the vertex
	Often starts at forehead or neck
	Frequently bilateral
Character	Pressure sensation or pain
	Tight band, vice or clamp like, squeezing
	'As if my head is going to explode'
	'On fire or stabbing from knives or needles'
	Daily, increasing throughout the day
Radiation	Forehead to occiput or neck – vice versa
	Over the vertex or around the side – band like
Precipitation	Stress, anxiety
Relief	Nothing
Associated symptoms	Nausea, tiredness
Clinical examination	Often unremarkable

psychological advice may be necessary. Drug therapy has little to offer. The shorter the history, the better the chance for effective treatment.

Key points

Important discriminating factors in a patient with a tension headache:
headache starts each morning and increases in severity throughout the day
vomiting does not occur
visual disturbance does not occur

Migraine

This common condition affects approximately 20% of women and 15% of men, who usually present with paroxysmal headaches before the age of 30 years. There is often a family history, but the genetic basis remains unknown. Many patients will identify specific things that will precipitate an attack of migraine.

Common migraine precipitants

Dietary	**Fasting**
	Alcohol
	Specific foods
Drugs	**Oral contraceptives**
Affective	**Anxiety/stress**
	Post stress relaxation
Physiological	**Exercise**
	Menstruation
Visual	**Bright light**

Migraine appears to be of neural/cerebral origin. This primary event somehow triggers the release of substances that influence vasomotor tone and neuronal activity. The resultant distension of arteries in the scalp and dura causes pain, whilst decreased neuronal activity is responsible for the aura.

Migraine is often described as common, classic or variant. As its name suggests, most patients (75%) have the common variety and only 20% experience classic symptoms. However, all of these people will have prodromal features and paroxysmal headaches.

Prodromal features occur in the 24 h before the headache and comprise changes in mood, ranging from excess energy and euphoria to depression and lethargy. In addition there can be craving or distaste for specific foods.

Paroxysmal headaches can be either unilateral, bilateral or unilateral progressing to bilateral. They occur on wakening or during the day and are described as throbbing or pounding.

Patients with classic migraine can also have other presenting symptoms.

- **Visual aura** described as, e.g. flashing lights, fragmented images and micropsia. The aura lasts for 30 min and is followed by headache.
- **Sensory disturbances** can signify the onset of an attack, with numbness and tingling of one or both hands or the face, lips and tongue.
- **Motor disturbances** include weakness, hemiparesis and dysphasia.

- Most patients experience nausea, prostration and vomiting. Other somatic symptoms include shivering, pallor, diarrhoea, fainting and fluid retention.

The attacks gradually subside after 48 h. Some relief is gained from rest in a dark room, but either vomiting or sleep usually relieves the pain.

Immediate treatment

- Rest in a dark, quiet environment
- Analgesia – usually aspirin, paracetamol or a non-steroidal anti-inflammatory drug, such as diclofenac or piroxicam melt.
- Antiemetics, e.g. metoclopramide or domperidone, can be given as either suppository or intravenous preparations.
- Sumatriptan (or other triptan), a selective 5HT1 antagonist, can be used (oral, subcutaneous, intranasal) if the patient either has failed to respond to analgesia and/or is vomiting. A 6 mg subcutaneous injection of sumatriptan provides prompt, effective relief in nearly 70% of patients. Only one dose should be given for each attack. However, if a second attack occurs, a further 6 mg can be given, provided at least 1 h has elapsed following the first dose (maximum: 12 mg in 24 h).

Key points

Sumatriptan is contraindicated in patients with ischaemic heart disease

Prophylactic treatment

- Seek and exclude known precipitants.
- Treat precipitants appropriately, e.g. amitriptyline if anxiety/stress related; atenolol for the tense patient.
- Oral pizotifen (1.5 g at night) is useful if a specific precipitant cannot be identified.

Key points

Migraine should not be:
diagnosed in anyone presenting for the first time over the age of 40 years, until other conditions have been excluded
confused with tension headache (non-paroxysmal, no vomiting or visual features)

Drugs

A variety of drugs can cause headache because of their effect on vascular muscle tone, e.g. nitrates and calcium channel antagonists. A medical history will establish the link and provocation tests are rarely required.

Key points

Caffeine is often added to analgesic preparations to enhance their effect. Remember that the addition or withdrawal of caffeine can also cause headache

Toxins

Alcohol: Both alcohol excess and withdrawal can cause headache.

Subarachnoid haemorrhage

Always consider this condition for any patient with an unexplained headache of acute onset. Please see Chapter 11 for further information.

Giant cell arteritis

The differential diagnosis of a sudden headache, in any patient over the age of 50 years, includes giant cell arteritis as described earlier in this chapter.

Cluster headache

This distinctive condition comprises:

- unilateral headache with ipsilateral:
  - corneal injection and epiphora (90%)
  - nasal congestion or rhinorrhoea (90%)
  - transient Horner's syndrome (25%).

It can be present at any age, commonly between 20 and 50 years, and predominantly affects men (male: female = 10:1). The headache is centred around the orbit and is described as severe, boring or stabbing with radiation to the forehead, temple or cheek and jaw. Brief bouts of this unilateral pain last 30–120 min each day for between four and 16 weeks. Characteristically, the pain occurs shortly after the onset of sleep, although it can occur during the day. During attacks the patient is usually crying, restless and prefers walking. The cause remains unknown, but alcohol can precipitate an attack as can other vasodilators.

Oxygen delivered through a face mask at a dose of 8 l/min for 10 min, early on during an attack, often terminates or diminishes the intensity of the attack. This is postulated to be the result of oxygen being a vasoconstrictor leading to increased production of serotonin in the CNS.

Prophylactic ergotamine should be given approximately 1 h before an attack. Suppositories are the most useful preparation and should be continued for one week. If the headache recurs, treatment should be restarted on a weekly basis until the cluster ends. Oral sumatriptan and verapamil are alternatives if the patient does not respond to ergotamine.

Coital migraine/cephalgia

This is a severe headache that begins suddenly during sexual intercourse or immediately following orgasm. It is more common in males and nearly 50% will have a previous history of migraine. Some patients require propranolol 40–80 mg before intercourse. This drug can be stopped once the patient has remained asymptomatic for one month.

Key points

Subarachnoid haemorrhage must be excluded in patients who present with coital migraine

Hyponatraemia

This can be associated with headache, nausea, vomiting and weakness. The diagnosis and management are considered in detail in Chapter 26.

Time Out 14.3

a List the features that would differentiate between tension headache, migraine and cluster headache.

b In a patient presenting with headache and no abnormal signs, under what circumstances would you consider doing a lumbar puncture?

SUMMARY

Headache of acute onset accounts for less than 2.5% of new emergency attendances. Of these, 15% will have an immediately life-threatening condition. These need to be identified and treated in the primary assessment. Some of the remaining patients will have sinister pathology. The characteristics of headache that suggest a serious underlying cause are:

- new onset
- acute onset
- progressive
- wakens from sleep
- worst ever.

Important associated signs that should be sought include:

- photophobia
- meningeal irritation
- fever
- altered mental state
- neurological dysfunction.

Physical examination should be thorough, with particular emphasis on:

- meningeal irritation
- papilloedema
- pyrexia
- pericranial signs
- focal neurological features
- rash.

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CHAPTER 15

The patient with abdominal pain

OBJECTIVES

After reading this chapter you should be able to describe:

- the different mechanisms of abdominal pain
- the primary assessment and resuscitation of the patient with abdominal pain
- the associated secondary assessment
- emergency treatment and definitive care.

INTRODUCTION

Abdominal pain is a common complaint and can be the presenting symptom of a wide range of conditions which have their origin both inside and/or outside the abdomen. Making an accurate diagnosis and starting appropriate treatment for the patient with abdominal pain may be difficult. Although the majority do not have an immediately life-threatening problem, identification of those who require urgent investigation and treatment is essential to avoid preventable morbidity and mortality. The structured approach gives priority to life-threatening conditions and initial resuscitation, and a PHRASED history and examination are particularly important in the patient with abdominal pain.

ANATOMY AND PATHOPHYSIOLOGY

The basic pathological processes in intra-abdominal causes of abdominal pain are:

- inflammation (e.g. gastroenteritis, appendicitis, pancreatitis, pyelonephritis)
- perforation (e.g. peptic ulcer, carcinoma of the colon)
- obstruction (e.g. intestine, bile duct, ureter)
- haemorrhage (e.g. leaking aortic aneurysm, bleeding ulcer, ectopic pregnancy)
- infarction (e.g. bowel, spleen).

An alternative classification is based on the system affected (see the next four boxes):

- Gastrointestinal causes
- Vascular causes
- Urological causes
- Gynaecological/obstetric causes

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Gastrointestinal causes of abdominal pain

Non-specific abdominal pain	Daily
Gastroenteritis	Daily
Acute appendicitis	Daily
Gall stone disease and acute cholecystitis	Weekly
Peptic ulcer disease	Weekly
Intestinal obstruction	Weekly
Diverticular disease	Weekly
Pseudoobstruction	Monthly
Acute pancreatitis	Monthly
Perforated viscus	Monthly
Hernia (incarcerated/strangulated)	Monthly
Hepatitis	Monthly
Malignancy (carcinoma, lymphoma)	Monthly

Vascular causes of abdominal pain

Leaking abdominal aortic aneurysm	Monthly
Infarction of bowel, kidney or spleen	Monthly
Inflammatory bowel disease	Monthly
Primary infective peritonitis	Only in exams

Urological causes of abdominal pain

Lower urinary tract infection/pyelonephritis	Daily
Ureteric/renal colic	Daily
Acute urinary retention	Daily
Testicular torsion	Monthly

Gynaecological/obstetric causes of abdominal pain

Miscarriage	Daily
Pelvic inflammatory disease	Daily
Ovarian cyst	Monthly
Ectopic pregnancy	Monthly
Labour	Monthly
Retained products of conception	Annually
Placental abruption	Annually
Red degeneration of a uterine fibroid	Only in exams

Abdominal pain may also arise from the retroperitoneum, the pelvis and, occasionally, outside the abdomen (see box below).

Extraabdominal causes		
Psychosomatic (including drug seeking behaviour)		**Monthly**
Chest (myocardial infarction, basal pneumonia, pulmonary embolus)		**Monthly**
Haematological (sickle cell crisis, haemophilia)		**Annually**
Metabolic (diabetic ketoacidosis, Addisonian crisis, uraemia, porphyria, drug withdrawal symptoms)		**Annually**
Neurological	**– spine/disc disease**	**Weekly**
	– radiculopathy	**Monthly**
	– herpes zoster	**Annually**
Vasculitis (e.g. polyarteritis nodosa, systemic lupus erythematosus)		

This classification of abdominal pain by system is neither comprehensive nor user friendly in clinical practice. An alternative classification – which may be clinically more useful – is to consider three mechanisms by which abdominal pain may occur:

- visceral pain
- parietal pain
- referred pain.

Visceral pain

Visceral pain is characteristically caused by inflammation, ischaemia, neoplasia and distension of either the wall of a hollow viscus or the capsule of a solid intra-abdominal organ. Most gastrointestinal organs are served by afferent nerves from both sides of the spinal cord. Visceral pain is usually perceived in the midline, transmitted via autonomic nerve fibres in the wall or capsule of the organ; and the site of pain is characteristically poorly demarcated and may not correspond to the site of tenderness on examination. Pain is often described as cramp-like, colicky, dull, burning or gnawing, and associated with autonomic features such as nausea, vomiting, pallor and sweating.

The pain is usually localised to one of three regions according to the embryological origin of the organ involved.

- Foregut structures – stomach, proximal duodenum, liver, gall bladder and pancreas – characteristically produce pain in the epigastrium.
- Midgut structures – distal duodenum, small intestine, appendix and ascending and proximal part of the transverse colon – produce periumbilical pain.
- Hindgut structures – descending colon, kidneys, bladder, ureter and pelvic organs – produce pain that is characteristically felt in the hypogastrium and/or lower back.

Parietal pain

Parietal or somatic pain is characteristically caused by inflammation (bacterial or chemical) of the parietal peritoneum. It is mediated by segmental spinal nerves associated with specific dermatomes. Consequently the pain is more precisely localised to the structure from which the pain originates. This corresponds to the site at which tenderness and guarding develop. The pain is also characteristically sharper and is aggravated by movement, coughing and sometimes breathing.

Referred pain

Referred pain is localised to a site distant from the organ that is the source of pain. The organ involved and the site at which the pain is felt share a common embryological origin and associated peripheral nerves share a common segmental origin. A classical example is pain felt in the shoulder tip, supraclavicular area and side of the neck due to irritation (by blood or pus) of the diaphragm that is derived from the fourth cervical segment. Pain may be referred to the abdomen from the chest (e.g. inferior myocardial infarction, basal pneumonia), the back and external genitalia.

PRIMARY ASSESSMENT AND RESUSCITATION

The presence of a rapidly life-threatening condition presenting with abdominal pain should be recognised by the end of the primary assessment (see box).

Life-threatening conditions in the patient with abdominal pain

Hypovolaemic shock:	
• gastrointestinal bleeding	Daily
• leaking abdominal aortic aneurysm/aortic dissection	Monthly
• ectopic pregnancy	Monthly
• splenic rupture (usually traumatic but may be spontaneous)	Annually
Acute pancreatitis	Monthly
Small bowel infarction	Monthly
Sepsis (e.g. following perforation of colon)	Monthly
Acute myocardial infarction/acute right heart failure	Monthly
Diabetic ketoacidosis	Monthly

Patients with abdominal pain generally have a patent **airway**. It is important to be aware of the risk of aspiration with profuse vomiting, particularly if the conscious level is reduced. A nasogastric tube should be passed early in patients with small bowel obstruction to drain fluid and air and reduce the risk of aspiration.

In the patient with abdominal pain, abnormalities on **breathing** assessment such as tachypnoea and signs of hypoxaemia (including low oxygen saturation) may occur for a variety of reasons. Severe abdominal pain may cause splinting of chest movement. There may be symptoms and signs of pathology within the chest (causing pain localised to the upper abdomen), such as basal pneumonia. Other clinical abnormalities (e.g. tachypnoea) may be a manifestation of shock (due to intra-abdominal or retroperitoneal haemorrhage) or sepsis. Deep sighing respiration (Kussmaul's respiration) suggests metabolic acidosis (e.g. diabetic ketoacidosis or sepsis).

Initial treatment is with high concentrations of inspired oxygen via a facemask with a non-rebreathing reservoir bag. However, intubation and ventilation may be required if oxygenation is inadequate despite supplemental oxygen, e.g. in patients with severe acute pancreatitis who develop acute respiratory distress syndrome or in patients with septic shock.

An immediately life-threatening condition in the patient with abdominal pain most commonly becomes apparent when assessing circulation (see next box and Chapter 6).

Assessment of circulation: signs of circulatory failure

Pulse: tachycardia, low volume
Blood pressure: hypotension (but remember systolic blood pressure may be normal in a patient who has lost up to 30% of circulating volume);
Pulse pressure is narrow in hypovolaemic shock (>Grade II)
Cerebral perfusion: agitation, confusion
Peripheral perfusion: pallor, cool extremities, sweating, prolonged capillary refill time

Occult bleeding can occur in the gut lumen, peritoneal cavity or retroperitoneum. While signs of hypovolaemia occurring early after the onset of pain suggest haemorrhage, hypovolaemia may occur later due to loss of extracellular fluid:
- with diarrhoea and vomiting
- into obstructed bowel
- into inflamed retroperitoneum or peritoneal cavity.

Patients with septic shock are classically vasodilated and warm with hypotension and a fever, but many (particularly if presenting late) are peripherally vasoconstricted and have a normal or even low body temperature. Atrial fibrillation is a risk factor for mesenteric artery embolism.

Key point

If a patient over 50 years has a clinical presentation of 'ureteric colic', always consider the possibility of aortic aneurysm or dissection, particularly if there is any sign of circulatory failure

Insert two large-bore (14-gauge) peripheral intravenous cannulae. Take blood for baseline full blood count, biochemistry (including glucose stick test and amylase), blood crossmatch or save, coagulation screen, blood cultures and, when appropriate, sickle screen and pregnancy test.

If hypovolaemia due to blood loss is suspected, start fluid resuscitation with 2 litres of warmed crystalloid, followed by blood; if there is evidence of anaemia as well as circulatory failure, it may be preferable to substitute blood for crystalloid earlier.

A portable ultrasound scan (if available) may confirm the cause of hypovolaemic shock (e.g. a leaking aortic aneurysm or an ectopic pregnancy). This should not delay urgent referral to a surgeon or gynaecologist when prompt surgery may be life saving (see next two boxes).

Indications for urgent referral to a surgeon

Abdominal pain or tenderness plus a pulsatile mass and/or a history of aortic aneurysm, or a leaking aortic aneurysm suspected for any other reason
Gastrointestinal bleeding in a patient of 60 years or over or in a patient of any age with signs of shock, haemoglobin less than 10 g/dl, significant coexistent disease or varices
Any evidence of free intraperitoneal fluid in the patient with abdominal pain and signs of circulatory failure
Suspected pancreatitis
Suspected mesenteric ischaemia
Suspected testicular torsion

Indications for urgent referral to a gynaecologist

Suspected ectopic pregnancy
Suspected torsion or rupture of an ovarian cyst

Key point

Urgent surgical (or gynaecological) referral of the patient with abdominal pain and shock may be life saving; do not wait for the results of investigations

If a diagnosis of either myocardial infarction or pulmonary embolism is possible, investigate and start specific treatment (see Chapters 8 and 10). If septic shock (see Chapter 9) is suspected, treat with intravenous broad-spectrum antibiotics before the result of cultures is known.

If there is any depression of conscious level in the patient with abdominal pain, check that hypoxaemia and shock are being adequately treated, and consider hypoglycaemia, diabetic ketoacidosis and sepsis.

Time Out 15.1

a What is the differential diagnosis of abdominal pain and shock in (i) a 75-year-old man and (ii) a 25-year-old woman?
b What are the management priorities?

SECONDARY ASSESSMENT

A PHRASED history and careful examination are important for making a diagnosis in the patient with abdominal pain. An improvement in diagnostic and decision making skills has been attributed to the use of computer-assisted diagnosis based on a proforma (Fig. 15.1). This ensures more effective collection of information from the patient's history and examination findings.

'Phrased' History

The well-'phrased' history may be applied to the patient with abdominal pain.

Problem

It is important to establish the patient's main complaints, particularly as abdominal pain may be a presenting symptom of such a wide variety of conditions.

History of presenting problem

The **site**, any change in location and **radiation** of pain are important. Inquire about the site of pain at onset in particular: pain which is lateralised from the outset is consistent with pain from a paired structure (e.g. kidney, ureter, gonad), whereas pain which is felt centrally (or bilaterally) is consistent with a gastrointestinal cause. The level of early pain – epigastric, periumbilical or hypogastric – may suggest the affected viscus, pain being referred to the segment corresponding to the root level of the afferent nerves to the organ involved. Ill-defined pain may indicate visceral pain early in the disease process, referred pain or a metabolic,

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Abdominal Pain Chart

NAME REG NUMBER

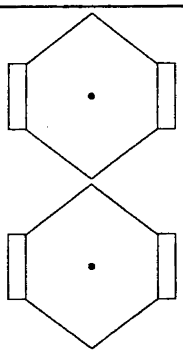
MALE FEMALE AGE FORM FILLED BY

PRESENTATION (999, GP. etc) DATE TIME

PAIN

SITE

ONSET



PRESENT

RADIATION

AGGRAVATING FACTORS
movement
coughing
respiration
food
other
none

RELIEVING FACTORS
lying still
vomiting
antacids
food
other
none

PROGRESS
better
same
worse

DURATION

TYPE
intermittent
steady
colicky

SEVERITY
moderate
severe

HISTORY

NAUSEA
yes no

VOMITING
yes no

ANOREXIA
yes no

PREV INDIGESTION
yes no

JAUNDICE
yes no

BOWELS
normal
constipation
diarrhoea
blood
mucus

MICTURITION
normal
frequency
dysuria
dark
haematuria

PREV SIMILAR PAIN
yes no

PREV ABDO SURGERY
yes no

DRUGS FOR ABDO PAIN
yes no

♀ LMP

pregnant

vag. discharge

dizzy/faint

EXAMINATION

MOOD
normal
distressed
anxious

SHOCKED
yes no

COLOUR
normal
pale
flushed
cyanosed

TEMP PULSE

SP

ABDO MOVEMENT
normal
poor nil
peristalsis

SCAR
yes no

DISTENSION
yes no

TENDERNESS

REBOUND
yes no

GUARDING
yes no

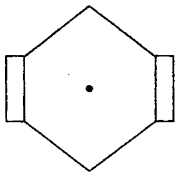
RIGIDITY
yes no

MASS
yes no

MURPHY'S
+ve –ve

BOWEL SOUNDS
normal absent + + +

RECTAL – VAGINAL TENDERNESS
left
right
general
mass
none



History and examination of other systems on separate case notes

INITIAL DIAGNOSIS & PLAN

RESULTS
amylase
blood count (WBC)
computer
urine
X-ray
other

DIAG & PLAN AFTER INVEST

(time)

DISCHARGE DIAGNOSIS

Fig. 15.1 Abdominal pain chart.

toxic or psychological cause. Migration of pain is characteristic of an inflamed viscus; e.g. pain migration from the periumbilical region to the right lower quadrant in acute appendicitis or from the epigastrium to the right upper quadrant in cholecystitis.

Pain arising from the stomach or duodenum is characteristically localised to the epigastrium in the midline. Pain from the gall bladder is also felt in the epigastrium and/or right upper quadrant, sometimes radiating to the area below the inferior angle of the right scapula (dorsal segmental radiation). Pain from pancreatitis is generally felt in the upper abdomen, with radiation through to the back. Other important conditions to consider in the patient who reports pain radiating to the back are leaking aortic aneurysm and renal or ureteric disease. Small bowel pain is characteristically felt symmetrically, centrally and may radiate to the back, while pain from the large bowel is felt in the hypogastrium and may radiate to the back and thighs.

Sudden **onset** of severe pain is characteristic of either a vascular problem such as a leaking abdominal aortic aneurysm, torsion of a gonad or a perforated viscus. The pain of acute pancreatitis may come on relatively rapidly, but over minutes rather than seconds. Whereas pain due to an inflammatory condition, such as diverticulitis or appendicitis, progresses more gradually, over hours or days. Collapse associated with the onset of pain suggests leaking aortic aneurysm, acute pancreatitis, perforated ulcer or (in females) ruptured ectopic pregnancy.

Note the **duration** of pain and whether it can be characterised as steady (present all the time at a similar intensity – e.g. bowel infarction), *intermittent* (pain resolves for periods of time – e.g. pain from gastroenteritis) or *colicky* (present all the time with fluctuating intensity). Determine whether the pain is improving, worsening or remaining much the same over a period of 1–2 h or more.

Somatic pain is characteristically described as sharp, whereas vague terms are used for visceral pain. Otherwise, it is difficult to draw diagnostic inferences from the description of the **character** of abdominal pain by patients who may use a variety of terms in different ways. The **severity** of pain may be assessed by the patient's own account and by observation of the patient who may appear distressed, sweating or crying out. Although conditions such as a perforated viscus, bowel infarction or acute pancreatitis often cause severe pain, this does not reliably distinguish them from non-specific abdominal pain.

Ask about the main **exacerbating/relieving** factors – *movement* (particularly movements which cause the patient to tense the anterior abdominal wall muscles and movement of inflamed peritoneal surfaces), *coughing and deep inspiration*. The effect on pain of vomiting (which may provide transient relief in small intestinal obstruction), food and antacids should also be noted. Also inquire about any relationship to position and to micturition.

Other gastrointestinal symptoms including anorexia, nausea and vomiting are common and relatively non-specific. The frequency of vomiting and its relationship to the onset of pain may be significant; pain before nausea and vomiting suggests a surgical cause (such as peritonitis and obstruction), whereas nausea and vomiting followed by pain is more characteristic of gastroenteritis. Ask about blood or bile in the vomit; faeculent vomiting indicates intestinal obstruction. Pre-existing indigestion may lead to a diagnosis of a perforated or bleeding peptic ulcer and previous symptoms of cholelithiasis suggest a cause for acute pancreatitis.

Diarrhoea and constipation may also be non-specific (either may occur in patients with appendicitis). In common with vomiting, diarrhoea from the beginning of an illness is characteristic of gastroenteritis, whereas onset after several hours may occur in appendicitis and peritonitis. Ask about blood or mucus in

diarrhoea (inflammatory bowel disease, ischaemic bowel or malignancy). Change in bowel habit over a period of time raises the possibility of malignancy especially in a patient aged 55 years or over.

Abdominal pain, vomiting and absolute constipation are virtually pathognomonic of intestinal obstruction. Early vomiting and more frequent episodes of colicky abdominal pain are features of more proximal small bowel obstruction; on arrival at hospital the patient may not yet be aware of constipation. By contrast, constipation will be prominent in the patient with large bowel obstruction.

Relevant medical history

The past history should include details of:

- similar episodes of pain, investigation (leading to a diagnosis of inflammatory bowel disease, for example), treatment and outcome
- previous abdominal surgery (with possible subsequent adhesions, the leading cause of small bowel obstruction)
- other conditions – including diabetes mellitus; cardiac, cerebrovascular and respiratory disease; and psychiatric illness – may be relevant to the cause of abdominal pain or complicate its treatment.

Allergies

Note any previous allergies and adverse reaction to drugs, particularly antibiotics, analgesics and, if surgery is a possibility, topical antiseptics, dressings and anaesthetic agents.

Systems review

- Burning dysuria, frequency and urgency, with or without haematuria, are characteristic of urinary tract infection, but an inflamed appendix or diverticulum adjacent to the ureter or bladder may cause urinary symptoms and pyuria. Establish whether dysuria is an exacerbation of the patient's abdominal pain or a different pain.
- A gynaecological history should be taken from women with abdominal pain, to include information about pregnancies, menstrual pattern, contraception, abnormal vaginal discharge, and – if pelvic inflammatory disease is suspected – new or multiple sexual partners.
- Ask patients with upper abdominal pain in particular about chest pain, shortness of breath, cough and haemoptysis. Coexistent cardiovascular disease may be a clue to the diagnosis of intra-abdominal vascular pathology.

Essential family and social history

A family history of intra-abdominal conditions (such as inflammatory bowel disease or carcinoma) or of an inherited condition (Marfan's, sickle cell disease, acute intermittent porphyria, haemophilia) and ethnic origin may be relevant to diagnosis and treatment. A group of people affected by vomiting, diarrhoea and abdominal pain suggests an infectious agent or carbon monoxide poisoning.

Drugs

This part of the history should include medicines taken either for the present problem or other conditions (particularly corticosteroids, non-steroidal anti-inflammatory drugs, anticoagulants and antibiotics), alcohol consumption and drug use.

Examination

Objectives of examination include:

- assessment of the patient's general condition – the ABCDEs should be re-assessed
- localisation of an intra-abdominal source of pain (generally related to the area of maximum tenderness)
- detection of any extra-abdominal cause of pain.

General examination

Pallor, sweating and signs of distress in a patient with abdominal pain suggest (but are not diagnostic of) a more serious cause for abdominal pain, such as a vascular event, perforated viscus or acute pancreatitis. Classically, patients with visceral pain (e.g. ureteric colic) roll around. In contrast, those with peritonitis lie immobile, showing signs of pain if the bed on which they are lying is inadvertently knocked. The lethargic patient may be septic.

Mucous membrane pallor may indicate anaemia due to chronic blood loss. Look at the sclerae for jaundice. Stigmata of chronic liver disease (spider naevi, palmar erythema, Dupuytren's contracture, leuconychia and clubbing, abnormal veins around the umbilicus, loss of body hair, gynaecomastia and testicular atrophy, ascites and signs of encephalopathy) may provide useful clues to the cause of either upper gastrointestinal bleeding or abdominal distension. Features of uraemia in patients with abdominal pain are rare. The pigmentation of Addison's disease, seen in scars, the flexor creases of the palm, over pressure areas and on the buccal mucosa opposite the molar teeth, may be missed if not actively sought. Purpura in characteristic distribution over the lower limbs will suggest the possibility of Henoch–Schönlein purpura, but abdominal pain can precede the rash; petechiae can represent an underlying haematological abnormality; both mandate consideration of meningococcal septicaemia. Erythema nodosum is one of the extra-intestinal manifestations of inflammatory bowel disease and photosensitivity is a feature of porphyria.

Pyrexia is significant. A normal temperature, particularly in the elderly, does not exclude conditions such as cholecystitis and appendicitis; although perforation of the appendix is usually associated with a temperature greater than 38°C. A temperature above 38.5°C, especially with a history of rigors, is a common feature of bacterial infections, such as pyelonephritis, acute salpingitis or ascending cholangitis. A furred tongue and foetor are common and non-specific; the smell of acetone, if detected, may facilitate the rapid diagnosis of diabetic ketoacidosis. Signs of dehydration imply extracellular fluid loss due to gastroenteritis, diabetic ketoacidosis or intestinal obstruction.

Rapid shallow respiration may be a feature of either peritonitis or pneumonia. Look also for the deep (Kussmaul's) sighing respiration of diabetic ketoacidosis or other metabolic acidoses. Signs of circulatory failure due to hypovolaemia would usually have been detected in the primary assessment; other causes of tachycardia include sepsis, untreated pain and anxiety.

A brief systematic examination of the cardiovascular system (including peripheral pulses), the chest and back is important to identify any extra-abdominal cause of pain and to assess the patient's general condition.

Abdominal examination

Adequate exposure is vital if subtle but important signs (such as a small incarcerated femoral hernia) are not to be missed.

On **inspection** look for:

- the contour of the abdomen (particularly distension) and any gross deformity
- visible peristalsis (suggesting intestinal obstruction) or the abdominal wall held immobile in peritonitis; get the patient to move the anterior abdominal wall by coughing and watch the patient's facial expression. (If there is peritonitis, coughing or any movement of the bed is likely to cause sharp pain due to the movement being transmitted to the inflamed peritoneum.)
- scars from previous surgery (which may be the clue to adhesions as the cause of small bowel obstruction)
- visible pulsation of an aneurysm
- discolouration or bruising around the umbilicus (Cullen's sign) or in the flank (Grey Turner's sign) which are rare but important features of haemorrhagic pancreatitis; retroperitoneal haemorrhage from an aortic aneurysm may also produce flank bruising.

Palpation (with a warm hand) should be gentle and start in an area of the abdomen away from the site of pain. Distinguish between the symptom of pain and the sign of tenderness on examination, and the site of each. Localised tenderness suggests the site of an inflammatory source of the patient's pain. However, it may be absent in appendicitis, either because the pain is visceral early in the inflammatory process or because the appendix is retrocaecal. Conversely, abdominal tenderness may be present in biliary colic, small bowel obstruction or gastroenteritis. A particular pitfall for the unwary is the elderly patient with bowel infarction due to mesenteric artery thrombosis or embolism: characteristically these patients present with pain of sudden onset which is steady and severe, apparently out of proportion to the limited tenderness on examination. If the diagnosis is not suspected there may be a dangerous delay in arranging surgery.

The presence of guarding (reflex contraction of the abdominal wall muscles in response to palpation) and rebound tenderness suggest local peritonitis and rigidity is a sign of generalised peritonitis.

Palpate for any masses and/or organomegaly. A distended bladder may be identified on palpation or percussion.

Percussion may give information about the size of solid organs and distinguish between gas and fluid as the cause of abdominal distension (shifting dullness in ascites). Percussion tenderness may identify signs of localised or generalised peritonitis, in which case subsequent examination should be modified to avoid unnecessary painful palpation.

On **auscultation**, loud high pitched (tinkling) bowel sounds suggest obstruction and absent bowel indicate peritonitis (e.g. perforated viscus) or ileus; neither sign is sensitive and normal bowel sounds do not exclude serious intra-abdominal pathology.

Listen for bruits over the upper quadrants of the abdomen and costovertebral angles (for renal artery stenosis or aneurysm).

Inflammation close to the psoas muscle may be confirmed by stretching the psoas muscle. Lie the patient on the unaffected side and then passively extend the thigh at the hip on the affected side. In a different manoeuvre, passive rotation of the flexed thigh to the limit of internal rotation. This may produce pain in the hypogastric region, if there is a perforated appendix, abscess or other collection overlying the fascia of obturator internus in the pelvis.

Examination of the abdomen should include examination of the flanks, the external genitalia (in particular the scrotum and testes) and the inguinal and femoral canals for herniae; an incarcerated hernia is the second most common cause of small bowel obstruction. **Vaginal examination** – when indicated – may

provide information about gynaecological causes of abdominal pain or an inflamed appendix palpable in the pelvis.

On **rectal examination**, assess for perianal disease (in inflammatory bowel disease), pelvic tenderness, abnormal masses, blood or melaena, sphincter tone and, where relevant, the prostate.

Investigations

All patients with abdominal pain should have a **blood glucose stick test** (to exclude diabetes) and **urinalysis**. Microscopic haematuria can occur with either ureteric colic or urinary tract infection (but remember also the possibility of infective endocarditis, symptoms of which include abdominal pain). Pyuria (more than 5–10 white cells/cubic mm) is commonly due to urinary tract infection but it may also be due to inflammation of an adjacent organ, such as appendicitis or diverticulitis. In the presence of jaundice or suspected biliary tract disease test the urine for urobilinogen and bilirubin. A **urine pregnancy test** (for the β subunit of human chorionic gonadotrophin) should be done in all women of childbearing age, regardless of the history.

On the **full blood count,** a raised white cell count supports the diagnosis of a significant cause for abdominal pain but is neither sensitive nor specific for a surgical condition. For example, a patient with an acute appendicitis may have a normal white cell count, while a raised white cell count may be due to pyelonephritis or other bacterial infection. Nevertheless, a white cell count of greater than 15×10^9/l in a patient with acute pancreatitis is one factor associated with increased mortality (see box of adverse prognostic factors in the section on acute pancreatitis). The haemoglobin concentration does not reflect acute blood loss but may indicate chronic bleeding where serial values may be useful.

A **serum amylase** of greater than five times the upper limit of normal, suggests acute pancreatitis, but a normal amylase does not exclude pancreatitis and a lesser rise may be seen in a variety of conditions which cause abdominal pain (including cholecystitis and peptic ulcer). A raised serum lipase may be a better test, but is not always available. Estimation of urea and electrolytes does not usually contribute to the diagnosis of the patient with abdominal pain, but forms part of the assessment of the general condition of any patient who is haemodynamically unstable or dehydrated, e.g. due to vomiting or diarrhoea. A **liver enzyme profile and prothrombin time** are required for patients who are jaundiced. Baseline coagulation studies are indicated in the patient who is bleeding, has a suspected coagulopathy or requires blood transfusion.

An **arterial blood gas** sample will assess oxygenation and acid–base status in the patients with hypovolaemia, pancreatitis or a suspected pulmonary problem.

An **electrocardiogram** is recommended in patients over the age of 40 (or younger if there is a specific indication), as either acute myocardial infarction or pulmonary embolism can cause abdominal pain. In addition, an ECG may identify coexistent cardiac disease which predisposes to an intra-abdominal vascular event (e.g. atrial fibrillation leading to mesenteric artery embolism) or complicates treatment.

The role of **radiology** is primarily to confirm or refute a diagnosis suspected on clinical assessment and is not a substitute for an effective history and clinical examination.

Plain X-rays

The principal indications for plain X-rays in patients with abdominal pain are suspicions of one of the following:

- intestinal obstruction
- perforated viscus
- toxic megacolon
- foreign body
- ureteric/renal colic
- chest pathology.

The abdominal film may also yield useful information in peritonitis and/or suspected mesenteric ischaemia, but should not be used indiscriminately in patients with abdominal pain.

Standard views are the erect chest X-ray (CXR) and the supine abdominal film. The erect CXR should be taken after the patient has been sitting upright for 5–10 min (after which as little as 1–2 ml of free air may be shown); it may also show pneumonia or other pathology in the chest. If the patient is unable to stand or sit, the left lateral decubitus view of the abdomen is an alternative when looking for 'free gas'. An unsuspected abdominal aortic aneurysm may be outlined by a calcified vessel wall. The erect abdominal view does not generally add useful information. An unprepared barium enema may be required urgently to elucidate the cause of suspected large bowel obstruction and to exclude pseudo-obstruction.

A KUB (kidney, ureter, bladder) X-ray is the initial imaging for patients with ureteric colic. Although more than 80% of ureteric calculi are radiopaque, they are often missed on plain X-ray. This film should be followed by either an intravenous urogram (IVU), ultrasound scan or more commonly computed tomography (CT) to demonstrate the size and location of a stone and the extent of obstruction to the ureter and kidney.

Ultrasound

Urgent ultrasound scan is indicated in the following situations:

- suspected abdominal aortic aneurysm
- right upper quadrant pain, jaundice or suspected cholelithiasis
- acute renal failure
- suspected urinary tract colic or obstruction, particularly if there is any contraindication to an intravenous urogram
- lower abdominal pain in women of childbearing age
- suspected intra-abdominal abscess/bleeding.

Visualisation of a normal appendix may be useful in ruling out appendicitis.

Computed tomography

This is an important urgent investigation, but the patient must be well enough to be moved to the CT suite. It is particularly useful:

- as an alternative for imaging suspected urinary tract obstruction
- in suspected perforation of a viscus
- to visualise retroperitoneal structures, including the pancreas and aorta
- in the subsequent investigation of selected patients with acute abdominal pain, to help with making a definitive diagnosis and planning treatment.

In many countries non-contrast CT (CT urogram) is now the initial radiological investigation of choice for suspected urolithiasis. It is quicker than IVU, avoids the use of contrast and can identify other pathologies, e.g. abdominal aortic aneurysm.

Angiography and labelled red cell scans

These have a beneficial role in evaluating intestinal ischaemia and gastrointestinal haemorrhage (after negative endoscopy of the upper and lower tracts). The choice

of investigation may be influenced by the rate of bleeding as labelled red cell scans are more sensitive.

Potential pitfalls in the assessment of patients with abdominal pain

Elderly patients with acute abdominal pain have a higher mortality. Several factors may contribute to this, some of which put the elderly at risk of delayed diagnosis:

- Different spectrum of disease: a greater proportion have a malignancy or a vascular cause for pain (which may not initially be recognised).
- General peritonitis may be due to a perforated colon rather than a perforated ulcer or appendix.
- A different presentation of intra-abdominal disorders: in comparison with younger patients, the pain is often not as marked. Fever, tachycardia and leucocytosis are uncommon with inflammatory conditions such as appendicitis. These factors can lead to a delay in diagnosis and an increased risk of perforation.
- Delayed presentation is more common.
- Coexistent illness is likely to make the elderly more vulnerable to complications.

Key point

Glucocorticoids can mask both clinical and laboratory responses to inflammation or perforation of a viscus in the abdomen, including the degree of pain and tenderness and fever

Similarly other immunosuppressive drugs, coexistent diabetes mellitus and immunodeficiency can influence the patient's response to inflammatory conditions. These patients may display minimal clinical signs and normal laboratory tests, despite a serious intra-abdominal disorder.

Finally, the clinical features of surgical conditions may be unexpectedly nonspecific in late pregnancy.

EMERGENCY TREATMENT

After the primary assessment, a well-'phrased' history and examination of the patient, a differential diagnosis can be formulated and appropriate investigations requested. Treatment should be initiated simultaneously with assessment and investigation. Consider:

- analgesia
- review of fluid resuscitation
- antiemesis and nasogastric suction
- antibiotics
- urethral catheter.

Analgesia

Early judicious analgesia is advocated for any patient with acute abdominal pain, including those who require referral for a surgical opinion. If opioid analgesia is given as a dilute solution by slow intravenous injection, the dose can be titrated against the patient's pain.

Key point

Acute abdominal pain is not a contraindication to opioid analgesia

Adequate analgesia reduces suffering with no evidence that appropriate analgesia makes diagnosis of a surgical condition more difficult, providing the patient's condition is reviewed regularly and that necessary investigations are done. A patient who is not distressed is more likely to give a clearer coherent history and cooperate with an examination.

Review fluid resuscitation

Reassess the patient for signs of intravascular volume depletion and the response to fluid resuscitation.

Dehydration in patients with acute abdominal pain may be due to a combination of factors including vomiting and diarrhoea, inadequate oral intake and 'third space' loss (including loss into the bowel lumen or retroperitoneum). Pathology affecting the small bowel mucosa and intestinal obstruction can produce profound electrolyte disturbances. Replace fluid and electrolytes with an appropriate crystalloid solution; this may be either definitive treatment for gastroenteritis or preparation for urgent surgery in patients with intestinal obstruction.

Central venous pressure monitoring is often required in the elderly or those with cardiac disease. Careful fluid balance is necessary in any patient who is seriously ill.

Antiemesis and nasogastric suction

An antiemetic is often needed for the patient with acute abdominal pain, especially if opioid analgesia has been used, although effective analgesia may also relieve vomiting. A nasogastric tube should be passed to decompress the stomach in patients with small bowel obstruction, pancreatitis and persistent vomiting despite the use of an antiemetic.

Antibiotics

Antibiotics are needed for either localised infection such as pyelonephritis or where clinical sepsis is thought to have an intra-abdominal source (e.g. perforation of the colon in an elderly patient). In patients who are septic and/or in whom perforation is suspected, broad-spectrum intravenous antibiotics should be started as soon as possible without waiting for the result of blood cultures (according to local protocols).

Urinary catheter

Measurement of urine output (via a urethral catheter) is needed in any patient who is seriously ill.

DEFINITIVE CARE

After secondary assessment and emergency treatment, some patients with abdominal pain will require referral to a surgeon (see next box). This is likely if the pain:

- has preceded other symptoms
- has persisted for more than 6 h

- is asymmetrical and distant from the umbilicus and accompanied by distension, bile stained or faeculent vomiting or significant abdominal tenderness.

Indications for surgical referral

Suspected generalised peritonitis
- **significant diffuse tenderness, with or without a rigid silent abdomen**

Suspected localised peritoneal inflammation
- **significant localised tenderness with or without other signs of peritoneal irritation**
- **tenderness and a mass**
- **tenderness and a fever**

Suspected bowel obstruction
- **pain and bile stained or faeculent vomiting**

Tenderness plus uncontrolled vomiting

Suspected pancreatitis

Suspected aortic aneurysm

Suspected bowel infarction/ischaemia

Gastrointestinal bleeding
- **upper gastrointestinal bleeding in a high risk patient**
- **lower gastrointestinal bleeding**

Age greater than 65 years

Patients with ureteric colic should be referred to the urology team (or general surgeons depending on local arrangements). Gynaecological assessment is required for women with suspected ectopic pregnancy, miscarriage, pelvic inflammatory disease or complications of an ovarian cyst. Other patients, including those with gastroenteritis, gastrointestinal haemorrhage or pyelonephritis will be admitted under the medical team. Diverticulitis and acute pancreatitis can be treated 'medically', but the ideal situation is combined management by physicians and surgeons on a high dependency unit.

Some patients may be discharged (with or without arrangements for outpatient follow-up) if they have uncomplicated cholelithiasis, ureteric colic or gastroenteritis or where a diagnosis has not been made but the patient appears clinically well and no serious condition is suspected. Advice should be given to these patients to return to hospital without delay if their symptoms deteriorate or new symptoms develop. A proportion may have presented at an early stage of an intra-abdominal problem such as appendicitis. However, if in doubt or the patient is unable to cope at home, admit for observation.

SPECIFIC CONDITIONS

Acute gastroenteritis

Gastrointestinal infection is one of the commonest abdominal disorders, and symptoms commonly include abdominal pain. Worldwide, intestinal infections account for significant morbidity and mortality. The elderly are particularly vulnerable to the effects of dehydration and electrolyte imbalance and may present with life-threatening cardiovascular collapse.

Pathophysiology

There are three different pathophysiological mechanisms that can be used to explain the clinical features and treatment rationale.

Inflammatory diarrhoea (dysentery) can follow bacterial invasion of the mucosa of the colon and distal small intestine. This leads to both impairment of absorptive function and to loss of blood, protein and mucus which contribute to diarrhoea.

Bacterial infections, which produce inflammatory diarrhoea, include *Salmonella enteritidis*, *Shigella* and *Campylobacter jejuni*. Cytopathic toxins are produced by *Clostridium difficile* which is the commonest cause of antibiotic associated colitis and by verotoxin producing *Escherichia coli*, one type of which (O157:H7) is associated with haemolytic uraemic syndrome. *Entamoeba histolytica* also produces dysentery of varying severity.

The patient may report blood and pus in the diarrhoea (which characteristically contains faecal leucocytes). Severity varies from mild self-limiting diarrhoea to severe colitis which may be complicated by toxic megacolon, perforation and sepsis.

Non-inflammatory (secretory) diarrhoea is classically due to enterotoxin of *Vibrio cholerae* in the small bowel. The toxin blocks passive absorption of sodium (and water) and stimulates active sodium (and water) excretion. This leads to an outpouring of isotonic sodium and water into the bowel lumen, which exceeds the absorptive capacity of the small intestine and colon. Active sodium absorption by a glucose dependent mechanism is, however, generally unaffected; hence rehydration may be achieved by oral glucose solutions which contain both sodium *and* carbohydrate.

Characteristically the patient has profuse watery diarrhoea (and vomiting), which may lead to severe dehydration, shock and death.

Viruses (e.g. rotavirus), *Giardia lamblia* and *Cryptosporidium,* toxins of *Staphylococcus aureus* and *Bacillus cereus* (in food poisoning) and *enterotoxogenic E. coli* (a major cause of traveller's diarrhoea) may also produce secretory diarrhoea.

Systemic infection results from infection that penetrates the mucosa of the distal small bowel, invades lymphatic structures and causes a bacteraemia. Invasive organisms include *Salmonella typhi* (typhoid or enteric fever), *Salmonella paratyphi* and *Yersinia enterocolitica*.

Although about 50% of patients with typhoid may develop diarrhoea and fever, other features are prominent (including headache, cough, malaise, myalgia, abdominal tenderness and hepatosplenomegaly, relative bradycardia and 'rose spots' on the trunk). Complications include small bowel ulceration and occasionally perforation.

Diagnosis and assessment of severity

The diagnosis is essentially clinical, supported by the result of investigations in some cases.

Clinical features

Diarrhoea, nausea and vomiting, abdominal pain, tenesmus and fever occur in various combinations. Pain may be cramp-like and transiently relieved by the passage of diarrhoea, but (with *Salmonella* or *Campylobacter infection*) may mimic a surgical acute intra-abdominal emergency.

To make the diagnosis of gastroenteritis there should be a history of diarrhoea and vomiting, although this will not always be the case. A history of affected family or other contacts supports a diagnosis of gastroenteritis; foreign travel,

ingestion of suspect food and/or immune compromised state may be risk factor(s) for infection.

Antibiotic therapy may suggest *C. difficile* colitis. The elderly and patients who are immune compromised are at increased risk from complications of infection with *Salmonella*. Patients with enteric fever may be constipated rather than have diarrhoea at the time of presentation. The diagnosis will depend on evaluation of systemic symptoms and signs in a patient who has potentially been exposed to infection (recent travel to the tropics).

On the secondary assessment examine for signs of dehydration which – particularly in the elderly – may be accompanied by circulatory failure, fever, systemic signs of bacteraemia and abdominal signs. Record the patient's weight and stool output.

Key point

Do not diagnose gastroenteritis in patients with abdominal pain and vomiting, without diarrhoea. Consider other conditions, e.g. acute pancreatitis, appendicitis

Investigations

Stool specimens (at least three on consecutive days) should be sent to microbiology (to reach the laboratory immediately) for microscopy (leucocytes, red blood cells, ova, cysts and parasites) and culture (particularly for *Salmonella, Shigella, Campylobacter* and *E. coli* O157). If amoebiasis is suspected, a 'hot stool' specimen should be sent directly to the laboratory (and the laboratory forewarned) to enable detection of trophozoites. Suspicion of *Clostridium difficile* should prompt specific examination for the associated toxin.

Check the electrolytes, urea and creatinine in any patient with signs of dehydration or requiring intravenous therapy. Request the following additional investigations in any patient who is febrile or systemically unwell:

- full blood count
- C-reactive protein
- blood cultures
- chest X-ray
- serum lactate (a useful marker of the severity of *C. difficile* infection)
- thick and thin blood films for malaria (if history indicates possible infection)

Treatment

Most patients require only supportive therapy as acute gastroenteritis is a self-limiting disease irrespective of the causative organism and most patients require only supportive therapy for self-limiting disease.

If there are signs of volume depletion, treat initially with 1–2 litres of 0.9% saline and reassess. Volume and rate of replacement may be determined clinically (by signs of peripheral perfusion, jugularvenous pulse, auscultation over lung bases and urine output) or, in the critically ill patient, by central venous pressure measurement. Add potassium, if appropriate, once the serum result is known and there is evidence of urine output.

After restoring the circulating volume, correct dehydration gradually, replacing deficit and maintenance requirements for water and electrolytes. The majority of patients with gastroenteritis can be managed with oral rehydration alone, taking

advantage of the active glucose dependent mechanism for absorption of sodium. Proprietary rehydration powders for reconstitution are available.

Antibiotics should be used for the following specific indications:

- cholera
- typhoid
- occasionally those with non-typhoid *Salmonella* or Campylobacter (associated bacteraemia and systemic symptoms, immune compromise, significant coexistent medical problem, e.g. malignancy, sickle cell disease, prosthetic device)
- *C. difficile* colitis, particularly if antecedent antibiotic therapy cannot be stopped (according to local policy). If *Clostridium difficile* is suspected (especially the elderly, patients with prior antibiotic use, patients who are immunocompromised or have a raised WBC) start treatment with metronidozole. Early liaison with a microbiologist/gastroenterologist is advised.
- specific parasitic infections (amoebiasis, giardiasis)
- Antibiotic medication neither prolongs nor increases illness complications. An antiemetic (e.g. prochlorperazine by IM injection) may be helpful.

Infection control staff should be involved in the in-patient management of gastroenteritis, especially in relation to isolation, barrier nursing and cohorting of outbreaks.

The need for surgical intervention is rare, except for complications such as perforation. Inform the local Public Health Department of notifiable diseases. Those whose occupation involves handling food require appropriate advice regarding time away from work.

Acute pancreatitis

The majority of patients with acute pancreatitis have a self-limiting illness and recover with supportive treatment on a general ward. About 20–25% will develop severe acute pancreatitis, requiring vigorous resuscitation and multidisciplinary care on the intensive treatment unit. These patients are likely to be severely hypovolaemic due to retroperitoneal fluid loss, generalised extravasation of fluid through leaky capillaries and loss of extracellular fluid from profuse vomiting. They have a mortality of 25–30%.

Cause

The common causes are gall stones and alcohol, accounting for about 80% of cases. Others include metabolic conditions (hyperlipidaemia, hypercalcaemia), drugs, trauma, infection, ischaemia, autoimmune, genetic, post-ERCP and hypothermia. In about 10% of patients no cause is found.

Clinical features

Characteristically, patients with acute pancreatitis report an acute onset of pain in the upper half of the abdomen. The initial pain may be felt in the epigastrium, right or left upper quadrant or rather vaguely in the centre of the abdomen; the pain may radiate to the back or encircle the upper abdomen. A small proportion of patients describe pain that is either overwhelming generalised pain or localised to the chest. The pain is often severe, aggravated by movement or inspiration and may be colicky. Nausea is common. During the first 12 h, most patients vomit; this may be profuse and repeated.

Most patients with acute pancreatitis are shocked; tachypnoea and tachycardia may reflect hypoxaemia, hypovolaemia and pain. Cyanosis may occur early but is less common than in patients who have suffered an intra-abdominal vascular problem or myocardial infarction. Jaundice occurs in about one quarter of

patients, particularly those who have either gall stone pancreatitis or an alcohol related illness.

The abdomen looks normal, moves with respiration and can be distended in the upper half of the abdomen, where there may be a mass. The majority of patients have tenderness over the upper half of the abdomen and occasionally this is restricted to the right upper quadrant. About half of patients have guarding, but rebound tenderness and rigidity are less common. Bowel sounds are reduced or absent in about one third of patients (and duration of ileus is an indicator of severity).

Gall stone pancreatitis presents with jaundice, pain and tenderness localised to or maximal in the right upper quadrant and a positive Murphy's sign. Seriously ill patients with acute pancreatitis may be pyrexial, tachypnoeic and hypotensive (but sometimes peripherally vasodilated) and have pleural effusions, ascites, Cullen's and/or Grey Turner's sign and/or a prolonged paralytic ileus.

Investigations

A serum amylase level greater than 3–4 times the upper limit of normal confirms the clinical diagnosis. However the serum amylase is not always raised in acute pancreatitis. Patients with alcoholic pancreatitis often have a normal amylase as may those presenting late. The amylase level returns to normal soon after the onset of an episode of acute pancreatitis, and the urinary amylase should be checked. Conversely a raised amylase is not specific. A significantly raised (greater than two times normal) serum lipase is considered more specific, but is less commonly available.

A plain abdominal X-ray is generally not diagnostic but may show an elevated diaphragm, localised gastroduodenal ileus or a sentinel loop of small bowel, or pancreatic calcification indicative of previous disease. In patients where the diagnosis is not clear, ultrasound scan or contrast enhanced CT may be helpful. Both may show a swollen pancreas or fluid in the lesser sac; CT may show non-perfused necrotic areas of pancreas and give information about severity and pseudocyst formation.

Key point

A normal amylase does not exclude acute pancreatitis

Early complications

The most significant early complication is multiple organ failure.

- Cardiovascular collapse: hypovolaemia and myocardial depression
- Respiratory failure: pleural effusions, atelectasis, pulmonary infiltrates, intrapulmonary shunting, acute respiratory distress syndrome
- Acute renal failure
- Coagulopathy
- Metabolic: hypocalcaemia, hyperglycaemia.

Severity and prognosis

Complications, including multiple organ failure, may develop rapidly and unpredictably. Identify patients at increased risk of developing severe acute pancreatitis. This will ensure that they receive high dependency or intensive care and may help avoid potentially unnecessary hazardous interventions. Evidence of three or more factors in the modified Glasgow Scoring System (next box) is associated with

increased morbidity and mortality; the greater the number of factors present, the worse the prognosis.

Adverse prognostic factors in acute pancreatitis

On admission:
Age > 55 years
White blood cell count > 15 × 10^9/l
Blood glucose > 10 mmol/l (no diabetic history)
Serum urea > 16 mmol/l (no response to IV fluids)
PaO_2 < 8 kPa

Within 48 hours
Serum calcium < 2.0 mmol/l
Serum albumin < 32 g/l
Lactate dehydrogenase > 600 IU/l

Management

Baseline investigations should include electrolytes, calcium, glucose, renal function, liver enzymes, coagulation screen, full blood count, arterial blood gases, chest X-ray and ECG.

Treatment

The priorities are to correct/prevent hypoxaemia and restore circulating volume. This limits ischaemic damage to the pancreas and reduces the risk of multiple organ failure. Those with severe disease may have a clinical picture similar to that of acute respiratory distress syndrome; if adequate oxygenation cannot be achieved with supplemental oxygen ($FiO_2 = 0.85$), the patient should be intubated and ventilated.

Rapid infusion of high volumes of crystalloid and synthetic colloid (up to 4–5 litres or more during the first 24 h) may be required. Monitoring in patients with severe disease should include a urinary catheter and central venous pressure measurement, to guide fluid resuscitation. Blood transfusion may be required for a falling haemoglobin level (due to haemorrhagic pancreatitis). Patients with persistent circulatory failure despite adequate fluid replacement may require inotropic support; those with renal impairment may need either haemofiltration or dialysis.

Pain should be treated with intravenous opioid, titrated to effect, possibly followed by patient controlled analgesia. A nasogastric tube will reduce nausea and vomiting in those with severe vomiting or an ileus. Address the cause where possible, e.g. discontinuation of drug or alcohol. Arrange ultrasound of the gall bladder and if gall stones are demonstrated in the bile duct, request an opinion on early endoscopic retrograde cholangiography and either stenting or sphincterotomy with stone extraction.

Antibiotics are given:

- for suspected cholangitis (cholestatic jaundice and fever)
- in severe acute pancreatitis as prophylaxis against infection of necrotic pancreatic tissue from bacterial translocation
- to cover endoscopic retrograde cholangiography.

Early surgery may be needed:

- to debride infected necrotic pancreatic tissue
- to exclude other treatable intra-abdominal pathology
- to remove gall stones after acute pancreatitis has subsided.

Time Out 15.2

List eight adverse prognostic factors in patients with acute pancreatitis.

Acute upper gastrointestinal bleeding

Cause and clinical presentation

Melaena, haematemesis and symptoms of hypovolaemia and/or anaemia are the common presenting features of acute upper gastrointestinal bleeding. However, there may be a history of abdominal pain due to inflammation and/or ulceration in the oesophagus, stomach or duodenum. Ingestion of non-steroidal antiinflammatory drugs (NSAIDs) is an important contributory factor in patients with peptic ulcer disease. Other causes of upper gastrointestinal bleeding include varices, Mallory–Weiss tear, oesophagitis and tumour.

Haematemesis and/or melaena suggest bleeding from the oesophagus, stomach or duodenum, although black stools may occasionally be due to bleeding into the distal small bowel or 'right' colon. Vomiting of fresh blood, compared with altered blood, suggests more serious bleeding. Rapid upper gastrointestinal bleeding can present with dark red blood per rectum, although (particularly in the absence of hypotension) this is more likely to originate in the lower gastrointestinal tract.

Primary assessment and resuscitation

The airway should be managed as described in Chapters 3 and 4. Patients with a reduced level of consciousness (e.g. those with hepatic encephalopathy) are at risk of aspiration, and may require endotracheal intubation. Restore intravascular volume, initially with warmed crystalloid (0.9% sodium chloride) and subsequently blood (see Chapter 9 for further details). Packed cells may be preferable in patients with anaemia. Vitamin K and fresh frozen plasma may be required for patients with liver disease, or for those on warfarin prothrombin complex concentrate can also be used. A central venous pressure line should be inserted in patients with evidence of shock, particularly if there is a history of cardiovascular disease, sign(s) of rebleeding or if the patient is on a β blocker. Emergency surgery (preceded by endoscopy) may be required for those with bleeding and hypovolaemia, unresponsive to fluid resuscitation and treatment of any coagulopathy. Early surgical consultation is therefore necessary.

Secondary assessment

The history should include details of the duration and severity of bleeding, recent dyspepsia, vomiting, alcohol or drugs (NSAIDs, bisphosphonates, SSRIs, anticoagulants, β blockers), jaundice, previous gastrointestinal haemorrhage and other medical problems. Look for signs of chronic liver disease and splenomegaly or malignancy. Melaena may only be apparent on rectal examination. Ensure that the important early investigations have been done including a full blood count, crossmatch, coagulation screen, biochemistry including liver enzyme profile, hepatitis serology, chest X-ray and a 12-lead ECG if appropriate.

Evidence of rebleeding includes:

- signs of hypovolaemia (fall in central venous pressure, rise in heart rate, fall in systolic blood pressure)
- fresh haematemesis or melaena
- fall in haemoglobin (3 g/dl over 48 h).

Definitive care

After resuscitation, early endoscopy (within 12–24 h) will identify the source of bleeding, provide prognostic information on the risk of rebleeding and offer an opportunity for haemostatic therapy. Emergency endoscopy should be done in patients with severe, continued or recurrent bleeding, persistent or recurrent signs of hypovolaemia, haemoglobin less than 8 g/dl or suspected varices.

Patients with an increased mortality risk (see next box) should be admitted to a high dependency area.

Adverse prognostic features in patients with gastrointestinal haemorrhage

Age ≥ 60 years
Signs of hypovolaemia/shock (systolic blood pressure < 100 mm Hg)
Haemoglobin concentration < 10 g/dl
Severe coexistent disease
Continued bleeding or rebleeding
Varices

The need for surgery for a bleeding peptic ulcer is determined by the severity, persistence or recurrence of bleeding and patient risk factors. A surgical team should be informed of all patients, especially those at increased risk (see previous box). In general, surgery should be considered for patients:

- with severe, continuing gastrointestinal bleeding
- aged > 60 years, or younger with other risk factor(s), who have either persistent bleeding requiring four units of blood or one rebleed
- aged < 60 years old with no risk factor(s), who have either persistent bleeding requiring six to eight units of blood or two rebleeds.

Key point

Patients with an increased risk of death from a gastrointestinal bleed (e.g. the elderly with persistent or recurrent bleeding) may benefit most from prompt surgery

Vascular causes of acute abdominal pain

Vascular causes of abdominal pain are important because they include conditions which are life-threatening but treatable if recognised early. Early recognition may be difficult because:

- these conditions are relatively uncommon
- initial symptoms – though severe – may be non-specific
- 'surgical' signs of an acute abdomen may be lacking
- affected patients are often elderly and have coexistent medical problems

Delay in diagnosis and referral for surgery when appropriate may result in increased mortality and morbidity. The possibility of a vascular cause for abdominal pain should always be considered in patients over the age of 50 and particularly above the age of 70. The three most common vascular causes of abdominal pain are abdominal aortic aneurysm, acute mesenteric ischaemia and myocardial infarction presenting with abdominal pain.

Abdominal aortic aneurysm

A leaking abdominal aortic aneurysm is the commonest intra-abdominal vascular emergency and may present as:

- vague abdominal pain
- a preceding history of back pain for hours or days, with or without a previously diagnosed aneurysm
- shock with a distended tender abdomen (if the patient has not exsanguinated before reaching hospital)
- atypical abdominal pain
- severe pain of sudden onset in the abdomen radiating to the flank and back, with a pulsatile mass, an abdominal bruit and reduced pulses in one or both lower limbs (due to emboli or shock), accompanied by signs of hypovolaemia.

Key point

In the patient previously known to have an abdominal aortic aneurysm, beware of attributing pain to another cause, however well the patient may appear

However, a majority of these patients will not be known to have an aneurysm, pain may not be severe, a mass may be difficult to detect and signs of hypovolaemia may be minimal. Pain in the abdomen, flank or back in these patients may be misdiagnosed as ureteric colic or acute pancreatitis. Others present with collapse, with neurological symptoms (spinal cord affected) or pain in the lower limbs (distal emboli). Risk factors include age over 65 years, male, hypertension, smoking, known vascular disease, as well as conditions such as Marfan's syndrome. If the diagnosis is not to be missed, the possibility of an aortic aneurysm must be actively considered in any middle aged or elderly patient with a history of abdominal pain, back pain or collapse, even though there is no evidence of haemodynamic compromise.

If abdominal aortic aneurysm is suspected, the principles of management are:

- resuscitation aiming for a systolic blood pressure of about 90 mm Hg (if the patient is conscious)
- carefully titrated IV opioid analgesia
- immediate surgical referral
- crossmatch blood and warn blood transfusion staff
- portable ultrasound (the aneurysm may also be outlined by calcification on an abdominal X-ray)
- rapid transfer to the operating theatre once the diagnosis has been made (because of the possibility of sudden decompensation).

Key point

Always consider the possibility of an aortoenteric fistula in a patient who presents with upper gastrointestinal haemorrhage and has an abdominal aortic aneurysm especially if it has been repaired

Acute mesenteric infarction

Acute intestinal ischaemia commonly affects the superior mesenteric artery. If diagnosis and treatment are delayed, complications include necrosis of the small

bowel, ascending colon and proximal transverse colon. Diagnosis depends on a high index of suspicion, particularly in patients at increased risk (see next box), and appropriate history and examination.

Risk factors for acute mesenteric ischaemia

Elderly (older than 50 years, greater risk with increasing age)
Known atheromatous vascular disease
Source of embolus (atrial fibrillation and other arrhythmias, myocardial infarction, ventricular aneurysm, valvular heart disease, infective endocarditis)
Prolonged hypoperfusion
Procoagulant disorders

Characteristically the pain is acute, severe out of proportion to physical signs and poorly localised in the periumbilical region or below. There may be a short preceding history of abdominal pain after eating. An alternative presentation is of pain with an insidious onset over 24–48 h, initially poorly localised and becoming generalised throughout the abdomen. The pain is colicky initially, becoming steady and unrelenting. Vomiting is common, sometimes with haematemesis.

The patient is pale, distressed and usually has diarrhoea with blood. As bowel infarction develops the abdomen becomes distended with worsening tenderness, guarding and rebound, and absent bowel sounds. Fever and shock due to bacteraemia often occur.

The key to management is clinical suspicion at an early stage when abdominal signs are minimal. An abdominal X-ray may show dilatation of the intestine with multiple fluid levels; the appearance of gas in the portal vein indicates intestinal necrosis.

Treatment includes vigorous fluid resuscitation, opioid analgesia, antibiotics and urgent surgical referral with a view to laparotomy once the patient has been resuscitated.

Myocardial infarction

An acute inferior myocardial infarction may present with upper abdominal pain. If nausea and vomiting are prominent features, a primary intra-abdominal problem may be suspected. In acute cardiac failure, distension of the liver capsule causes right upper quadrant pain mimicking a biliary or upper gastrointestinal tract problem.

Complete heart block complicating inferior myocardial infarction and causing collapse can be mistaken for intra-abdominal bleeding. All patients with abdominal pain over 40 (and younger if there is any reason to suspect the diagnosis) should have an ECG.

Inflammatory bowel disease

Ulcerative colitis

Ulcerative colitis is an inflammatory disease of uncertain cause affecting the rectum and colon. Many patients experience a gradually progressive illness in which symptoms related to bowel habit are prominent. However, some present with an acute illness characterised by fever, abdominal pain, diarrhoea with blood and mucus and tenesmus. A proportion of these patients develop fulminant colitis (associated with pancolitis).

Key point

Abdominal signs and leucocytosis may be masked if the patient is on steroids. However, remember that steroids can cause a leucocytosis

Features of severe colitis

Severe diarrhoea (more than six stools a day) with blood
Systemic features: tachycardia, signs of hypovolaemia, fever and drowsiness, weight loss
Progressive abdominal pain, distension and tenderness over the colon
Raised ESR, CRP and white cell count, low haemoglobin and albumin, electrolyte disturbance

Toxic megacolon is a medical emergency and the possibility of this complication should be considered in all patients with severe colitis. In addition to the features of severe colitis (see box above), abdominal X-ray shows dilatation of the colon with a diameter greater than 6 cm and loss of haustrations. Bowel perforation occurs in patients with fulminant colitis, with or without toxic megacolon. Symptoms and signs of perforation may be obvious, but if the patient is on steroids these may be masked, and a deterioration in the patient's condition may be the only clue to this complication. Free air may be seen on X-ray.

Key point

The patient may be 'toxic' without any evidence of colonic dilatation

The patient with severe colitis/toxic megacolon should be managed by both medical and surgical gastroenterologists. The initial treatment includes resuscitation with fluid and electrolyte replacement, intravenous steroids and antibiotics. Parenteral nutrition is frequently required, as is blood transfusion. If surgery is delayed until after the colon has perforated, mortality is significantly increased. In the acutely ill patient colectomy is needed for:

- perforation
- features of severe colitis (with or without toxic megacolon) which deteriorate or do not improve after 24–48 h on medical treatment
- massive continuing haemorrhage.

Crohn's disease

Crohn's disease is a chronic granulomatous inflammatory disease of undetermined cause. Any part of the gastrointestinal tract may be involved often with 'skip lesions', but the ileum is affected in most patients.

The clinical presentation of Crohn's disease is variable. Abdominal pain, diarrhoea, anorexia, weight loss and fever are common features. Although a chronic illness with recurrent symptoms over years is common, patients with terminal ileitis can present acutely and be misdiagnosed as acute appendicitis. Think of Crohn's disease (as opposed to appendicitis) if the pain is poorly localised to the right lower quadrant and of more than 48-h duration, or there is a history of

previous surgery (remember the possibility of a retrocaecal inflamed appendix). Other findings include a palpable mass, perianal signs (more frequently than in ulcerative colitis), sepsis and extraintestinal features. Crohn's colitis can also present with a clinical picture similar to that of ulcerative colitis.

Initial investigations include stool samples for microbiology to exclude infectious diarrhoea, abdominal X-ray, haematology and biochemistry, followed by specialist investigation. Treatment includes fluid and electrolyte replacement as required and nutritional supplements, medical treatment and surgery for complications.

SUMMARY

Abdominal pain may be due to any one of a wide variety of conditions, both intra- and extra-abdominal.

Primary assessment and resuscitation

A minority of patients will have life-threatening conditions. Rapid diagnosis and immediate treatment are required. Consider the following:

- Is the patient's airway at risk (recurrent vomiting with a depressed level of consciousness)?
- Is oxygenation and ventilation adequate (often impaired with chest pathology, severe acute pancreatitis and sepsis)?
- Are there signs of circulatory failure (when abdominal pain is due to a condition causing life-threatening hypovolaemia or sepsis)?

Urgent surgical (or gynaecological) referral is required, as part of resuscitation, for haemorrhagic shock. Septic shock due to an intra-abdominal cause is a multidisciplinary emergency; treatment includes vigorous fluid resuscitation, IV broad-spectrum antibiotics and early specialist consultation.

Secondary assessment and emergency treatment

- A detailed history and careful examination are the most important elements in making a diagnosis.
- Selective imaging and investigation may confirm the diagnosis and/or provide useful supplementary information.
- Careful attention to fluid and electrolyte replacement, analgesia, antibiotics and nasogastric drainage are important, particularly when the patient needs surgery.

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CHAPTER 16

The patient with hot red legs or cold white legs

OBJECTIVES

After reading this chapter you will be able to describe:

- the assessment and initial management of common medical problems which arise in the lower limb
- the complications that may arise from limb pathology.

INTRODUCTION

Trauma is the most common condition to affect the lower limb. In addition:

- systemic diseases and dermatological problems often lead to symptoms in the legs
- degenerative diseases may cause pain in the hip and the knee
- oedema usually gravitates to the legs
- chronic venous disease is common in older people.

However, a variety of acute medical problems may also arise in the lower limb. This chapter describes the assessment and initial management of the most common of these conditions (see next box).

Common acute medical problems in the lower limb

Venous thrombosis	**Daily**
Phlebitis	**Daily**
Venous problems in IV drug users	**Daily**
Cellulitis	**Daily**
Arterial embolism	**Weekly**
Intraarterial injection	**Monthly**
Compartment syndrome	**Monthly**
Rupture of a Baker's cyst	**Monthly**

GENERAL PRINCIPLES OF ASSESSMENT OF THE LOWER LIMB

Key point

Always look at the 'whole patient' before examining their legs

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For a full initial assessment, see Chapter 3.

The history of a condition that affects the legs takes the same format as any other history. Pain, swelling and loss of function (i.e. inability to weight bear) are usually the most important features. Always consider:

- recent injury/surgery
- smoking
- recent infections
- diabetes
- venous or other vascular disease
- pregnancy or use of oral contraception
- known malignancy (may cause coagulopathy)
- drug use
- long distance travel (venous stasis)
- family history.

The examination of the lower limb should follow the sequence of 4 four-letter words:

LOOK
FEEL
MOVE
X-RAY

Look for: site, spread, symmetry, systemic effects, swelling, bruising and redness
Feel for: temperature, tenderness, oedema and pulses
Move for: passive and active range of movements, function (including weight bearing)
X-ray for: suspected traumatic conditions.

Investigations should usually include blood glucose, full blood count and plasma chemistry. Special imaging may be appropriate (see later).

VASCULAR CONDITIONS OF THE LOWER LIMB

Venous thrombosis

Facts and figures about deep vein thrombosis (DVT):

- Around 2–5% of the population have a venous thrombosis at some time during their lives (see later box on incidence).
- DVT occurs in up to 40% of postoperative patients and increases in incidence with age. Three per cent of such patients progress to pulmonary embolism.
- DVT is estimated to occur in nearly one in every thousand pregnancies and 15–20% of these women will have pulmonary embolis if left untreated. Pulmonary embolism is the commonest cause of maternal death in the UK and similar developed countries.

Factors predisposing to thrombosis in pregnancy

- Increased concentrations of clotting factors and fibrinogen
- Production of inhibitors of fibrinolysis by the placenta
- Venous stasis
- Changes in blood vessels and patterns of blood flow
- Relative immobility

Time Out 16.1

Make a list of some types of patients who are at a high risk of DVT.

Thrombosis of the deep veins of the lower limb or pelvis may be caused by changes in:

- blood coagulation (smoking, oral contraceptive, procoagulant conditions)
- blood vessels (pregnancy)
- blood flow (immobility, plaster casts).

There is:

- swelling and oedema distal to the occlusion
- warmth, redness and deep tenderness of the thigh or calf
- dilated superficial veins.

Key points on clinical findings in suspected DVT

The classic signs depend on venous occlusion. In contrast, there may be no signs in the presence of an extensive, non-occlusive but potentially lethal thrombus. Moreover, there is some evidence that non-occlusive thrombi float in the middle of the vein and break free very easily

Homan's sign cannot be relied on – it is non-specific and may cause a pulmonary embolus and should not be done

Calf muscle tear, ruptured Baker's cyst and superficial phlebitis may all be mistaken for DVT; oedema may occur with ischaemia

DVT may be the first sign of occult malignancy – hence the need for abdominal and pelvic examination and investigation

Diagnosis

The first step in diagnosis of DVT involves assigning a clinical probabilty of DVT to the patient. On the basis of Well's criteria (see Table 16.1 below) patients can be classified as being 'likely' or 'unlikely' to have DVT. Patients deemed 'likely' to have DVT require diagnostic imaging.

Table 16.1 Incidence of venous thromboembolic disease in women

Characteristics	Cases per 100,000 women per year
Healthy, non-pregnant, not taking oral contraceptive	5
Using a second-generation pill (i.e. containing levonorgestrel)	15
Using a third-generation pill (i.e. containing desogestrel or gestodene)	25
	100,000 pregnancies
Pregnant	60

All of the above numbers increase with age and other known risk factors such as obesity

Clinical prediction rule to rank DVT risk	
Ask about:	Score
Active cancer (ongoing treatment, diagnosed in last 6 months or having palliative care)	+1
Paralysis, paresis or plaster immobilisation of a leg	+1
Recently bedridden >3 days or major surgery within past 4 weeks	+1
Look for:	
Localised tenderness over distribution of the deep veins	+1
Entire leg swollen	+1
Calf circumference 10 cm below tibial tuberosity >3 cm greater than other calf	+1
Pitting oedema only in the symptomatic leg	+1
Collateral dilated (but not varicose) veins	+1
An alternative diagnosis as/or more likely than DVT	−2
Match the patient's score to the risk	
Score	Risk of DVT
2 or more	Likely
1 or less	Unlikely

If a DVT is suspected, the patient should have a D-dimer assay. This test measures the breakdown products of cross-linked fibrin and is thus more specific for thrombosis than measurement of fibrin degradation products which arise from the breakdown of fibrinogen and fibrin monomer. A positive D-dimer mandates further diagnostic imaging, as above. A negative D-dimer in an 'unlikely' patient excludes the diagnosis.

The subsequent tests depend on local availability. Plethysmography, and its various modifications, can be used – often for screening. Doppler ultrasound studies are replacing the traditional venogram in many centres. If neither investigation is immediately available, the patient must be treated on clinical suspicion alone.

Management

Once the diagnosis is confirmed, treatment depends on local protocol. Anticoagulation is required with either an intravenous infusion of standard unfractionated heparin or, more commonly nowadays, with low molecular weight heparins given subcutaneously (e.g. enoxaparin 1.5 mg/kg every 24 h). The availability of subcutaneous treatment has increasingly led to patients with DVT being treated at home by community nurses. Heparin is discontinued when adequate oral anticoagulation is established. This is continued for a minimum of 3 months.

Thrombosis of veins distal to the popliteal vein (below-knee DVT) is common because of the venous sinuses present in the soleus muscle of the calf. Clinicians treat below-knee DVT with anticoagulation, but the condition is increasingly managed conservatively with:

- elastic stockings
- non-steroidal anti-inflammatory drugs
- rest and elevation
- clinic review.

Key point

At least 5% of below-knee deep vein thromboses spread to the proximal veins and 50% of all above-knee venous thromboses embolise to the lungs

For diagnosis and management of suspected pulmonary embolism, see Chapter 8.

Phlebitis

Inflammation of the long or short saphenous vein usually occurs in patients with varicose veins. Phlebitis (usually in the arm) can also follow intravenous therapy. The vein is red, hot and tender.

Management

Phlebitis usually settles with topical therapy and oral non-steroidal anti-inflammatory drugs. If the patient is pyrexial, an antibiotic can be added. Superficial thrombophlebitis affecting varicose veins is treated with non-steroidal anti-inflammatory drugs.

Venous disease in intravenous drug abusers

Repeated injection into any vein causes chronic venous obstruction. There is swelling and oedema of the whole lower limb and dilatation of the superficial vessels; sinuses are often found in the groin. Acute thrombosis may occur, in which case the limb becomes hot, red and painful.

Management

This DVT is potentially life-threatening and should be treated with heparin as described earlier.

Arterial embolism

Thrombi which embolise to peripheral arteries may arise from several sites:

- the left atrial appendage (usually in the presence of atrial fibrillation)
- the left ventricle (invariably on an area damaged by a recent myocardial infarction or dilated ventricle or an aneurysm)
- an atheromatous plaque
- an aortic aneurysm
- a thrombosis in a deep vein in a patient with a patent foramen ovale (paradoxical embolism).

All of these possibilities should be considered although the first two are by far the most common. Emboli tend to lodge at the sites of bifurcation of arteries. Their effects depend on the extent of the occlusion of the circulation and on the degree of collateral circulation that exists. Common sites that involve the lower limb include:

- the aortic bifurcation (bilateral ischaemia to the level of the knees)
- the origin of the deep femoral artery (ischaemia to the mid-calf)
- the bifurcation of the popliteal artery (ischaemia of the foot).

Sudden occlusion of the femoral artery causes the six 'P's:

Findings in arterial occlusion of the lower limb

- **Pain**
- **Pallor**
- **Pulselessness**
- **Paraesthesiae**
- **Paralysis**
- **Perishing cold**

Management

Embolism must be treated within 6 hours of the onset of symptoms or else propagation of thrombus distal to the embolus will greatly worsen prognosis.

Treatment includes:

- oral aspirin (300 mg)
- intravenous analgesia
- intravenous fluids
- referral to a vascular surgeon for embolectomy and/or fibrinolytic therapy.

Intraarterial injection

Irritant substances may cause critical ischaemia if injected into an artery. Intravenous drug users are the commonest sufferers from this problem; temazepam is the drug most usually involved. The ischaemia results from a mixture of vasospasm and multiple small emboli. Severe pain is the prominent symptom but the other signs described above (six 'P's) may be absent.

Management

This is similar to that described earlier for arterial embolism. Drugs that cause arterial dilatation may be considered.

Acute compartment syndrome

Closed compartment syndrome is caused by swollen, contused muscle or bleeding inside a rigid fascial envelope. The onset may be delayed after injury and insidious. Early symptoms are pain – particularly on muscle stretching – and paraesthesia. The affected part may also (but not invariably) be pale and cool with a slow capillary refill. Ischaemia results from compression of small blood vessels and so the presence of distal pulses is of no help in excluding the diagnosis.

Compartment syndrome can easily develop unseen under a plaster cast or below an eschar from a burn. The most common site to be affected is the lower leg, which has four anatomical compartments, but the syndrome is also seen in the forearm (three fascial compartments), buttock, thigh, foot and the hand.

Key point

In compartment syndrome, the limb may not be broken, distal pulses may be present and pulse oximetry may be normal

Management

Suspicion of compartment syndrome is an indication for immediate orthopaedic referral. Manometry is useful, particularly in patients with a depressed level of consciousness. There are four compartments in the lower leg and all may require extensive fasciotomy.

> **Key point**
>
> **Arteriography will reveal arterial lesions but will not demonstrate compartment syndrome**

OTHER MEDICAL CONDITIONS OF THE LOWER LIMB

Rupture of a Baker's cyst

Bursae in the popliteal fossa occur either spontaneously or may be connected to the knee joint. An enlarged and isolated popliteal bursa (a Baker's cyst) can be associated with rheumatoid arthritis. If this cyst bursts, it causes pain in the upper calf as the synovial fluid is squeezed between the calf muscles. The condition is often misdiagnosed as either a muscle injury or a DVT.

> **Key point**
>
> **A popliteal aneurysm may also present as an extra-articular swelling behind the knee**

Management

Ultrasound is the initial investigation. Arthrography is diagnostic. The patient should be referred to an orthopaedic surgeon or a rheumatologist.

Cellulitis of the lower limb

Infection of the skin may arise around a wound or without any obvious port of entry. The skin is red, swollen and tender, although the degree of pain is very variable. A cause for the infection should be sought but is not usually found. Without treatment, cellulitis can progress rapidly, leading to lymphangitis, lymphadenopathy and sepsis.

Management

Analgesics and antibiotics should be prescribed. The infection may be streptococcal and/or staphylococcal and so a combination of penicillin V and flucloxacillin is usually appropriate.

Admission for observation, elevation and intravenous therapy is indicated for extensive or rapidly progressing lesions. Lesser cases can be treated with oral antibiotics at home but should be reviewed within 36 hours. The limits of the infection should be marked on the skin with a pen so that changes are obvious at review.

Athlete's foot is a fungal infection which gives rise to an itchy whitish area between the toes (usually in the web space between the third and the fourth toes). It may be the cause of an ascending cellulitis and so the toes should always

be examined in patients with cellulitis of the leg. If found, athlete's foot is treated with a topical cream, such as clotrimazole.

Time Out 16.2

Make a list of signs that differentiate arterial embolism from compartment syndrome.

SUMMARY

A number of serious medical and surgical problems are commonly seen in the lower limb.

- The assessment and management of these conditions follows a logical sequence.
- Trauma is the commonest problem to affect the leg but medical (especially vascular) conditions must always be considered.
- If untreated, some of these conditions can present a threat to life or limb.

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CHAPTER 17

The patient with hot and/or swollen joints

OBJECTIVES

After reading this chapter you should be able to:

- discuss the general principles of recognising, assessing and managing patients presenting with hot and/or swollen joints.

INTRODUCTION

A systematic approach will help in the diagnosis and management of these patients. This entails:

- recognition of the condition
- assessment of the patient
- determining the cause
- organising appropriate investigation relevant to the suspected diagnosis
- providing appropriate treatment.

Key point

The aim is to immediately identify and treat those patients with septic arthritis

GENERAL PRINCIPLES OF MANAGING PATIENTS WITH HOT AND/OR SWOLLEN JOINTS

Recognition (the history)

This is usually the easy part. The patient presents with any, or all, of the following symptoms and signs related to their joints:

- red joints
- swollen joints
- hot joints
- tender joints
- painful joints
- decreased function.

Together, these signs and symptoms represent manifestations of inflammation. Swelling indicates organic disease and may be due to intra-articular fluid, synovitis, bone hypertrophy or a swollen periarticular structure.

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The nature of the pain provides clues to the cause of the condition. Pain at the end of the day implies a mechanical cause and is therefore often found in patients with osteoarthritis. In contrast, pain due to inflammatory disease tends to be worse in the morning and after rest, and may improve with exercise.

Loss of function is usually due to the joint being painful and stiff. The latter is most marked in the morning in inflammatory disease. In addition, with chronic joint problems, tendon and articular damage can exacerbate both neurological impairment and muscle weakness.

Assessment

There are many causes for a 'hot and/or swollen joint'. As treatment varies with the cause, the clinician needs to try to identify the likely causes as soon as possible. Differentiation can be helped greatly in this task by assessing the six 'S's (see box below).

The six 'S's of hot joint assessment

Single or several joints involved
Site(s)/symmetry
Sequence of symptoms
Systemic effects
Supplementary features
Specific findings on joint examination

Systemic effects include pyrexia and gastrointestinal upset. Supplementary features include the signs of other organ involvement.

The social impact of the condition needs to be considered when developing a management plan. This includes assessment of the patient's current occupational, social and domestic situation and how this has been affected following the onset of arthritis.

Causes

One of the most important clues is the number of joints involved. There are four causes which can potentially manifest as either mono- or polyarthropathies.

Potential causes of both mono- and polyarthropathy

O – Osteoarthritis	**Daily**
R – Rheumatoid arthritis	**Weekly**
C – Connective tissue disease	**Annually**
S – Spondyloarthritis	**Annually**

Once these have been excluded, you can divide the remaining causes into those which affect either a **single** joint or **several** joints.

Causes of monoarthropathy (Common)

S – Sepsis	**Weekly**
I – Injury	**Weekly**
N – Neoplasm	**Annually**
G – Gout/pseudogout	**Daily**
L – Loose body	**Weekly**
E – Erythrocytes	**Monthly**

Causes of polyarthropathy (Rare)

S – Sexually transmitted disease	**Annually**
E – Endocrine/metabolic*	**Annually**
V – Viral	**Monthly**
E – Endocarditis	**Only in examinations**
R – Rheumatic fever	**Only in examinations**
A – Allergies/drug associated	**Only in examinations**
L – Lyme disease	**Only in examinations**

*E.g. Wilson's disease, alkaptonuria.

Investigations

Appropriate investigations should be requested to confirm or refute your suspected diagnosis. These consist of:

- blood tests
- radiography
- joint aspiration.

Blood tests

Although many blood tests can be requested, it is best initially to concentrate on the baseline measurements given in Table 17.1.

Non specific markers of active inflammation are:

- normocytic normochromic anaemia
- raised platelet count
- elevated erythrocyte sedimentation rate and C-reactive protein
- low albumin.

Table 17.1 Blood tests

Test	Condition
Uric acid	Gout
Rheumatoid factor	Present in 50–75% of rheumatoid arthritis and 30% of collagen/vascular disease
Antinuclear antibodies	Common in connective tissue disorders
Full blood count	Anaemia common in chronic conditions
Erythrocyte sedimentation rate	Reflects severity of inflammation
C-reactive protein	Reflects severity of inflammation

Radiography

During the development of the arthritis, the radiological features may vary from nothing to complete joint disruption (see next box). In addition, particular conditions have specific radiological features.

Radiological features of a hot joint (each finding can be present in isolation or in combination with others)

Normal
Soft tissue swelling
Joint space widening
Underlying bone lucency
Joint space narrowing
Bone destruction
Crystal deposition
Subluxation/dislocation
Loose body

Joint aspiration

See practical procedure in Chapter 33. The aspirate should be tested for the following:

- Gram stain
- Culture – bacterial and viral
- Crystals – with polarising microscope
- Red blood cells
- White blood cells
- Glucose
- Protein

Treatment

In the acute phase, all inflamed joints benefit from analgesia and splintage. However, appropriate treatment for the specific cause must also be started.

SPECIFIC TYPES OF HOT JOINTS

Septic arthritis

Causes

Staphylococcus aureus is the causative organism in 80% of cases, often spreading via the bloodstream, rarely from adjacent local osteomyelitis. It is more common in patients with one or more of the following risk factors:

- chronic disease
- rheumatoid arthritis
- intravenous drug use
- immunocompromise
- prosthetic joint.

The six 'S's

- **Single/multiple joint involved?** – Single
- **Site(s)?** – Any, but commonly around the knee in teenagers

- **Sequence of symptoms?** – Develops gradually over a matter of hours to days. In addition to the signs of a hot joint, there is usually little or no movement and it is very tender
- **Systemic effects?** – Pyrexia and other signs of bacteraemia. Look carefully for potential portals of entry of infection.
- **Supplementary features?** – Related to the risk factors.
- **Specific findings on joint examination?** – There is usually a hot, tense joint effusion and marked pain on any compression/movement.

Investigation

Blood culture may show the causative organism. The joint aspirate may show pus, but a gram stain is necessary to help decide on an immediate antibiotic. This may be modified after the culture results are known.

Septic arthritis and pseudogout both give rise to pus in the aspirate and positive birefringent crystals in plane polarised light.

Treatment

Key point

Sepsis can destroy a joint in under 24 h!

Intravenous antibiotics must be started immediately the diagnosis is suspected. The choice will depend on local hospital policy but, using the gram stain, a good initial 'blind' combination is given in the next box.

Gram positive cocci	1 g flucloxacillin – 6 hourly – IV
Gram negative cocci	1.2 g benzylpenicillin – 6 hourly – IV
Gram negative rods	Gentamicin – dose according to body weight and renal function

Orthopaedic referral should be made without delay as urgent surgical drainage is probably required.

Gout

Cause

Normally nucleic acids in cells are metabolised into purines, which in turn are converted into uric acid. This is eliminated predominantly by the kidneys (67%) and the remainder by the gut. In gout, sodium monourate accumulates and crystals precipitate into joints, inducing an inflammatory reaction.

It follows from this metabolic pathway that uric acid can accumulate following reduced renal excretion of uric acid or increased production (see box below).

Causes of gout

Causes of reduced renal excretion of uric acid:

Intrinsic kidney disease

Diuretics (thiazide & loop)

Continued

Causes of gout (Continued)

Diabetic ketoacidosis
Lactic acidosis
Starvation
Causes of increased uric acid production:
Foods high in purines: **– all meats**
– meat extracts and gravy
– seafood
– yeast
– beer
– beans and lentils
Increased cell destruction:
Leukaemia
Polycythaemia
Some cancer therapies

The six 'S's

- **Single/multiple joint involved?** – Single initially
- **Site(s)?** – Classically the metatarsophalangeal joint of the hallux (other joints can be involved)
- **Sequence of symptoms?** – Comes on abruptly, typically at night
- **Systemic effects?** – Usually none
- **Supplementary features?** – Chronic tophaceous gout: tophi (urate deposits) develop in avascular areas after repeated attacks. Sites include pinna, tendons and the elbows. Urate nephropathy. Uric acid stones
- **Specific findings on joint examination?** – Gout is usually very painful on palpation of the synovial or perisynovial tissues, but not very painful on axial compression of the joint or minor passive movement.

Investigations

Aspirate – Negative birefringent needle shaped crystals

Key point

The serum urate may be normal even in acute gout

Treatment

Remove the precipitating cause. Patients should cut down on these foods, but dietary purines only contribute 1 mg/dl to the serum urate level so dieting only helps a small amount.

Analgesia: A non-steroidal anti-inflammatory drug will usually provide good symptomatic relief. Be wary of gastrointestinal complications, particularly in the elderly. It is therefore advisable to co-prescribe a proton pump inhibitor for gastromucosal protection. In patients unable to use non-steroidal anti-inflammatory drugs, colchicine can be helpful.

After three weeks, consider using allopurinol. This reduces the plasma urate level by blocking the production of uric acid, via inhibition of the enzyme xanthine oxidase (see next box).

Indications for allopurinol therapy

Recurrent attacks of gout
Chronic tophaceous gout
Polyarticular gout
Clinical or X-ray signs of gouty arthritis
Recurrent uric acid renal stones
Prophylaxis before cytotoxic chemotherapy for haematological malignancy

Key point

Aspirin in high doses (300 mg tds) can be used to treat gout, although seldom used today. In contrast, low doses (<150 mg daily) can cause gout

Pseudogout

Causes

Pseudogout results from the precipitation of calcium pyrophosphate crystals in joints. This is more likely when any of the following risk factors are present: old age, dehydration, illness, hypothyroidism, diabetes mellitus, any arthritis, high serum calcium and low serum magnesium or phosphate.

Tip

Pseudogout is the commonest cause of an acute monoarthropathy in the elderly

The six 'S's

- **Single/multiple joint involved?** – Single
- **Site(s)/symmetry?** – Mainly the knee
- **Sequence of symptoms?** – Less severe and longer lasting than gout. In chronic form it leads to destructive changes like osteoarthritis
- **Systemic effects?** – Usually none, unless associated with a metabolic condition, e.g. haemochromatosis
- **Supplementary features?** – Usually none
- **Specific findings on joint examination?** – Pseudogout is usually very painful on palpation of the synovial or perisynovial tissues, but not very painful on axial compression of the joint or minor passive movement.

Investigations

Aspirate the joint and check the fluid for weakly positive birefringent rod shaped crystals in plane polarised light. Calcium pyrophosphate deposits in cartilage, ligaments and joint capsules may also be seen on X-ray. This can occur in any cartilaginous joint but the knee is classically involved. There may also be joint destruction.

Treatment

Correct any cause and provide analgesia. As this analgesia is usually a non-steroidal anti-inflammatory drug, the same precautions need to be applied as described for gout.

Rheumatoid arthritis (disease)

Cause

For reasons that are unclear, patients with this condition develop a systemic, autoimmune inflammatory disease which acts mainly on the synovial linings. Often this leads to irreversible destruction of joints and, in some cases, systemic complications. This type of arthritis is common, affecting approximately 1% of the adult population, particularly females (ratio 3:1) and peaks in the fourth decade.

As part of the inflammatory process, B lymphocytes secrete autoantibodies, known as rheumatoid factors, which act on the synovium as well as extra-articular sites. The latter explains the systemic symptoms which are present in these patients.

The six 'S's

- **Single/multiple joint involved?** – Usually more than three joints are involved but rarely it affects a single joint. When it does, it is typically a large joint, such as the knee, ankle, shoulder or wrist.
- **Site(s)?** – Hands and feet initially. Symmetrical.
- **Sequence of symptoms?** – Initially, it tends to affect the hands and feet. In the elderly, it can present as an acute hot joint. With time, larger joints become involved while previously affected joints remain painful, stiff and fusiform in shape. In keeping with its inflammatory aetiology, the stiffness is most noticeable in the morning and typically lasts over 30–60 min. When the patient moves about, the nocturnal accumulation of soft tissue fluid disperses and the stiffness eases. In contrast, the presence of erythema extending beyond the joint margin can indicate septic arthritis or cellulitis.

  In addition, the onset may be acute self-limiting attacks (palindromic), rapid with severe polyarticular involvement (explosive), non-articular features (systemic) or limited to one joint (mono).

As the disease process progresses, ulnar deviation and volar subluxation of the metacarpophalangeal joints occur, along with swan neck and *boutonnière* deformities of the fingers, and Z-deformities of the thumbs. Some changes in the feet, especially subluxation of the metatarsophalangeal joints, are common. In addition, there can be extensor tendon rupture and intrinsic muscle wasting of the hands and feet. It is important to remember that the atlantoaxial joint can also be involved. This, along with other changes in the cervical spine, can lead to subluxation and potential spinal cord compression during airway maneouvres. Rheumatoid arthritis can also complicate airway management through reduced range of movement in the neck and temporomandibular joints.

- **Systemic effects?** – Anaemia, weight loss
- **Supplementary features?** – In addition to rheumatoid nodules (hence seropositive), these include:
  - respiratory system – pulmonary fibrosis, pleuritis, bronchiolitis obliterans, cricoarytenoid involvement
  - cardiovascular system – valvulitis, vasculitis, pericarditis
  - liver – hepatitis
  - central nervous system – carpal tunnel, multifocal neuropathies
  - eyes – episcleritis, scleritis, dry eyes, scleromalacia
  - kidney – amyloid
  - reticuloendothelial – lymphadenopathy, splenomegaly
  - skin – rash, nodules.

- **Specific findings on joint examination**? – There is joint swelling and tenderness along the joint line. Pain on compression of metacarpophalangeal or metatarsophalangeal joints is an early feature of rheumatoid arthritis. Ulnar deviation and volar subluxation of the metacarpophalangeal joints, swan neck and *boutonnière* deformities of the fingers, and Z-deformities of the thumbs as outlined above, are found at various stages of the disease. There are some changes in the feet, especially subluxation of the metatarsophalangeal joints.

Key point

Rheumatoid disease affects multiple systems. Early referral to a rheumatologist is essential

Investigation

Laboratory investigations help only to support the diagnosis of rheumatoid arthritis or to exclude alternative diagnoses. They do not help to confirm or exclude rheumatoid arthritis. Investigation results can all be negative at the onset of the disease.

Rheumatoid factor can be detected in about 50–70% of patients with rheumatoid arthritis, but can be absent in up to 60% of patients with early disease. Therefore, negative rheumatoid factor should not be taken as evidence against rheumatoid arthritis. A positive result should also be interpreted with caution, as it occurs in several conditions (e.g. TB, hepatitis C, SLE) as well as occurring in 5% of healthy individuals.

Key point

Rheumatoid factor should never be used on its own to diagnose rheumatoid arthritis. Results can all be negative at the onset of the disease

A chest X-ray may show pulmonary fibrosis, nodules and rarely pleural effusions

Plain radiographs of hands and feet are normal or show only soft tissue changes in up to 70% of patients with early rheumatoid arthritis.

Treatment

Cases of suspected rheumatoid arthritis should be referred to a rheumatologist, to confirm the diagnosis and take over the long-term care of these patients. In the meantime, analgesia must be provided. Start treatment with paracetamol and add a non-steroidal anti-inflammatory drug.

Disease-modifying drugs (e.g. methotrexate, sulphasalazine and monoclonal antibodies such as infliximab) are used if there is no response to the non-steroidal antiflammatory or synovitis has persisted for more than six months. The difficulty for the clinician is that many patients with early undifferentiated inflammatory arthritis have self-limiting disease such as postviral arthritis. It is important not to expose such patients to potentially toxic therapy with these drugs.

Other potential therapies include:

Non-specific

- Regular exercise
- Household aids

Specific
- Intra-articular injection
- Surgery

Gonococcal arthritis (*Neisseria gonorrhoeae*)

Cause

This occurs most frequently in young people. A purulent effusion is uncommon (<30%).

The six 'S's

- **Single/several joints involved?** – Multiple
- **Site(s)/symmetry?** – Involves midsized joints such as knee, wrist and elbow
- **Sequence of symptoms?** – Usually migratory arthralgia
- **Systemic effects?** – Skin rash and other symptoms associated with gonococcal infection
- **Supplementary features?** – Associated with red rash/vesicles over the distal part of the limbs. May also get tenosynovitis of the wrist and hands
- **Specific findings on joint examination?** – The signs are those of an acutely inflamed joint, without any specific to gonococcal arthritis.

Investigations

- Aspirate – send for GCFT (gonococcal fixation test)
- Serum for GCFT
- Urethral or high vaginal swab

Treatment

- Antibiotics
- Refer to genitourinary medicine for treatment, contact tracing and screening for other sexually transmitted diseases

Viral

Arthralgia is a common symptom of viral infection along with malaise, fatigue, fever, myalgia, headache and sore throat. These features will occur before any arthritis.

The six 'S's

- **Single/several joints involved?** – Multiple
- **Site(s)/symmetry?** – Symmetrical, small joints, of the hands, wrists, knees (parvovirus, rubella)
- **Sequence of symptoms?** – Self limiting (usually), stiffness is most marked in the morning
- **Systemic effects?** – As above
  – Morbilliform rash and lymphadenopathy with rubella
- **Supplementary features?** – As above
- **Specific findings on joint examination?** – Painful, tender, swollen joints.

Investigations

Positive viral serology, Interestingly rheumatoid factor may be positive in rubella.

Treatment

This is symptomatic with analgesia and NSAIDs (for further detail on NSAIDs, see Chapter 9).

Spondyloarthritis

This represents a collection of conditions that are seronegative (for rheumatoid factor) and have several common symptoms. The types you are most likely to see can be remembered by the acronym 'PEAR'.

P – Psoriatic arthritis
E – Enteropathic arthritis
A – Ankylosing spondylitis
R – Reactive arthritis (Reiter's syndrome)

The six 'S's

- **Single/several joint involved?** – Multiple or monoarthritis of large joints (small joints involved in psoriatic arthritis); characterised by enthesopathy, i.e. develop inflammation at the site of ligamentous insertion into bone
- **Site(s)/symmetry?** – Spine and sacroiliac joints; asymmetrical when involves several joints
- **Sequence of symptoms?** – Depends on the specific type of spondyloarthritis (see later)
- **Systemic effects?** – Can include uveitis
- **Supplementary features?** – Depends on the specific type of spondyloarthritis, but can include calcification of tendon insertions, uveitis, aortic regurgitation, upper zone pulmonary fibrosis, and amyloidosis
- **Specific features on joint examination?** – Asymmetrical, dactylitis, oligoarticular, sacroiliitis.

Psoriatic arthritis

Commonly, this involves the terminal interphalangeal joints asymmetrically, but there are other different presentations. The patient usually also has the skin and nail manifestations of psoriasis.

Enteropathic arthritis

Usually involves the knees and ankles. Typically this person has associated inflammatory bowel disease.

Ankylosing spondylitis

Typically seen in young males presenting with back stiffness and pain, especially in the morning. Although rare, they may eventually get the 'question mark' spinal posture (kyphotic spine with hyperextended neck) due to progressive spinal fusion, a fixed spinocranial ankylosis and restricted respiration. In 50% of cases, the hip is involved and in 25%, the knee and ankle are affected. Associated with anterior uveitis and aortic regurgitation.

Reactive arthritis (Reiter's syndrome)

This comprises a triad of urethritis, conjunctivitis and seronegative arthritis. In the UK this typically presents in young male patients with lower limb involvement including enthesitis (heels) dactylitis, asymmetrical oligoarticular changes. Asymmetrical with extra-articular features including oral ulceration, circinate balanitis and keratoderma blennorrhagicum. A recent history of non-specific urethritis or diarrhoea can occasionally be elicited.

Investigations

HLA B-27 is present in 88–96% of patients with ankylosing spondylitis, but only 20% of the HLA B-27 population develops ankylosing spondylitis. The spinal

X-ray may also show 'bamboo spine' as well as squaring of the vertebra and erosions of the apophyseal joints. Obliteration of the sacroiliac joint is also commonly visible in established cases. Bilateral sacroiliitis is the characteristic radiological feature.

Treatment

Early rheumatological referral is necessary. Exercise must be started early to maintain as much mobility and posture as possible. In the other types of spondyloarthritis, exercise should be started once the acute symptoms have settled. Treat the underlying condition.

Time Out 17.1

Construct an algorithm to include the important steps in the diagnosis and management of a hot swollen joint.

Key point

Do not leave a septic joint until the next day – infection can destroy a joint in under 24 h

SUMMARY

A hot joint is easy to recognise, but has many causes. A systematic approach is therefore needed. This includes a well-'phrased' history followed by a six 'S' assessment and appropriate investigations.

Advanced Life Support Group

CHAPTER 18
The patient with a rash

OBJECTIVES

After reading this chapter you will be able to:
- understand the common terms used in dermatology
- discuss cutaneous manifestations of life-threatening illness
- apply a structured approach to the assessment and management of the patient with a rash.

INTRODUCTION

The skin is a large organ which may be affected by a primary disorder or manifest signs of systemic illness. It may also provide the signs required to diagnose immediately life-threatening illnesses. Careful assessment using a structured approach is necessary to distinguish the serious from a coincidental or innocuous rash. Early recognition of these signs will allow prompt, potentially lifesaving, disease specific therapy to be initiated.

This chapter will provide you with a structured approach to common dermatological problems – some of which are life-threatening.

USEFUL TERMINOLOGY

- **Angio-oedema:** Similar to urticaria but involves the subcutaneous tissues, especially the face, lips and tongue. Presents with swelling rather than wheals. It is rarely life-threatening, but be wary of laryngeal compromise. Hypotension/anaphylaxis can occur (uncommonly), as can bronchospasm and gastrointestinal disturbance. ABC treatment should include intramuscular adrenaline, intravenous hydrocortisone and antihistamines.
- **Bulla:** A fluid-filled blister greater than 1 cm in diameter, e.g. as in pemphigoid.
- **Ecchymoses:** Bruises (i.e. confluent petechiae).
- **Erosion:** Partial epidermal loss (no scar usually).
- **Erythema:** Redness that blanches on pressure, indicating dilated capillaries. It should be distinguished from purpura, which can be red, orange, purple or brown but do not fade on firm pressure.
- **Erythroderma:** Widespread erythema (i.e. greater than 90% body surface area affected).
- **Macule:** Flat lesions, any colour, e.g. a freckle, and less than 5 mm in diameter.
- **Nikolsky's sign:** Gentle rubbing of the skin causes the epidermis to separate from the underlying dermis with subsequent erosions.
- **Nodule:** A raised rounded lesion greater than 1 cm in diameter.

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- **Papule:** A raised rounded lesion less than 1 cm in diameter, e.g. a mole. Therefore a maculopapular rash consists of raised and flat lesions. These are often less than 1 cm diameter, e.g. as seen in a penicillin rash.
- **Patch:** Larger version of a macule (greater than 5 mm in diameter).
- **Petechiae:** Pinpoint haemorrhage.
- **Plaque:** A raised patch, e.g. a plaque of psoriasis on an elbow.
- **Purpura:** A condition that can be red, orange, purple or brown but does not fade on firm pressure (unlike erythema).
- **Pustule:** A pus filled blister, usually less than 1 cm in diameter, e.g. as in a furuncle (i.e. boil).
- **Ulcer:** Full loss of epidermis and some dermis (scar possible).
- **Urticaria:** Formation of pruritic, transient (<24 h) wheals (like nettle rash). These are collections of dermal oedema surrounded by erythema. Systemic symptoms are extremely rare.
- **Vesicle:** A fluid-filled blister less than 1 cm diameter, e.g. as in herpes labialis.

IMMEDIATELY LIFE-THREATENING EMERGENCIES

Life-threatening illnesses involving the skin may present with signs relating to airway, breathing or circulation.

Immediately life-threatening illnesses with signs in the skin

Airway	**Obstruction**	**Anaphylaxis**
		Angio oedema
Breathing	**Bronchospasm**	**Anaphylaxis**
Circulation	**Shock**	***Meningococcal sepsis***
		Gonococcal sepsis
		Cellulitis
		Anaphylaxis
		Erythroderma

These conditions can be usefully grouped into four different areas by the type of rash associated with the underlying condition. These are:
- urticaria
- erythema
- purpura and vasculitis
- blistering disorders.

Urticaria

This is a common presentation and 15–20% of the population will present to the emergency department with varying degrees of severity. Urticaria can present in many different ways with many associated features, e.g., with:
- anaphylaxis
- angio oedema or
- urticaria alone.

Anaphylaxis

This may present with predominantly dermatological features including pruritus and urticaria. These features are usually florid but may be subtle.

A – Airway
Anaphylaxis can present with upper airway obstruction.

B – Breathing
This can be affected with anything from mild to severe bronchospasm.

C – Circulation
The patients are often hypotensive and can have complete cardiovascular collapse, which is due to a combination of increased vascular permability and vasodilatation.

Specific features
Anaphylaxis can present with gastrointenstinal disturbance. The specific management of this condition is considered in detail in Chapter 9.

Angio oedema

This is a condition that may present with some features similar to anaphylaxis. It is characterised by swelling of the subcutaneous tissues, predominantly affecting the face. Notably these lesions are rarely itchy in contrast to the intense pruritus often associated with urticaria.

A – Airway
Involvement of mucous membranes or the tongue, larynx or pharynx may result in airway obstruction. Angio oedema may herald the onset of anaphylaxis.

Specific features
Frequently no cause is apparent and attacks may be recurrent. Angio oedema should be treated in the same manner as anaphylaxis. Rarely patients may have angio oedema resulting from C1 esterase inhibitor deficiency. This should be treated similarly to other causes of angio oedema but may require specific treatment with either fresh frozen plasma or C1 esterase inhibitor concentrate where available.

Urticaria specific features

Dermographism
This is produced by firm stroking of the skin and leads to the development of an urticarial 'wheal' within 30 min.

Solar and heat urticaria
This is rare and solar urticaria settles once the light stimulus has been removed.

Cholinergic urticaria
This is produced by exercise, heat or emotional stress and is associated with itching, headaches and nausea.

Cold urticaria
This is usually familial or acquired and can be associated with underlying illnesses, e.g. connective tissue disorders. This can be treated with cypoheptadine 2–4 mg twice a day, although there are side effects, specifically drowsiness and increased appetite.

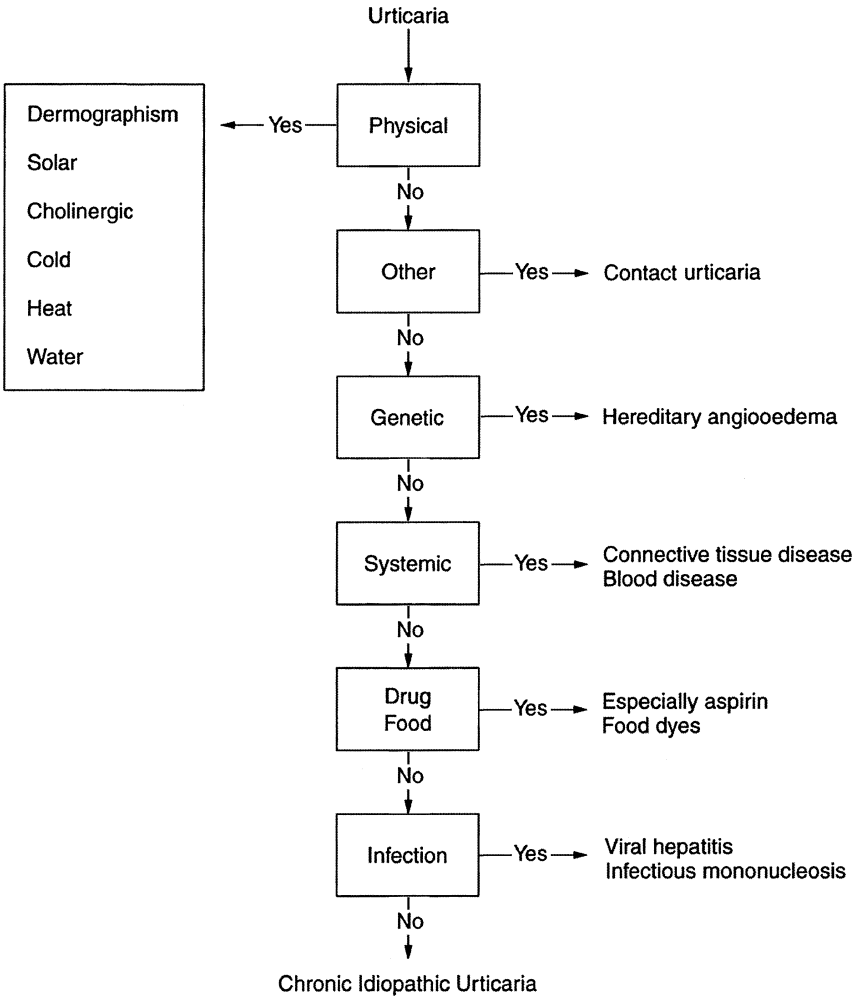

Fig. 18.1 The diagnosis of urticaria.

Contact urticaria

This is produced by foods, textiles, animal dander, plants, tropical medicines and cosmetics. Foods that are commonly involved are fish, eggs and nuts. The most important thing is to remove the causative agent and treat with antihistamines (Fig. 18.1).

Erythema

The patient with red skin (erythema). This is defined as **redness that blanches on pressure** and indicates dilated capillaries. It should be distinguished from purpura which can be red, orange, purple or brown but does not fade on firm pressure. Erythroderma means widespread erythema (i.e. greater than 90% of the body surface area affected).

Erythroderma and Exfoliation

They may result from a number of causes (see next box) although the common feature is marked vasodilatation. Shock may occur from a number of mechanisms including vasodilatation, fluid loss and endotoxin related. A striking clinical feature is the heat radiated by these patients, which may result in problems with thermoregulation.

Causes of erythroderma and exfoliation

Psoriasis
Toxic epidermal necrolysis
Drug eruptions
Staphylococcal scalded skin syndrome
Toxic shock syndrome
Lymphoma
Seborrhoeic dermatitis
Contact dermatitis
Idiopathic
Necrotising Fasciitis

Primary assessment

A–B – Airway and breathing

In view of the widespread erythema, the oxygen consumption is greatly increased, thus high concentrations of inspired oxygen are required.

C – Circulation

Patients with erythroderma, especially where the skin has peeled leaving a large moist area, will continue to lose large amounts of fluid. This fluid should be replaced. The amount of fluid required can be calculated with standard formulae used for burn injuries. Hypovolaemia should be corrected with crystalloid as described in Chapter 9. Inotropic support or vasoconstrictor drugs may occasionally be required to treat the shock after adequate fluid resuscitation. This step should be undertaken in conjunction with specialist advice from Intensive Care. Vascular access preferably in unaffected skin.

D – Disability

Do not forget confusion as a sign of untreated shock

E – Exposure

Distribution of eruption – reassess frequently (necrotising fasciitis, cellulitis) if it grows rapidly it is more urgent. Thermoregulation – prevent heat loss.

Secondary assessment

This will often reveal the underlying cause for erythroderma. A well-'phrased' history may help to identify specific diseases.

Psoriasis, seborrhoeic/contact dermatitis

It is important to obtain a history of previous skin diseases and an enquiry should be made about the recent use of the systemic or topical steroids as abrupt withdrawal may precipitate an acute flare up. There may be other precipitating factors in the history which has caused an acute flare up, of which the patient will be aware.

Toxic epidermal necrolysis

See blistering eruptions.

Drug eruptions
A full drug history must be obtained, including all topical medications. This enquiry must not be limited to prescribed medications but should include over the counter medications, herbal remedies and cosmetic use. The reaction to topical applications may be local or systemic.

Staphylococcal scalded skin syndrome
This commonly occurs in children and there will be history of recent or current infection, e.g. conjunctivitis or otitis media. The patient will be irritable and have a fever, with an orange red macular rash, which is tender. There is a positive Niklosky's sign and within 48 h there is a blistering eruption. Sheets of the epidermis are lost and healing is complete in 5–7 days.

Toxic shock syndrome
This has menstrual and non-menstrual forms and there is also a streptococcal variety – all show similar features. The mortality rate is 1–5% in toxic shock syndrome and 25–75% in streptococcal toxic shock syndrome. The history may include recent tampon use. There is usually an infection with *staphococcal aureus* in the non menstruating toxic shock syndrome and the predisposing factors include influenza, sinusitis, tracheitis, IVDA, HIV, burns, infected contact dermatitis, gynecological infection, post partum and post operative infections. There is usually a fever of greater than 38.9°C, diffuse macular erythroderma with hypotension, nausea, diarrhoea, myalgia and headache. This may lead to acute kidney injury and septic shock. Commonly there is desquamation in 1–2 weeks.

Lymphoma
Erythroderma can occur in patients with Hodgkin's lymphoma and presents with severe pruritus, weight loss and night sweats. Erythroderma is also seen in lymphocytic leukaemia.

Necrotising fasciitis
This usually presents after an insect bite, wound or abscess or following infection of needle tracts (in an IV drug user) or surgical wounds. Mortality rate can be as high as 25% and in some cases there is no associated initial wound. It is characterised by fever, vesicle formations with serious fluid drainage and the erythema rapidly spreads and may become violet in colour. There may be pain, crepitus or painless ulcers. It is caused by synergism between the streptococcal and anaerobic bacterii causing a rapid spread.

Specific treatment of causes of erythroderma

Specific treatment can be started after resuscitation, which includes providing oxygen and IV fluids and a working diagnosis. These may include:

- Covering weeping or open lesions with saline soaked dressings. This may reduce contamination and secondary infection. If large areas are to be dressed, dry sterile dressings should be used to reduce heat loss in the early stages. It may be useful to photograph the rash before covering it.
- Intravenous flucloxacillin should be given (unless contraindicated) if staphylococcal infection is suspected. Topical and prophylactic antibiotics are of no benefit and may cause later complications.
- Intravenous opioid analgesia may be required.
- If necrotising fasciitis is suspected, immediate surgical referral and antibiotics should be arranged.

  Disease specific treatment should be initiated on the basis of specialist advice.

Summary

The assessment and management of the erythematous patient is shown in Fig. 18.2.

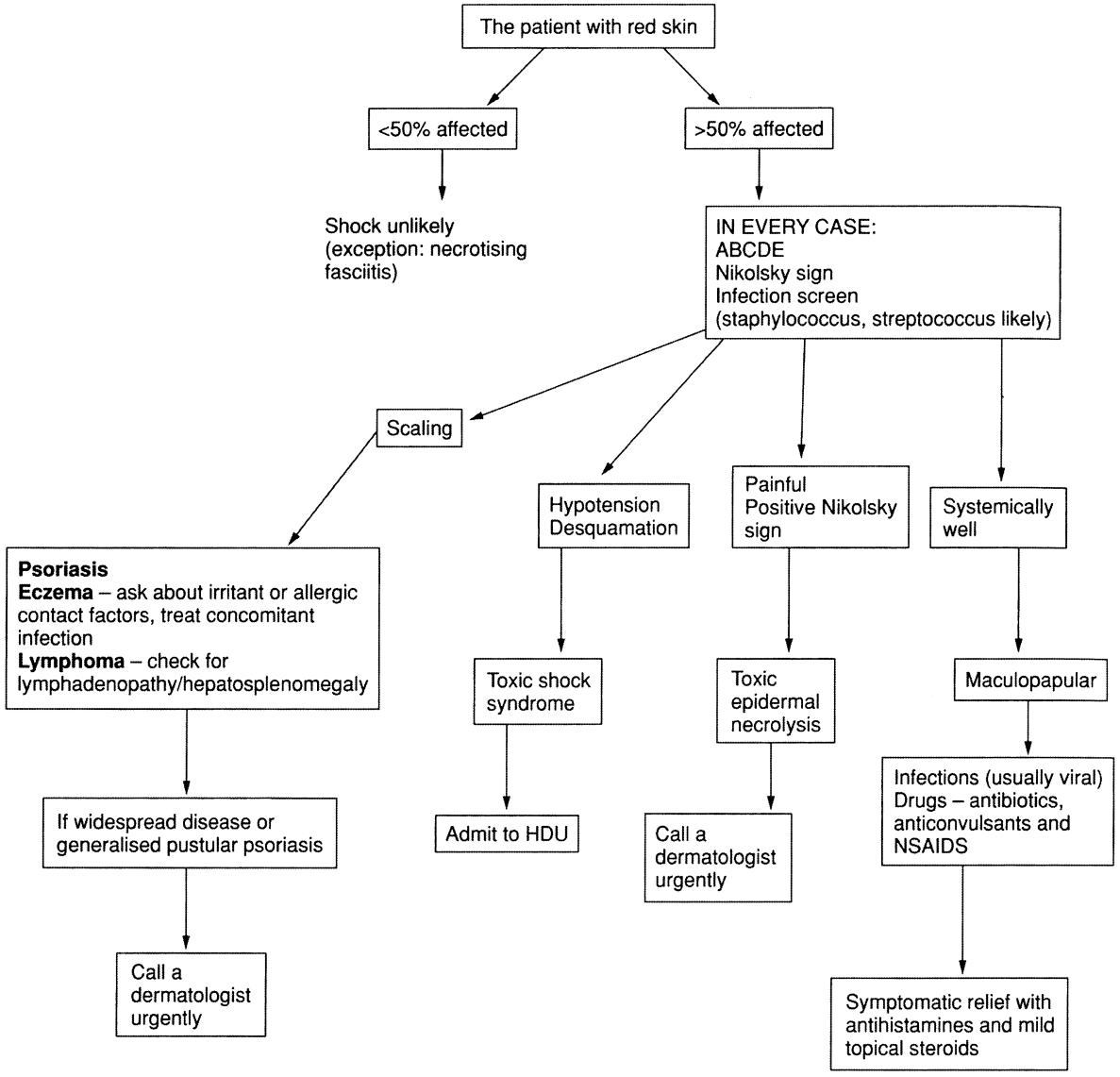

Fig. 18.2 Assessment and management of the erythematous patient.

> **Key point**
>
> **Nikolsky's sign – shearing stress on epidermis causes new blisters**

Purpura and vasculitis

Purpura are caused by red cells leaking out of blood vessels into the dermis. Although the main cause is inflammation of these blood vessels, i.e. vasculitis (see later), other conditions have to be considered.

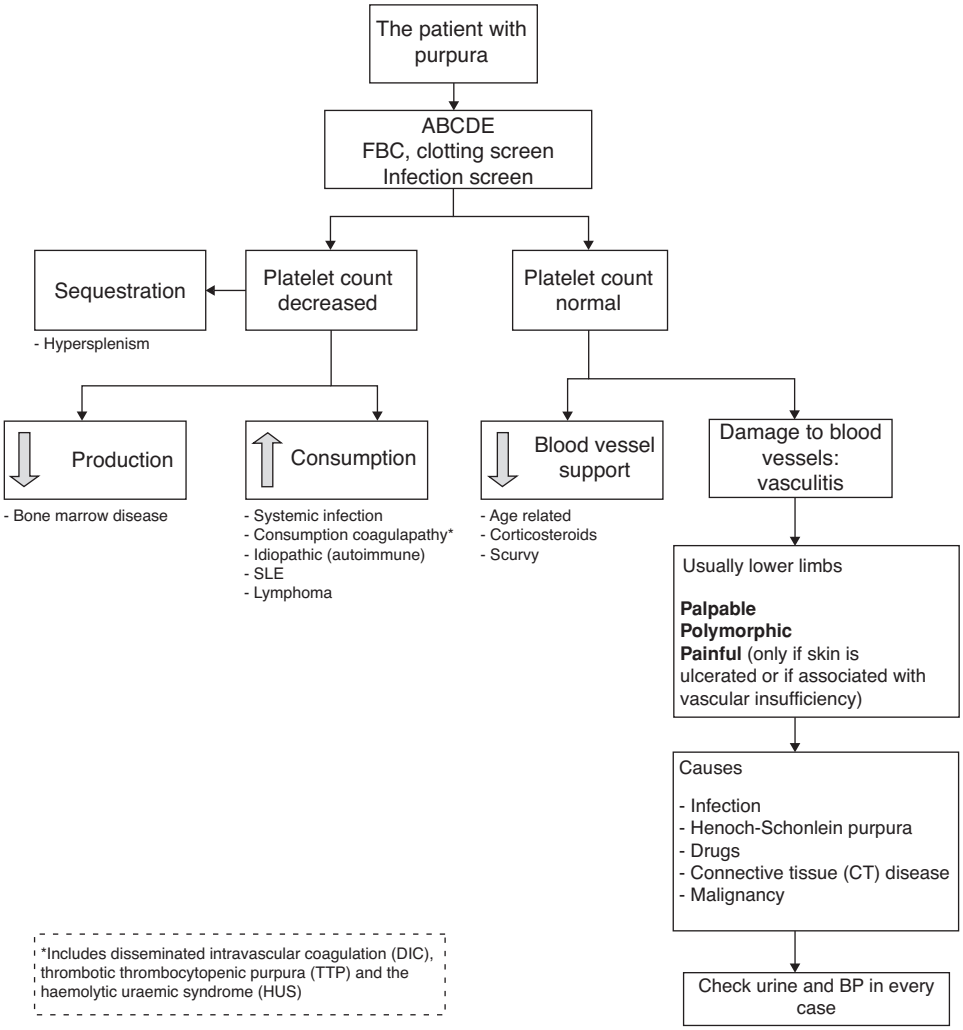

Fig. 18.3 The patient with purpura: an algorithm to aid differential diagnosis.

Key point

Unlike erythema, purpura does not blanch on firm pressure

Purpura may be part of either a rapidly progressive – often septic – illness or, in contrast, a component of a longstanding stable vasculitis. The hallmark of a purpuric rash is the failure to blanch with pressure. This is best seen by pressing on the rash with a microscope slide or a clear drinking glass.

Purpura may be caused by an abnormality of the blood or the vessels. These points are summarised in Fig. 18.3.

Key point

The presence of a purpuric rash in an ill patient is due to overwhelming sepsis until proven otherwise

Primary assessment

There are a number of infections that produce a characteristic clinical picture of a purpuric rash associated with shock.

Infections associated with shock and purpura

Meningococcus (*Neisseria meningitidis*)
Gonococcus (*Neisseria gonorrhoeae*)
Staphylococcus aureus
Rickettsia
Arbovirus

The most common cause is *meningococcal septicaemia*. It is important to remember that this can occur without any symptoms or signs of meningitis. Its management is discussed fully in Chapters 9, 11 and 14 but remember that cefotaxime or ceftriaxone IV (80 mg/kg) should be given immediately. A third-generation cephalosporin is used as penicillin resistant strains of *Neisseria meningitidis* have been isolated. If possible blood cultures should be taken before the first dose of antibiotics.

Key point

If in doubt, treat with ceftriaxone/cefotaxime IV (80 mg/kg) first and investigate for alternative causes later

Secondary assessment

The well-'phrased' history may reveal the presence of systemic symptoms, e.g. urinary symptoms, and abdominal pain combined with fever or joint symptoms may suggest a systemic vasculitis such as Henoch–Schönlein purpura, or polyarteritis, or a systemic infection. A detailed drug history must be obtained. Further investigations should include urine analysis for blood and protein, full blood count, and urea and electrolytes.

The comprehensive physical examination should seek specific clues as to the underlying cause of purpura or vasculitis. Changes to the mental state may be present but may be subtle. This can occur with meningitis, connective tissue diseases or with intracranial bleeding or thrombosis (e.g. in thrombotic thrombocytopenia).

Definitive care of the underlying problem will frequently involve the input of a number of specialties including haematology, immunology, rheumatology, general medicine and intensive care. Do not delay resuscitative treatment for a specialist opinion.

Blistering disorders

- Blisters are accumulations of fluid that occur in two common sites.
- Within the epidermis (intraepidermal) – often having a thin roof and therefore burst early, like herpes, pemphigus.
- Under the epidermis (subepidermal) – often thick walled, like pemphigoid.
- Any may contain blood.

Although there are more comprehensive classifications, the differential diagnosis of blistering eruptions is based on whether they are painless (Fig. 18.4) or painful (Fig. 18.5).

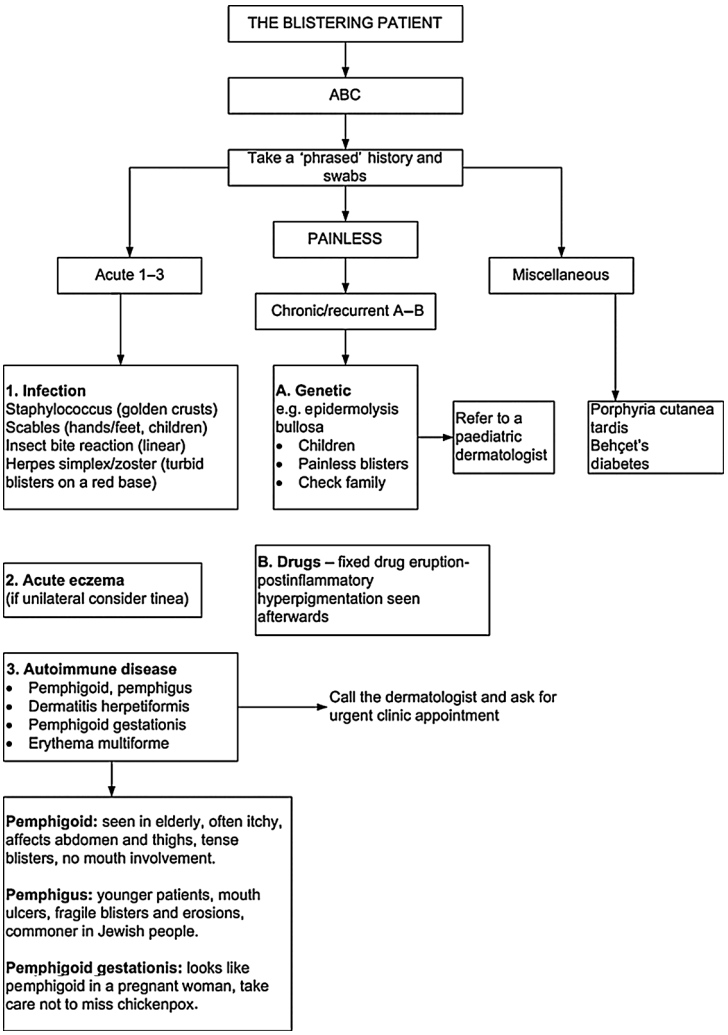

Fig. 18.4 Painless blistering eruptions.

IS THIS STEVENS–JOHNSON SYNDROME (SJS) OR TOXIC EPIDERMAL NECROLYSIS (TEN)?

Terminology in these conditions can be confusing. Stevens–Johnson syndrome is considered to be erythema multiforme characterised by purpuric macules and 'target lesions' in association with mucous membrane (ocular, oral, genital) involvement. However, erythema multiforme is often maculopapular rather than the classic target appearance. To help differentiate it from the usual maculopapular eruptions, there is mucosal involvement, pain and possible progression to toxic epidermal necrolysis. This is heralded by the development of the Nikolsky sign.

Toxic epidermal necrolysis, with full thickness loss of the epidermis, presents with tenderness and widespread red macules and target lesions of the skin followed by exfoliation (like a scald). It may follow Stevens–Johnson syndrome or appear *de novo* and often starts in the flexures. This is a dermatological emergency, which requires swift recognition and treatment. The mortality rate is 30–35%, which increases in the elderly (who are usually on the most medication). Management needs to be on a high dependency unit.

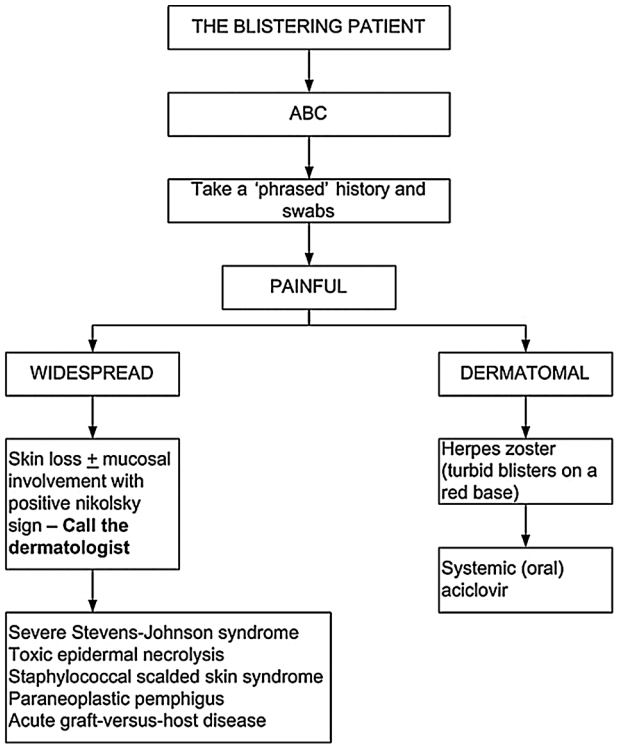

Fig. 18.5 Painful blistering eruptions.

Causes of Stevens–Johnson syndrome

- Sulphonamides and NSAIDs
- Herpes simplex, streptococcus, mycoplasma

Causes of toxic epidermal necrolysis

- Drugs are the commonest cause of toxic epidermal necrolysis (TENs) including anticonvulsants, antibiotics (especially sulphonamides) and NSAIDs
- Infection (bacterial, viral, fungal)
- Idiopathic
- As a sequel to Stevens–Johnson syndrome
- Rarely as a paraneoplastic phenomenon
- Human Immunodeficiency Virus (HIV)

If you suspect Stevens–Johnson syndrome/toxic epidermal necrolysis

ABCDE (IVI should be peripherally placed in uninvolved skin; oral intake of fluids may be impossible). Admission to critical care must be considered.
pain relief is crucial
take a detailed drug history and stop any offending drugs
an infection screen is needed as sepsis is the main cause of death

do not use flamazine (crossreacts with sulphonamides and causes neutropenia)
do not give steroids
call the dermatologist urgently to confirm the diagnosis and supervise treatment
keep the environmental temperature 30–32°C to prevent heat loss.
a prompt referral decreases the risk of infection, mortality rate and length of hospital stay.

Differential diagnosis of toxic epidermal necrolysis

Staphylococcal scalded skin – seen in children, may see evidence of impetigo, especially around the mouth and nose.
Paraneoplastic pemphigus – usually associated with haematological malignancy; may look very similar to Stevens–Johnson syndrome with subsequent toxic epidermal necrolysis but responds to steroids; characteristic histology.
Acute graft-versus-host disease.

Infection and the skin

The skin may be directly involved in an infective process. Cellulitis is a common problem that can occasionally become life- or limb threatening if necrotising fasciitis develops or sepsis ensues. The infecting organism causing cellulitis is usually a group A streptococcus. First-line treatment should be with penicillin.

Primary herpes zoster (varicella or chicken pox) is an unpleasant illness. However, it may become life-threatening in immunocompromised patients (e.g. post transplant, high dose steroids, acquired immunodeficiency syndrome). The rash is characterised by the simultaneous presence of vesicles, pustules and crusted lesions. It is important in the immunocompromised patient to recognise the illness as early as possible, when only a handful of vesicles may be present. The illness may be complicated by pneumonitis. Immunocompromised patients with primary herpes zoster should be treated with an intravenous antiviral agent, such as aciclovir.

Time Out 18.1

a List the four categories of dermatological conditions that can be immediately life-threatening.
b Draw the algorithm for each condition.

SUMMARY

- Life-threatening skin conditions are rare but may be rapidly fatal.
- Resuscitation may involve specific treatments including adrenaline for anaphylaxis and benzyl penicillin for *meningococcal septicaemia*.
- A careful history and examination may be required to elicit subtle features in the early stages of life-threatening illness.
- Seek specialist advice early.
- Most dermatological conditions presenting as an emergency are not life-threatening.

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CHAPTER 19
The patient with acute confusion

OBJECTIVES

After reading this chapter you will understand:
- why acute confusional states occur
- the underlying cause
- the immediate management.

INTRODUCTION

Acute confusional state is a common condition. These medical emergencies can occur at home or arise in any hospital patient, usually as an unexplained behavioural change. The particular role of the reticular formation in the brain stem and the cerebral cortex in maintaining consciousness has been discussed in chapter 11 on the unconscious patient. This interlink to neurological activity is often suppressed in the acute confusional state, commonly caused by an acute illness (usually an infection, or the adverse effect of a drug).

Key point

Always consider an acute confusional state in any patient who is difficult, uncooperative or unable to give a reliable, accurate, history

CAUSES OF ACUTE CONFUSION

There are many causes of confusion; some of the common ones are listed in Table 19.1.

INITIAL ASSESSMENT

This should follow the standard format. Rather than describe a complete assessment, this will be tailored to specific features relevant to the patient with an acute confusional state.

Primary assessment

Ensure airway patency.
Exclude hypoxaemia.
Exclude shock; check glucose.
Exclude meningeal irritation, reduction in Glasgow Coma Score and lateralising signs. Make sure pupil response is appropriate.
Check temperature. Examine for a rash.

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Table 19.1 Common causes of confusion

Systemic illness	Infection	Urinary or respiratory
	Organ failure	Hypoxaemia Hypoglycaemia Uraemia Liver failure
	Metabolic	Hypoglycaemia Electrolyte imbalance Hypercalcaemia
Primary neurological Disease		Status epilepticus Postictal state Non-dominant parietal lobe stroke Subarachnoid haemorrhage Subdural haematoma Raised intracranial pressure Viral encephalitis
Drugs		Recreational opioids Benzediazepines Tricyclics Antiparkinsonian treatment
Alcohol		Intoxication Withdrawal

SECONDARY ASSESSMENT – PHRASED HISTORY

This is only available from relatives or carers – or may not be available at all. However if present, the history usually describes impairment of consciousness over hours or days and typically worse at night. Confusion can manifest as global impairment of cognitive processes or impairment of thought, perception and memory. The patient is usually disorientated as assessed by the Abbreviated Mental State Score (see Chapters 3 and 7). There are often wild changes in behaviour which are out of character, and range from inactivity to irritability, hyperactivity and noisiness.

Thought processes are often slow, with delusion. Visual hallucinations classically dominate but perceptual defects can also include auditory and tactile hallucinations.

During the secondary assessment, it is important to repeat the primary assessment observations and check for any focal signs of infection.

Key point

Core temperature is more reliable than peripheral in the confused patient

Request a urine dipstick. Check for clinical features of uraemia or hepatic encephalopathy, including flapping tremor (asterixis) and stigmata of chronic liver disease. A comprehensive neurological examination is required, but this may be limited by lack of cooperation from the confused patient. Recheck the Glasgow Coma Score to ensure that there has been no deterioration and reassess the pupils.

Look for lateralising signs and features that may suggest drug use, in particular nystagmus and ataxia of Wernicke's encephalopathy. Exclude urinary retention and faecal impaction – which occasionally, in the elderly, may be responsible for a change in conscious level.

By the end of the secondary assessment, a series of investigations will have been requested, to either support the clinical diagnosis or find the underlying cause of confusion. The minimum list of immediate investigations is shown in the next box.

List of immediate investigations in the confused patient

Glucometer and formal blood glucose
Sodium, potassium, urea and creatinine
Plasma calcium
Full blood count
Prothrombin time
C reactive protein/ESR
Blood cultures, urine dipstick test, microscopy and culture, chest X-ray
Arterial gases
ECG

Management

Specific management is directed at the underlying cause. Sedation should not routinely be required. If, however, the patient is aggressive or is at risk of self-injury, then sedation should be given; ideally with the patient being fully monitored on a high dependency unit. Remember the risk of sudden collapse or paradoxical agitation with some of the commonly used sedating agents in these circumstances.

Most patients with an acute confusional state have an underlying organic cause, especially if there is no previous psychiatric history. Always check the drug chart in hospital patients who become confused. Consider status epilepticus if there are myoclonic movements of the eyelids, face or hands. Demented patients are often diagnosed as confused and vice versa. Remember that patients with dementia often deteriorate and become confused, in response to an acute illness. Features differentiating acute confusional state, dementia and acute function psychosis are given in Table 19.2.

Table 19.2 Clinical features of acute confusional state, dementia and acute functional psychosis

Characteristic	Acute confusional state	Dementia	Acute functional psychosis
Onset	Sudden	Insidious	Sudden
Course over 24 h	Fluctuating, nocturnal exacerbation	Stable	Stable
Consciousness	Reduced	Clear	Clear
Attention	Globally disordered	Normal, except in severe cases	May be disordered
Orientation	Usually impaired	Often impaired	May be impaired
Cognition	Globally impaired	Globally impaired	May be selectively impaired
Hallucinations	Usually visual, or visual and auditory	Often absent	Predominantly auditory
Delusions	Fleeting, poorly systematised	Often absent	Sustained, systematised
Psychomotor activity	Increased, reduced or shifting unpredictably	Often normal	Varies from psychomotor retardation to severe hyperactivity
Speech	Often incoherent, slow or rapid	Difficulty finding words, perseveration	Normal, slow or rapid
Involuntary movements	Often asterixis or coarse tremor	Often absent	Usually absent
Physical illness or drug toxicity	One or both present	Often absent	Usually absent

SPECIFIC CONDITIONS

Alcohol

There are many causes of confusion related to alcohol use (see the next box).

Causes of a confusional state associated with alcohol abuse

Hypoglycaemia
Acute intoxication
Alcohol withdrawal
Wernicke's encephalopathy
Head injury
Chronic subdural haematoma
Alcoholic hepatitis
Hepatic encephalopathy
Acute pancreatitis
Alcoholic ketoacidosis
Lactic acidosis
Other illnesses, particularly pneumonia

If a history is available, then it is possible to exclude some of the above causes – in particular eliminating features of alcohol withdrawal (Table 19.3).

Table 19.3 Clinical features that may occur with alcohol withdrawal

Last drink	Features
Within 6–12 h	Tremor, sweating, anorexia, nausea, insomnia, anxiety ('morning shakes')
	Mild confusional state with agitation
Within 48 h	'Rum fits' – one to six tonic-clonic fits, without focal features, occurring within a 6 h period
After 72 h	Delirium tremens (DT) with tremor, severe confusional state, agitation, visual and auditory hallucinations and paranoid ideation
	Tachycardia, sweating and fever

MANAGEMENT OF ALCOHOL WITHDRAWAL

Exclude hypoglycaemia. If treating hypoglycaemia with IV glucose, it is essential to also give high dose parenteral B vitamins to minimise the risk of precipitating Wernicke's encephalopathy. Rehydration is often required, as is treatment of any intercurrent illness. Vitamin supplements are also required. Thiamine is best given parenterally for at least 72 h (followed by oral thiamine 250 mg/day and vitamin B strong compound, 2 tablets daily). If however, Wernicke's encephalopathy is present, parenteral thiamine should be continued for one week.

The presence of delirium tremens or severe agitation is managed according to local policy and will probably include the use of chlorpromazine (contraindicated in the presence of liver disease), chlordiazepoxide or another benzodiazepine. Any patient who needs intravenous sedation must be managed on a high dependency unit.

ELECTROLYTE DISTURBANCES

Physiology

A 70-kg man has a: total fluid volume of 42 litres (60% body weight). This comprises: Intracellular fluid = 28 litres (67% body fluid), extracellular fluid = 14 litres (33% body fluid) of which the intravascular component = 3 litres plasma (5 litres of blood).

The distribution between intra- and extravascular compartments is determined by osmotic equilibrium, and the 'oncotic pressure' exerted by non-diffusible proteins.

Over 24 h the fluid balance is roughly:

Input (l ml water)	Output (1 ml water)
Drink; 1500	Urine 1500
Food: 800	Insensible loss: 800
Metabolism of food: 200	Stool: 200
Total: **2500**	Total: **2500**

Osmolarity is the number of osmoles per litre of solution.
Osmolality is the number of osmoles per kg of solvent (normally 280–300).

A mole is the molecular weight expressed in grams.

To estimate plasma osmolality calculate $2[Na^+ + K^+]$ + Urea + glucose. If the measured osmolality is greater than this (i.e. an osmolar gap of >10 mmol/l), consider diabetes mellitus, high blood ethanol, methanol or ethylene glycol.

SODIUM

Sodium control

Renin is produced by the juxtaglomerular apparatus in response to decreased renal blood flow and catalyses the formation of angiotensin 1 from angiotensinogen. This is then converted by angiotensin-converting enzyme to angiotensin 11. The latter has several important actions including efferent renal arteriolar constriction (so increasing perfusion pressure); peripheral vasoconstriction and stimulation of the adrenal cortex to produce aldosterone. This activates the sodium pump in the distal renal tubule, leading to reabsorption of sodium, in exchange for potassium and hydrogen ions (excreted in urine).

A high glomerular filtration rate results in high sodium loss, where as *high renal tubular blood flow* and haemodilution decrease sodium reabsorption in the proximal tubule.

Water control

Water is controlled mainly by sodium concentration. An increased plasma osmolality causes thirst and the release of antidiuretic hormone (ADH) from the posterior pituitary. This, in turn, increases the passive water reabsorption from the renal collecting duct, by opening water channels to allow water to flow from the hypotonic luminal fluid into the hypertonic renal interstitium.

Abnormalities

Hyponatraemia

The assessment of a patient with hyponotraemia is based on the history and the fluid balance status, i.e. whether there is evidence of:

- dehydration
- normal volume
- peripheral oedema.

Do not base treatment on the plasma sodium concentration alone.

There may be symptoms and signs of water excess such as confusion, fits, hypertension, cardiac failure, oedema, anorexia, nausea and, muscle weakness.

Diagnosis

The key decision is whether the patient is either dehydrated or overloaded with fluid. History, clinical examination and serum urine analyses are your guides.

Causes of hyponatraemia

- Diuretics, especially thiazides
- Water excess, either orally, or IV as excess 5% dextrose
- Others – see Fig. 19.1.

If the patient is not dehydrated, renal function good, and if Na >125 mmol/l, treatment rarely needed. If Na <125 mmol/l, restrict water to 0.5–1 l/day if tolerated. Consider frusemide (furosemide) 40–80 mg/24 h IV slowly/PO for a few days only. SIADH (below) is occasionally treated by producing nephrogenic diabetes insipidus with demeclocycline.

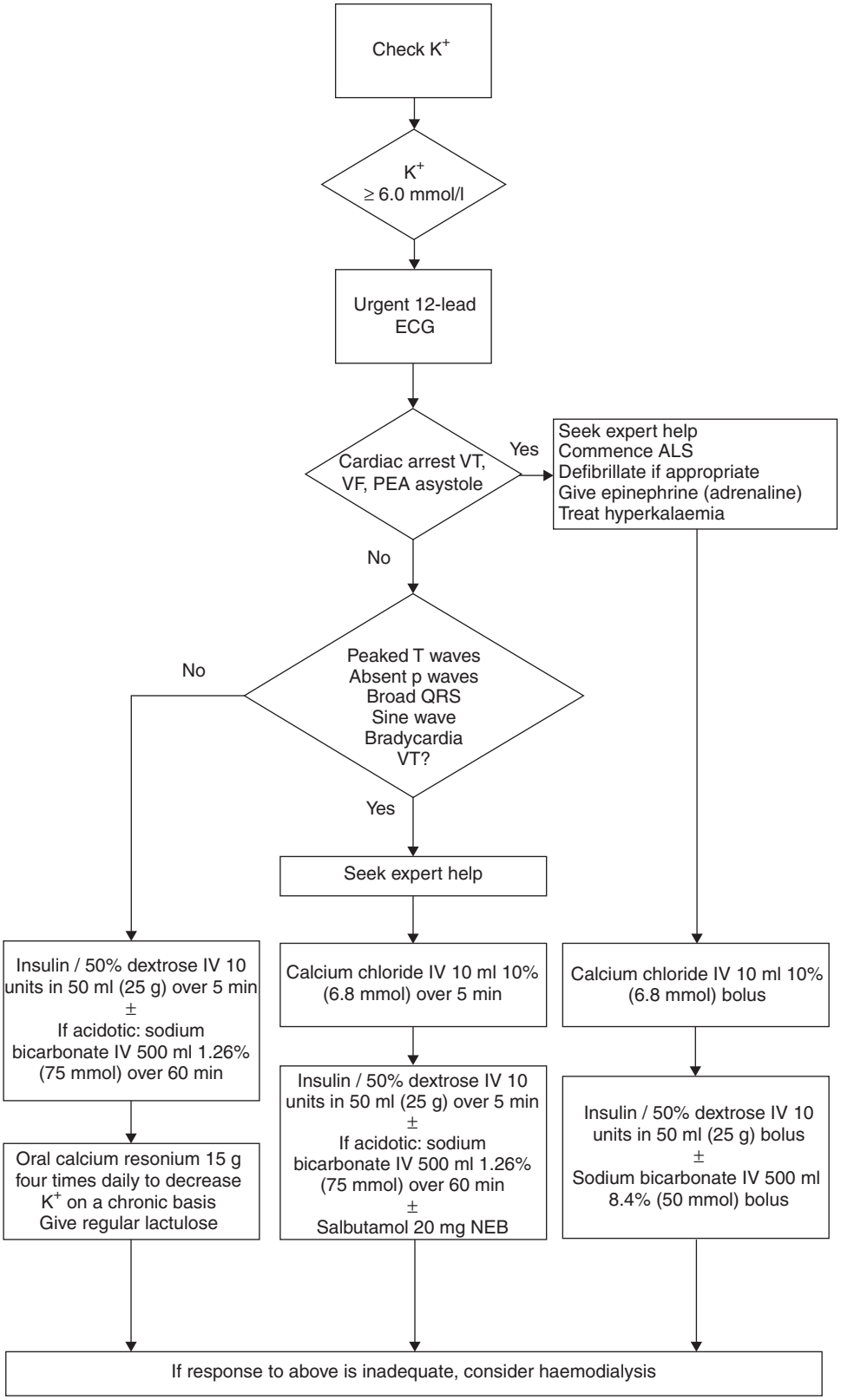

Fig. 19.1 Management of hyperkalaemia.

If the patient is dehydrated and kidney function good, 0.9% saline can be given. In emergency (seizures, coma) consider rapid IVI infusion of 0.9% saline or hypertonic saline (e.g. 1.8% saline) at 70 mmol Na^+/h. Aim for a gradual increase in plasma sodium by 1 mmol/h to about 125 mmol/l. Watch out for heart failure, and central pontine myelinolysis. Seek expert help.

Syndrome of inappropriate ADH secretion (SIADH)

This is an important cause of hyponatraemia, but is over-diagnosed. The diagnosis is made by finding a concentrated urine sodium (>20 mmol/l) in the presence of hyponatraemia (<125 mmol/l) and an inappropriately low plasma osmolality (<260 mmol/kg), with no evidence of hypovolaemia, oedema or diuretics.

Causes

- Malignancy (lung small-cell, pancreas, prostate, lymphoma).
- CNS disorders (meningoencephalitis, abscess, stroke, subarachnoid, subdural haemorrhage, head injury, Guillain–Barré, vasculitis (e.g. SLE).
- Chest disease (TB, pneumonia, abscess, aspergillosis)
- Metabolic disease (porphyria, trauma)
- Drugs (opiates, chlorpropramide, psychotropics, cytotoxics).

Hypernatraemia

Symptoms and signs

Thirst, confusion, coma and fits – with signs of dehydration: dry skin, reduced skin turgor, postural hypotension and oliguria if water deficient.

Laboratory features

Elevated PCV, albumin and urate.

Causes

- Usually due to water loss in excess of sodium loss.
- Fluid loss without water replacement (e.g. diarrhoea, vomiting, burns).
- Incorrect IV fluid replacement
- Diabetes insipidus. Suspect if large urine volume. This may follow head injury, or CNS surgery, especially pituitary
- Osmotic diuresis
- Primary aldosteronism: suspect if hypertension, hypokalaemia and alkalosis.

Management

Give water orally if possible. Otherwise, dextrose 5% IV slowly (4 l/24 h), guided by urine output and plasma sodium. Some authorities recommend giving 0.9% saline, since this causes less marked fluid shifts and is hypotonic in a hypernatraemic patient.

POTASSIUM

Physiology

Most potassium is intracellular, and thus serum potassium levels are a poor reflection of total body potassium. The concentrations of potassium and hydrogen ions in extracellular fluid tend to vary together. This is because these ions compete with each other as they are exchanged with sodium across most cell membranes (sodium is pumped out of the cell) and in the distal tubule of the kidney (sodium is reabsorbed from the urine). Electrical neutrality has to be maintained thus, if the hydrogen ion concentration is high, fewer potassium ions will be excreted into the urine. Similarly K^+ will compete with H^+ for exchange across cell membranes and extracellular K^+ will accumulate.

ABNORMALITIES

Hypokalaemia

If <2.5 mmol/l needs urgent treatment. Note that hypokalaemia exacerbates digoxin toxicity.

Symptoms and signs

Muscle weakness, hypotonia, cardiac arrhythmias, cramps and tetany.

ECG

Small or inverted T waves, prominent U waves, prolonged P-R interval, depressed ST segment.

Causes

- Diuretics
- Vomiting
- Pyloric stenosis
- Alkalosis
- Villous adenoma of the rectum
- Intestinal fistulae
- Cushing's syndrome/steroids/ACTH
- Conn's syndrome
- Purgative and liquorice use
- Renal tubular failure.

If the patient is using diuretics, a raised bicarbonate is the best indication that the hypokalaemia is likely to have been long-standing. The magnesium level may also be low, and hypokalaemia is often difficult to correct until magnesium levels are restored to normal. In hypokalaemic periodic paralysis, intermittent weakness lasting up to 72 h appears to be caused by potassium shifting from the extracellular to the intracellular fluid. Suspect Conn's syndrome if hypertensive, hypokalaemic alkalosis in someone not taking diuretics.

Treatment

mild: (>2.5 mmol/l, no symptoms) give oral potassium supplement (at least 80 mmol/24 h, e.g. Sando-K® 2 tablets bd). If the patient is taking a thiazide diuretic, hypokalaemia >3.0 mmol/l rarely needs treating.

severe: (<2.5 mmol/l, dangerous symptoms) give IV potassium cautiously, not more than 20 mmol/h, not more than 40 mmol/l. Do not give potassium if the patient is oliguric.

> **Key point**
>
> **Never give potassium as a fast 'stat' bolus dose**

Hyperkalaemia

A plasma potassium >6.5 mmol/l needs urgent treatment, but first ensure that this is not an artefact (e.g. due to haemolysis during or after venesection).

Symptoms and signs

Muscle weakness/paralysis and cardiac arrhythmias, sudden death.

ECG

Tall tented T waves, small flat P waves, wide QRS complex becoming sinusoidal, VF.

Causes

- Oliguric renal failure
- Potassium sparing diuretics
- Angiotensin converting enzyme inhibitors (especially if the patient has type IV renal tubular acidosis)
- Rhabdomyolysis
- Metabolic acidosis
- Excess K^+ therapy
- Artefact. Haemolysis of blood sample; delay in analysis – potassium leaks out of RBCs; thrombocythaemia – platelets leak potassium as sample clots in tube
- Addison's disease (see page XXX)
- Massive blood transfusion

Treatment

Ensure correct fluid balance. Treat or remove the underlying cause. Then consider:

- Check K^+ and other electrolytes (1–4 hourly) and monitor ECG
- $K^+ > 6.0$ – Give 100 ml 50% glucose solution with 10 units regular (rapid-acting) insulin IVI.
- $K^+ > 6.5$ – Treat as for (a), but infuse calcium gluconate, 10 ml 10% solution over 10–15 min (stabilizes cardiac cell membranes) until ECG changes revert to normal.
- $K^+ > 7.0$ – as for (a) and (b) above, and infuse 50–100 ml 8.5% sodium bicarbonate over 30 min (unless patient in respiratory failure). Repeat glucose and insulin 2–4 hourly prn
- Rectal calcium resonium 30 mg once daily to decrease K^+ on a chronic basis.

If response to above is inadequate, consider nebulised salbutamol, furosemide and haemodialysis.

CALCIUM

Physiology

About 40% of plasma calcium is bound to albumin. Usually it is the total plasma calcium which is measured. The unbound, ionised portion is important. Therefore, *adjust calcium level for albumin as follows*: Add 0.1 mmol/l to calcium concentration for every 4 g/l that albumin is below 40 g/l, but subtract 0.1 mmol from calcium contraction for every 4 g/l that is raised above 40 g/l albumin. However, many factors affect binding (e.g. other proteins in myeloma, cirrhosis, individual variation) so be cautious in your interpretation. If in doubt over a high Ca^{2+}, take a blood sample with the limb uncuffed and the patient fasted.

Calcium metabolism is controlled by:

- Parathyroid hormone (PTH): PTH secretion is controlled by ionised plasma calcium levels. A rise in PTH causes a rise in plasma calcium and a decrease in plasma phosphate: This is due to an increased calcium and phosphate: reabsorption from bone; and increased calcium but reduced phosphate reabsorption from the kidney. PTH secretion enhances active vitamin D formation.
- Vitamin D: Calciferol (vitamin D_3) and ergocalciferol (vitamin D_2) are biologically identical in their actions. Serum vitamin D is converted in the liver to 25-hydroxy Vitamin D (25-(OH)D). In the kidney a second hydroxyl group is added to form the biologically active 1.25-dihydroxy vitamin D (1.25-(OH)2-D), also called calcitriol, or the much less active 24.25-(OH)2-D. Calcitriol production is stimulated by reduced calcium reduced phosphate and PTH. Its

actions include increased absorption of calcium and phosphate from the gut; increased reabsorption of calcium and phosphate by the kidney; enhanced bone turnover; and inhibition of PTH release. Disordered regulation of 1.25-(OH)2-D underlies familial normocalcaemic hypercalciuria which is a major cause of calcium oxalate renal stone formation.

- Calcitonin: From thyroid C cells, causes a decrease in plasma calcium and phosphate, but its physiological role is unclear. It is a marker for medullary carcinoma of the thyroid.
- Thyroxine: (=Levothyroxine) may increase plasma calcium, although this is rare.
- Hypomagnesaemia: Prevents PTH release, and may cause hypocalcaemia.

Key point

A low Mg is associated with either a low calcium, or a low potassium or both

Abnormalites

Hypocalcaemia

Apparent hypocalcaemia may be an artefact of hypoalbuminaemia (above).

Symptoms and signs

Tetany, depression, perioral paraesthesiae, carpo-pedal spasm (wrist flexion and fingers drawn together), especially if brachial artery occluded with blood pressure cuff (Trousseau's sign), neuromuscular excitability, e.g. tapping over parotid (facial nerve) causes facial muscles to twitch (Chvostek's sign). Cataract formation, if chronically reduced calcium.

ECG

Prolongation of Q-T interval.

Causes

It may be a consequence of thyroid or parathyroid surgery. If the phosphate level is raised, consider either chronic renal failure, hypoparathyroidism or pseudohypoparathyroidism. If the phosphate level is either normal or reduced suspect either osteomalacia (high alkaline phosphatase), overhydration or pancreatitis.

Treatment

- Mild symptoms, give calcium 5 mmol/6 h PO. Do daily plasma calcium levels. In chronic renal failure, if necessary, add alfacalcidol; start at 0.5—1 mg/24 h PO.
- Severe symptoms, give 10 ml (2.32 mmol) calcium gluconate 10% IVI over 30 min (bolus injections are only needed very rarely). Repeat as necessary.

Hypercalcaemia

Symptoms and signs

Abdominal pain, vomiting, constipation, polyuria, polydipsia, depression, anorexia, weight loss, tiredness, weakness, hypertension, confusion, pyrexia, renal stones, renal failure, corneal calcification, cardiac arrest.

ECG

Q-T interval reduced.

Causes and diagnosis

Bloods: Measure U&Es, magnesium, creatinine, calcium phosphate, ALK PHOS. Most commonly malignancy (myeloma, bone metastases, PTHrP increased and 10 hyperparathyroidism). Pointers to malignancy are: low plasma albumin, low-ish chloride, hypokalaemia, alkalosis, raised phosphate and raised alkaline phosphatase. Other investigations (e.g. isotope bone scan, CXR, FBC) may also be of diagnostic value.

Treatment

Treat the underlying cause. Patients who have a calcium >3.5 mmol/l, or with hypotension, severe abdominal pain, vomiting, pyrexia, confusion, aim to reduce calcium as follows:

- Fluids: Rehydrate with IVI 0.9% saline, e.g. 4–6 litres in 24 h as needed. Correct hypokalaemia and hypomagnesaemia (mild metabolic acidosis does not need treatment). This will reduce symptoms, and increase renal calcium loss. Monitor U&E during treatment.
- Diuretics: Frusemide 40 mg/12 h IV, once rehydrated. Avoid thiazides.
- Bisphosphonates: A single dose of pamidronate (30 mg IVI over 4 h in 0.9% saline) will lower calcium over 2–3 days. Maximum effect is at 1 week. Inhibit osteoclast activity, and so bone reabsorption.
- Steroids: Occasionally used, e.g. in sarcoidosis.
- Salmon calcitonin: Now rarely used. More side effects than bisphosphonates, but quicker onset. Again inhibits osteoclasts.
- Other: Chemotherapy may reduce calcium in malignant disease, e.g. myeloma.

Magnesium

Physiology

Magnesium is distributed 65% in bone and 35% in cells. Its level tends to follow those of Ca^{2+} and K^{+}. Magnesium excess is usually caused by renal failure, but rarely requires treatment in its own right.

Hypomagnesemia

Symptoms and signs

Paresthesiae, fits, tetany, arrhythmias. Digitalis toxicity may be exacerbated.

Causes

Severe diarrhoea, ketoacidosis, alcohol, total parenteral nutrition (monitor weekly), accompanying hypocalcaemia, accompanying hypokalaemia (especially with diuretics).

Treatment

If needed, give magnesium salts either orally or IV (dose example: 10 mmol $MgSO_4$ IVI over 3 min–2 h, depending on severity; monitor Mg^{2+}).

Hypermagnesaemia

Symptoms and signs

Neuromuscular depression, followed sequentially by hypotension, CNS depression and finally coma.

Causes

- Acute on chronic renal failure
- Metabolic acidosis

- Magnesium containing medications
- Adreno-cortisol insufficiency

Treatment

With cardiac conduction defects urgent treatment is needed, intravenous calcium may be given cautiously with ECG monitoring. Forced diuresis can also be used, although dialysis is the treatment of choice especially in renal failure.

SUMMARY

- Acute confusion is a common medical condition.
- An infection or an adverse effect of drugs is the usual cause.
- Most causes of confusion are not immediately life-threatening.

PART IV
Organ Failure

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Advanced Life Support Group

CHAPTER 20
Organ failure

OBJECTIVES

After reading this chapter you will be able to:
- understand the concept of organ failure and its impact on other body systems
- describe the structured approach to management.

INTRODUCTION

Organ failure is a common medical problem. It may be complete or partial; acute or chronic and single organ or multiple organs. However, it is not a diagnosis but the final stages of an underlying condition.

Patients with organ failures often require intensive medical and nursing management: the presence of organ failure thus has resource implications as well as prognostic implications.

This chapter will consider the management of individual failing organs and the diagnosis and management of some of the underlying conditions.

ACUTE ORGAN FAILURE

Acute organ failures are a medical emergency. The structured approach in Chapter 3 will ensure failing organs are detected and supported.

Primary assessment and resuscitation
- Support life-threatening organ dysfunction
- Limit further organ damage

Secondary assessment and emergency treatment
- Detect and support failing organ systems
- Diagnose underlying disease process

Definitive care
- Treat underlying disease processes
- Place patient in a critical care environment for ongoing organ support

Classification of the level of critical care will ensure that patients with organ dysfunction are managed in the correct environment.

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Level of critical care	Organ systems failing	Environment
Level 1	None (at risk)	Ward (with close observation)
Level 2	One (excluding respiratory failure requiring invasive ventilation)	High dependency unit
Level 3	More than one (or respiratory failure requiring invasive ventilation)	Intensive care unit

The severity of acute multiple organ dysfunction may be scored using the Sequential Organ Failure Assessment (SOFA). This is mainly a tool for audit and research purposes but it illustrates that organ failure is not an all-or-nothing process, and despite its simplicity the score has prognostic implications (Table 20.1).

Table 20.1 SOFA score

Respiratory system	
PaO_2/FiO_2	SOFA score
>400	0
<400	1
<300	2
<200	3
<100	4

Nervous system	
Glasgow Coma Score	SOFA score
15	0
13–14	1
10–12	2
6–9	3
<6	4

Cardiovascular system	
MAP/vasopressors	SOFA score
No hypotension	0
MAP <70 mm Hg	1
Dop ≤5, or dob, any dose*	2
Dop >5, or adr ≤0.1 or noradr ≤0.1	3
Dop >15, or adr >0.1 or noradr >0.1	4

Dop, dopamine; dob, dobutamine; adr, adrenaline; noradr, noradrenaline.
*Doses of inotropes in μg/kg/min for at least 1 h

Liver	
Bilirubin (mg/dl)	SOFA score
<1.2	0
1.2–1.9	1
2.0–5.9	2
6.0–11.9	3
≥12.0	4

Coagulation	
Platelets	SOFA score
>150	0
≤150	1
≤100	2
≤50	3
≤20	4

Renal	
Creatinine (mg/dl) or urine output (u.o.)	SOFA score
<1.2	0
1.2–1.9	1
2.0–3.4	2
3.5–4.9 (or u.o. <500 ml/day)	3
>5.0 (or u.o. <200 ml/day)	4

Any patient with acute respiratory failure should be admitted to either a respiratory care unit or an other level 2–3 facility. Hypoxaemia is the most life-threatening facet of respiratory failure. The goal is to ensure adequate oxygen delivery to tissues, which is generally achieved with a PaO_2 above 8.0 kPa or SpO_2 of at least 92%. Apart from oxygen therapy, various types of respiratory support are used to treat respiratory failure. As well as treating the underlying cause (e.g. antibiotics for pneumonia), the ventilation can be supported in the following ways:

- through an invasive airway, e.g. by intubating the patient
- non-invasive respiratory support via a tight-fitting mask.

CHRONIC ORGAN FAILURE

Chronically dysfunctional organ systems may deteriorate acutely ('acute on chronic failure'). Chronic single organ failure typically produces abnormalities

in multiple other physiological systems which may affect the way a patient copes with a new acute illness. Consider, e.g., the cardiovascular, nutritional and immunological implications of chronic renal failure.

The management of chronic organ failure is beyond the scope of this manual. The principles are given below.

Principles of management of chronic organ failure

- Support failing organ systems.
- Limit further deterioration in organ function.
- Prevent and aggressively treat intercurrent illness.
- Manage multi system consequences of chronic organ failure.

RESPIRATORY FAILURE

Respiratory failure may be defined as an inability of the lungs to maintain normal gaseous composition of the blood when breathing air and is usually classified as:

Type 1 respiratory failure is hypoxaemia without hypercapnia. This is usually due to ventilation/perfusion mismatch and/or shunt.

Type 2 respiratory failure is hypoxaemia and hypercapnia. This is due to reduced alveolar ventilation (reduced total ventilation and/or increased dead space ventilation)

Hypoxaemic respiratory failure is characterised by a low PaO_2 leading to an elevated alveolar-arterial gradient and a low $PaCO_2$ reflecting adequate ventilation but inadequate gas exchange. Hypoxaemia is most commonly due to mismatch of ventilation and perfusion or intrapulmonary right to left shunt. Hypercapnia (or, at least, the reason why $PaCO_2$ rises) is often misunderstood. CO_2 retention occurs with uncontrolled oxygen therapy in patients with chronic hypoxaemia (e.g. some patients with chronic obstructive pulmonary disease). Ventilatory capacity is the amount of spontaneous ventilation that can be maintained without the development of respiratory muscle fatigue. Normally, ventilatory capacity matches demand. Hypercapnic respiratory failure results from either a reduction in ventilatory capacity or an increase in ventilatory demand that cannot be met by the patient's own ventilatory capacity.

These definitions, however, do not recognise early compensated respiratory failure where blood gases may be normal at rest due to compensatory mechanisms, predominantly hyperventilation. Tissue hypoxaemia at rest and on exercise arises as the result of failure of multi-system compensatory mechanisms (see box)

Compensatory mechanisms in respiratory failure

Respiratory	**Tachypnoea increases alveolar PO_2**
Cardiovascular	**Tachycardia and increased cardiac output increase oxygen delivery to tissues**
Haematological	**Polycythaemia increases blood oxygen carrying capacity**

There is inevitably a decrease in exercise ability. Thus, the diagnosis of respiratory failure is largely a clinical one based on symptoms and signs, supported by pulse oximetry and/or blood gas estimation.

Acute respiratory failure

This is most often seen in patients with one of the following:

- a severe asthma attack
- tension pneumothorax
- pulmonary embolus
- severe pneumonia (see Chapter 8).

The management, irrespective of the cause (see the box below) is to ensure a patent airway and supply high concentrations of inspired oxygen via an appropriate mask, assisting ventilation when necessary. The decision to assist ventilation is a **clinical** one, aided by investigations such as blood gas estimation. Assisted ventilation is discussed with acute on chronic respiratory failure below.

Causes of acute respiratory failure

Pulmonary	**Asthma, pneumonia, pulmonary embolus**
Cardiac	**Dysrhythmia, failure, arrest**
Neurological	**Unconsciousness, Status epilepticus, Guillain–Barré syndrome**
Neuromuscular	**Myasthenia gravis**
Trauma	**Head, neck, chest, spinal cord**

Chronic respiratory failure

Acute exacerbations of chronic pulmonary disease are amongst the most common reasons for acute medical admission. The common underlying causes, along with an estimation of their frequency, are listed below.

Causes of chronic respiratory failure

Parenchymal disease	**Chronic bronchitis**	**Daily**
	Emphysema	**Daily**
	Pulmonary fibrosis	**Weekly**
	Bronchiectasis	**Weekly**
	Pulmonary vascular disease	**Weekly**
Obstructive sleep apnoea		**Weekly**
Chest wall problems	**Kyphoscoliosis**	**Weekly**
	Extreme obesity	**Weekly**
Neuromuscular disorders	**Motor neurone disease**	**Annually**
	Cervical cord lesion	**Annually**

The commonest causes are now considered in more detail.

Chronic obstructive pulmonary disease

This is a collective term referring to patients who have chronic bronchitis and/or emphysema. The major cause of both conditions is cigarette smoking.

Pathophysiology

In **chronic bronchitis**, there is an initial increase in mucous production and reduction of ciliary motility reducing sputum clearance. This is followed by airway oedema, inflammation, bronchoconstriction and subepithelial airway

fibrosis. This has the greatest impact on small airways, causing fixed obstruction and leading to the well-known clinical consequences (see box below).

Pathophysiology	Clinical consequences
Increased mucus production, decreased mucus clearance	Cough and increased sputum volumes Susceptibility to infection
Airway inflammation and bronchoconstriction	Variability of symptoms, particularly wheeze
Airway narrowing	Ventilation perfusion mismatching with hypoxaemia Breathlessness, wheeze and decreased exercise ability Prolonged expiration, chest hyperinflation
Chronic hypoxaemia	Respiratory, cardiovascular and haematological compensatory changes (see box above) Hypoxaemia, pulmonary vasoconstriction and pulmonary hypertension leading to right heart failure

In **emphysema**, the alveolar walls are destroyed and, consequently, the alveolar spaces coalesce. This causes two main structural problems:

- Loss of elastic recoil, increasing lung size and compliance
- Loss of support for small airways, allowing dynamic airways collapse during expiration.

The net effect is to cause gross hyperinflation and **air trapping**, where exhalation cannot fully occur. Bronchodilators have limited clinical effect, because the pathology is at alveolar, rather than bronchiolar, level.

Chronic bronchitis and emphysema usually coexist in smoking related lung disease. The respiratory failure produced is typically type 1 (hypoxaemic), because of ventilation-perfusion mismatch. Chronic severe disease may lead to decreased alveolar ventilation and development of type 2 (hypercapnic/hypoxaemic) respiratory failure, as the patient cannot continue to meet the high work of breathing associated with the airway obstruction and 'wasted' dead space ventilation.

Pulmonary fibrosis

Diseases associated with diffuse pulmonary fibrosis are shown in the box.

Diseases associated with diffuse pulmonary fibrosis

Cryptogenic fibrosing alveolitis
Occupation, e.g. asbestosis/pneumoconiosis
Extrinsic allergic alveolitis (usually upper lobe and late in the disease)
Drug, e.g. busulphan, bleomycin, paraquat
Rheumatoid disease
Systemic sclerosis
Systemic lupus erythematosus
Sarcoid

Hypoxaemia is often severe and present at rest, whilst the $PaCO_2$ is generally normal or low. The latter is attributed to hyperventilation increasing the elimination of carbon dioxide. Lung compliance is markedly reduced and, although there may be thickening of the alveolar wall reducing gas diffusion, the main cause of the hypoxaemia is ventilation-perfusion mismatch.

Bronchiectasis

This condition is characterised by chronic dilatation of at least some of the bronchi. The bronchial wall is irreversibly damaged as a consequence of early inflammation or infection of either the bronchus or adjacent lung parenchyma. The normal transport of mucus is impaired and chronic local suppuration ensues.

A variety of conditions are associated with bronchiectasis and they are shown in the box.

Conditions associated with bronchiectasis

Infection:	**Measles pneumonia**
	Whooping cough
	Tuberculosis
	Aspergillosis
Immune related:	**Immunoglobulin deficiency**
	Complement deficiency
Inhalation:	**Gastric aspiration**
	Ammonia inhalation
	Foreign body inhalation
Others:	**α1 antitrypsin deficiency**
	Kartagener's syndrome
	Immotile cilia

The clinical hallmark of bronchiectasis is chronic copious sputum production, typically infected. The mainstay of chronic treatment is assisted sputum clearance with physiotherapy and postural drainage. Acute exacerbations require management of the respiratory failure as well as assisted sputum clearance and careful choice of antibiotics.

Management of acute exacerbations of chronic respiratory failure

The structured approach to medical emergencies (Chapter 3) is followed. In the **Primary Assessment and Resuscitation** phase, the priority is to:

- treat hypoxaemia with controlled oxygen therapy, with or without assisted ventilation (see below)
- assess the severity of the respiratory failure
- identify and treat the reason for the acute exacerbation
- monitor the response to treatment.

Controlled oxygen therapy

Hypoxaemia kills and must be treated. High concentrations of inspired oxygen should be given initially and then arterial oxygenation should be monitored with pulse oximetry and blood gas estimation. Titrate oxygen therapy to achieve an SpO_2 of 90–92% (PaO_2 of 8 kPa).

The major cause of hypoxaemia in a patient with COPD is impaired ventilation/perfusion matching. Patients will compensate by increasing the rate of

ventilation, but this increases the work of breathing (the pink puffer; type 1 respiratory failure).

In contrast, patients with severe COPD and hypercapnia usually have lower tidal volumes, due to a short inspiratory time and an increased respiratory rate ('blue bloater', type 2 respiratory failure). There is little evidence to support the theory that supplemental oxygen in COPD patients 'removes the hypoxaemic drive', causing alveolar hypoventilation and hypercapnia. The major effect is to increase dead space ventilation, probably secondary to worsening ventilation/perfusion mismatch due to a loss of hypoxaemic pulmonary vasoconstriction. Therefore, oxygen therapy should be given to ensure a saturation of 90–92% to reduce hypoxaemia and prevent further hypercapnia. However, in the acute situation, especially when the diagnosis remains in doubt, high concentrations of inspired oxygen should be given and adjusted according to arterial blood gas results. In patients who respond appropriately, it is only necessary to increase the flow to ensure a PaO_2 of 8 kPa. If life-threatening hypoxaemia persists, without increasing hypercapnia to an unacceptable level, the patient will require some form of assisted ventilation.

Key point

The presence of hypercarbia is not a reason to accept hypoxaemia

Hypercarbia is often present during acute exacerbations of chronic respiratory disease. The use of oxygen will usually cause a slight rise in $PaCO_2$, due to a small increase in alveolar dead space. This is usually well tolerated by the patient. On rare occasions, the rise in CO_2 is severe enough to cause obtundation and, ultimately, hypoventilation. These patients are best monitored clinically in a high dependency environment aided by blood gas estimation.

The reason for clinical deterioration is usually bronchospasm further impairing ventilation. Nebulised β_2 agonists will reduce this burden as will therapy with steroids and antibiotics. These will also help to reduce the luminal inflammatory response and infected secretions. Aminophylline is often beneficial in patients who have an acute exacerbation of COPD. This bronchodilator has other benefits, including inotropic stimulation, increased cardiac output and improved renal perfusion. This is of particular benefit in patients who have coexistent ventricular failure.

If the patient does not respond appropriately to treatment, reassessment is required to identify any of the possible causes listed in the next box.

Causes of treatment failure in respiratory failure

Untreated bacterial infection
Sputum retention
Coexistent pneumothorax
Inadequate bronchodilator therapy
Coexistent pulmonary oedema
Underlying dysrhythmia
Inappropriate sedation
Wrong diagnosis

If there is not a rapid improvement in the patient's condition after titrating oxygen therapy (see above) and treating the underlying condition, consider

ventilation. Doxapram as a respiratory stimulant rarely leads to a sustained improvement and is not often used as assisted ventilation is more readily available. Hence, early liaison with an anaesthetist/intensivist is needed.

Assisted ventilation

Advances in technology and application of assisted ventilation have improved outcome in patients with acute-on-chronic respiratory failure. It may be 'non-invasive', delivered by face mask, or 'invasive', delivered via an endotracheal tube or tracheostomy. Non-invasive ventilation has the advantage of maintaining an awake, conversant patient and has a lower risk of hospital acquired pulmonary infection.

Unfortunately, not all suitable patients can tolerate the close fitting masks for long periods. Both types require specialist knowledge to deliver.

Non-invasive ventilation is increasingly available in emergency departments, respiratory wards and medical high dependency areas using simple portable equipment.

The indications and relative contraindictions are shown in the following boxes.

Indications for non-invasive ventilation (NIV)

Respiratory acidosis pH <7.35, $PaCO_2$ >6.5 kPa despite controlled oxygen therapy
Moderate to severe breathlessness with accessory muscle use and paradoxical abdominal motion
Respiratory rate >25/min

Relative contraindications to non-invasive ventilation

Respiratory acidosis pH <7.25
Confused
Somnolence, agitation, lack of cooperation
High risk of gastric aspiration
Glasgow Coma Score <8
Copious or viscous sputum
Facial or pharyngeal trauma, deformity or recent surgery
Recent gastro-oesophageal surgery
Untreated pneumothorax

Non-invasive ventilation in the form of BiPAP (bilevel positive airway pressure) provides the possibility of giving two levels of respiratory support:

- EPAP (expiratory positive airway pressure, similar to CPAP) maintains the airways in an open state, improves alveolar gas exchange, improves oxygenation and increases the functional residual capacity.
- IPAP (inspiratory positive airway pressure) supports the inspiratory effort, reducing the effort of breathing, improves tidal volume and improves CO_2 removal.

Intubation and ventilation may be indicated when hypoxaemia and respiratory acidosis persist despite NIV, or when contraindications for NIV exist. Modern ventilatory techniques and appropriate use of tracheostomy have improved outcome.

The need for invasive ventilation in acute on chronic respiratory failure is a poor prognostic indicator- only a minority of patients survive 12 months. The prognosis improves if there is a treatable cause for the exacerbation.

In the **Secondary Assessment and Emergency Management** phase the priority is to identify and treat the cause of the exacerbation. This is often not clear-cut and typically a combination of therapy is indicated (see box below).

Cause of exacerbation	Therapy
Infection	Antibiotics
Bronchospasm	Bronchodilators, inhaled and/or intravenous steroids to reduce airway inflammation
Sputum retention	Physiotherapy, nasopharyngeal suction, Minitrach/Tracheostomy
Muscle wasting	Reduce work of breathing as able. Nutrition. Exercise programme when recovered sufficiently
Pulmonary oedema	Vasodilators, diuretics +/− inotropic support
Pneumothorax (even a very small one may be highly significant)	Intercostal drain with underwater seal

Summary

Acute on chronic respiratory failure is a common medical emergency. All patients should initially receive high concentrations of inspired oxygen and this should be titrated according to pulse oximetry and the results of blood gas analysis. Early intervention is required by either a respiratory physician or intensivist if the patient fails to respond to treatment with controlled oxygen therapy, bronchodilators, steroids and antibiotics.

Time Out 20.1

A 72-year-old lady, who has COPD presents with acute breathlessness. She has a respiratory rate of 28/min, a hyperexpanded chest with scattered wheezes and a prolonged expiratory phase. Her SpO_2 is 72% on 28% oxygen. What is your immediate management?

CARDIOVASCULAR FAILURE

Introduction

Cardiovascular failure may be defined as failure of the circulation to transport sufficient oxygenated blood to the tissues. In its most acute and severe form, this is **shock**, and is discussed in detail in Chapter 9.

In this section, another manifestation of cardiovascular dysfunction will be considered: cardiac failure leading to pulmonary oedema and/or right ventricular failure.

Acute pulmonary oedema

Pulmonary oedema occurs when there is excessive extravascular lung water, which interferes with pulmonary gas exchange. The factors influencing the accumulation of lung water are defined.

Factors increasing extravascular lung water	Disease process
Increased pulmonary capillary hydrostatic pressure	Cardiac failure
	Fluid overload (renal failure, iatrogenic)
	Neurogenic pulmonary oedema
Increased pulmonary capillary permeability	Sepsis
	Systemic inflammatory response syndrome
	Acute respiratory distress syndrome
	Neurogenic pulmonary oedema
Decreased plasma oncotic pressure	Hypoproteinaemia (rarely an isolated cause)
Negative pressure in alveoli	Airway obstruction with active inspiration ('Negative pressure pulmonary oedema')

The most common cause of pulmonary oedema is cardiac failure, which is now considered in detail.

Cardiogenic pulmonary oedema

Pathophysiology

In **acute cardiogenic pulmonary oedema**, there is an increase in left atrial pressure and consequent increase in pulmonary venous and pulmonary capillary pressure. The rise in left atrial pressure may be due to obstruction to the outflow from the left atrium (e.g. mitral stenosis) or a rise in left ventricular end diastolic pressure (e.g. left ventricular failure).

The distension of pulmonary vessels is sufficient to cause symptomatic breathlessness through vascular neural receptors. However, as the process progresses, fluid accumulates in the pulmonary interstitial spaces as lymphatic drainage is overwhelmed. Finally, fluid may accumulate in the alveolar space. By this stage the patient is fighting for breath and terrified, leading to sympathetic activation increasing left ventricular afterload and heart rate. This usually causes a further decrease in cardiac performance – hence the often explosive onset of pulmonary oedema.

As a consequence of this process, the following changes may occur.

- The lung becomes firm and less compliant producing **increased work of breathing.**
- Reflex **hyperventilation** is due to stimulation of vagal sensory 'J receptors' because of distortion of the lung tissue by oedema.
- Small airways become either narrowed by interstitial oedema or filled with oedema. When they open during inspiration they do so with a click which is represented clinically as **fine crackles**.
- During expiration, early airway closure occurs, producing **wheezing**.
- Reduced ventilation in less compliant areas leads to local **hypoxaemia** and reflex arteriolar constriction. This reduces perfusion and diverts blood to less affected areas. This improves the V/Q mismatch, but raises pulmonary artery pressures and increases right ventricular afterload.

In **acute on chronic heart failure**, there may be several other pathophysiological and compensatory mechanisms already active which influence the progression of the heart failure:

- Chronically reduced cardiac output leading to:

  - renal retention of sodium (renin–angiotensin–aldosterone mechanism) and water (ADH), increasing ventricular preload
  - sympathetic nervous system induced vasoconstriction, increasing ventricular afterload
  - sympathetic nervous system mediated increased heart rate and force of contraction (positive chronotropy and inotropy)
  - downregulation of myocardial β1 receptors reducing the positive inotropic effect of sympathetic stimulation (and exogenous adrenoceptor agonists such as dobutamine and dopamine)
  - disordered calcium release and binding within the heart muscle cells further limiting the effectiveness of endogenous and exogenous positive inotropes.
- Decreased myocardial compliance reducing diastolic ventricular filling
- Ventricular dilatation due to chronically increased preload
  - Functional valvular regurgitation
  - For a given end-diastolic pressure, the dilated ventricle has a greater tension within the myocytes (Laplace's law). A further increase in preload is therefore produced.

Principles of therapy in cardiogenic pulmonary oedema

- Clear excessive lung water:
  - improve left ventricular performance (cardiac output)
  - optimise heart rate and rhythm
  - decrease total body sodium and water load.

Preload needs to be reduced, both to improve ventricular performance and reduce pulmonary venous pressures. Afterload is usually severely increased and needs reduction if the consequent reduced blood pressure can be tolerated. Contractility may need improving if the above measures are ineffective or cannot be tolerated (Fig. 20.1).

The optimum **heart rate** depends on the condition producing the heart failure. Slower heart rates (less than 60–80/min) may reduce cardiac output, but faster heart rates impair diastolic ventricular filling, particularly if the ventricle is hypertrophied and non-compliant. The onset of atrial fibrillation may precipitate heart failure in conditions of slow ventricular diastolic filling, e.g. mitral stenosis.

Decreasing total body sodium and water with diuresis takes more time than the previous measures and requires adequate cardiac output and blood

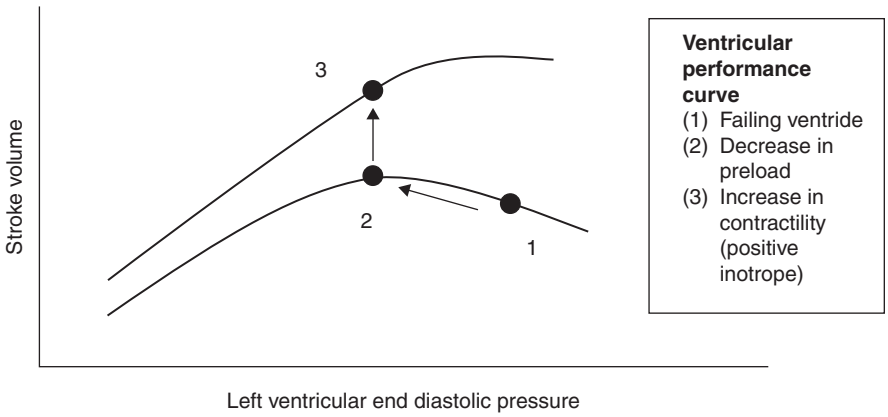

Fig. 20.1 Ventricular performance is improved by optimising preload, afterload and contractility. In cardiac failure, the ventricle is often on the unfavourable part of the performance curve (see Chapter 10).

pressure for renal perfusion. Overly aggressive diuresis may reduce preload sufficiently to reduce cardiac output if there is not a concomitant reduction in afterload. Fortunately, frusemide has an arterial and venous dilator effect that precedes the diuresis.

Key point

Preload and afterload reduction has a more rapid effect on pulmonary oedema than diuresis

Causes of cardiogenic pulmonary oedema

Myocardial disease	**Myocardial infarction and ischaemia**
	Hypertensive cardiomyopathy
	Dilated cardiomyopathy
	Hypertrophic obstructive cardiomyopathy
Endocardial disease	**Valvular heart disease**
	Endocarditis
	Atrial myxoma
Pericardial disease	**Constrictive pericarditis**
	Pericardial tamponade
Rhythm disturbances	**Tachy- and bradydysrhythmias**

Myocardial diseases, especially ischaemia and hypertrophy (due to hypertension), are by far the most common.

Clinical presentation

Breathlessness is invariably present, often accompanied by much distress. There may be cough and, in severe cases, frothy blood-stained sputum. Examination reveals a distressed, tachypnoeic and tachycardic patient, pale, sweaty and perhaps cyanosed.

Fine crackles are usually heard at the lung bases, sometimes with an associated wheeze. Blood pressure is often elevated, and cardiac examination may reveal a third heart sound. There may be specific features present relating to the cause of the cardiac failure, e.g. signs of valvular heart disease and perhaps symptoms and signs of chronic heart failure (orthopnoea, paroxysmal nocturnal dyspnoea, ankle swelling).

Recognition of an episode of pulmonary oedema is not always easy. None of the symptoms or signs are specific. Fortunately, the generic approach to the breathless patient remains valid and the history and investigations in the secondary assessment will often reveal whether pulmonary oedema is the cause of breathlessness and if so, the cause of pulmonary oedema.

Management of acute pulmonary oedema

Primary assessment and resuscitation

Give high concentrations of inspired oxygen by face mask. Sit the patient upright, unless severely obtunded. Consider using CPAP by face mask at 5–10 cm H_2O, as it reduces preload, afterload and increases alveolar pressures.

Assess heart rate, rhythm, blood pressure and perfusion. Obtain venous access. Treat severe brady or tachydysrhythmias as appropriate (see Chapter 10).

Drug therapy options

Intravenous diamorphine 2.5 mg is an excellent drug which reduces afterload, heart rate, distress and sympathetic overactivity: hypotension is a common side effect.

If the systolic BP>90 mm Hg give either GTN 200–400 μg sublingually to reduce preload and afterload or an intravenous GTN infusion, titrated up to 2 μg/kg/min (approximately 1–10 mg/h), may be added.

Alternatively, furosemide, 20–40 mg IV (more if on large doses already) may be given. This initially reduces preload through venodilation, then causes a diuresis.

If the systolic BP <90 mm Hg and the patient has pulmonary oedema and hypotension then outlook is poor unless a treatable cause is found. Positive inotropic support may 'buy time' until definitive treatment is established:

- Dobutamine may be used at 1–20 μg/kg/min. This inodilator increases heart rate and contractility and also reduces afterload by vasodilatation. Cardiac output increases, but blood pressure may either rise or fall and needs to be monitored carefully during therapy. Unfortunately, the tachycardia induced by Dobutamine may limit therapy.
- Dopamine 1–10 μg/kg/min. increases contractility, heart rate and blood pressure even in the low doses previously regarded as 'renal'. It is also a mild diuretic through Dopamine1 receptors in the renal tubules, but the renal vascular dilatation produced by dopamine in healthy subjects does not appear to improve outcome in renal failure in critically ill patients. It has the advantage over dobutamine that a rise in blood pressure is more reliably produced, but in higher doses it induces severe vasoconstriction, which is not beneficial to the failing heart.

Other inotropic agents are available and, as in many areas of medical practice, have produced conflicting results about outcomes in studies. There may be local protocols for their use. Noradrenaline is generally reserved for patients with profound hypotension (e.g. systolic BP <60 mm Hg).

Secondary assessment and emergency management

- A detailed history and examination should confirm the presence of pulmonary oedema and give a differential diagnosis for its cause.
- Investigations should always include 12-lead ECG, CXR, urea, electrolytes and full blood count.
- Elevated blood levels of brain natriuretic peptide shows promise as a relatively specific indicator of the presence of cardiogenic pulmonary oedema.
- More specific, investigations may be warranted, e.g. urgent echocardiogram if acute valvular dysfunction is suspected. An early echocardiogram is indicated in most cases of suspected cardiogenic pulmonary oedema.
- Thromboprophylaxis with low dose heparin is usually indicated.
- Emergency management may now be directed towards the underlying diagnosis. Thrombolysis angioplasty or urgent revascularisation may be indicated in myocardial infarction/ischaemia.
- Cardiac surgery may be indicated in acute valvular dysfunction. A senior opinion is always indicated if the management plan is not clear.

Reassessment

If the patient does not respond to the initial treatment, the following should be considered:

- Is the diagnosis correct?
- CPAP via face mask
- Optimise afterload reduction
- Inotropic support
- Monitor continuous pulse oximetry and ECG and hourly respiratory rate, pulse, blood pressure and urine output are always indicated. A central venous line to optimise inotropic delivery, intra-arterial monitoring (in shock or in aggressive vasodilatation therapy), cardiac output monitoring may all be useful.
- Intensive care opinion if there is ongoing hypoxaemia, shock or multiple organ failures.
- Urgent haemodialysis if there is fluid overload and no response to diuresis once shock has been corrected.
- Consider mechanical cardiac support, such as intra-aortic balloon pumping (IABP). This may 'buy time' if a treatable underlying cause of pulmonary oedema is present. Insertion (by an appropriately experienced practitioner) of an intra-aortic balloon pump may be a life-saving intervention in patients awaiting emergency surgery for acute mitral regurgitation secondary to papillary muscle rupture or those with ventricular septal defect as a complication of myocardial infarction. IABP reduces afterload and thereby reduces the severity of mitral regurgitation. It enhances forward cardiac output, reduces left atrial pressure and improves pulmonary oedema.

Definitive care

Placement will usually be dictated by the nature of the ongoing therapy and the extent and complexity of monitoring.

In addition to the specific therapeutic options mentioned above, consider long-term therapy after the acute event has been treated. This may include angiography, surgery, ACE inhibitors, β blockers, lifestyle changes and weight loss overseen by a specialist.

Causes of cardiogenic pulmonary oedema

- Left ventricular failure (see Chapters 8 and 9 for further details)
- Valvular heart disease
- Mitral stenosis

Pathophysiology

Chronic rheumatic heart disease is by far the most common cause. The mitral valve cusps are thickened and often fused, with associated thrombus on the atrial surface.

Calcification may also occur. The left atrium is characteristically enlarged and mural thrombus may be present proximal to the posterior mitral valve cusp.

Mitral stenosis reduces left ventricular filling. Consequently, cardiac output falls and pulmonary vascular resistance increases. Left ventricular cavity size usually remains normal. In contrast, the left atrium enlarges and chronic left atrial hypertension induces a rise in pulmonary capillary pressure and, hence, pulmonary oedema formation. Reactive pulmonary hypertension, repeated pulmonary emboli, frequent chest infections or even haemosiderosis, may occur.

Treatment

Pulmonary oedema associated with mitral stenosis responds well to diuretic therapy.

Heart rate and rhythm control is particularly important: tachycardia is not well tolerated. If the patient is in atrial fibrillation with a rapid ventricular response, then appropriate treatment is with digoxin. In addition, low molecular weight heparin should be started as a prelude to either cardioversion or formal anticoagulation, because of the high incidence of arterial embolism from left atrial thrombus.

Rarely, left atrial myxomas (present in 2 per 100,000 of the population) may present as progressive breathlessness, orthopnoea, paroxysmal nocturnal dyspnoea or fluid retention. The acute management is described under mitral stenosis. As there is a significant risk of emboli, surgery is the definitive treatment.

Mitral regurgitation

Pathophysiology

Of the many causes of mitral regurgitation (Table 20.2), the most common is the floppy mitral valve. Irrespective of the cause, however, the main physiological disturbance is an increase in left ventricular output. The pressure within the aorta is significantly greater than that in the left atrium, so the majority of the left ventricular ejection fraction enters the left atrium. The left ventricular output is maintained, however, by a sinus tachycardia. If severe, mitral regurgitation can lead to pulmonary oedema and/or a low output state. Atrial fibrillation is common and, when present, the risk of thromboembolism is high.

During diastole there is a large flow of blood from the left atrium to the left ventricle, comprising blood received from the pulmonary circulation combined with that regurgitated during the preceding systole. This increased volume will lead to left ventricular failure, raised pulmonary capillary pressures and hence pulmonary venous hypertension.

Treatment

Medical treatment does not differ from that described for mitral stenosis. Vasodilatation to reduce afterload is also helpful, especially in acute mitral regurgitation. The different mitral valve operations used currently for correction of mitral regurgitation are mitral valve repair (MVR) with preservation of part or all of the mitral apparatus, and MVR with removal of the mitral apparatus. Improved postoperative function occurs with repair.

Aortic stenosis

Pathophysiology

The causes of aortic stenosis are listed in the box.

Table 20.2 The causes of mitral regurgitation

Structure affected	Pathogenesis
Valve cusps	Floppy mitral valve, infective endocarditis, rheumatic heart disease
Chordae	Floppy mitral valve, connective tissue diseases, infective endocarditis
Papillary muscle	Acute myocardial infarction, cardiomyopathy
Valve ring	Left ventricular dilatation

Causes of aortic stenosis

Congenital bicuspid (fused commissure)
Rheumatic heart disease
Calcified 'senile' valve
Infective endocarditis

Aortic stenosis gives rise to left ventricular hypertrophy. This produces diastolic stiffness of the myocardium, higher end diastolic pressures and, eventually, pulmonary oedema. As the disease progresses, the left ventricular cavity becomes dilated, especially in severe cases.

Treatment

Aortic stenosis is a mechanical problem that will, in most cases, require surgical intervention. Acute pulmonary oedema, in this context, can be managed by diuretic therapy and bed rest before surgery. This, however, is only a temporary measure. Indications for surgery are:

- any symptoms attributable to aortic stenosis
- left ventricular failure
- critical stenosis (peak systolic gradient >50 mm Hg).

Aortic regurgitation

Pathophysiology

The causes of aortic regurgitation are listed in the box.

Causes of aortic regurgitation

Infective endocarditis
Rheumatic heart disease
Trauma
Rheumatoid disease
Marfan's syndrome
Dissecting aneurysm
Syphilis
Ankylosing spondylitis

Aortic regurgitation is associated with an increase in left ventricular stroke volume. The regurgitant flow is greatest in early diastole, when the difference in pressure between the aorta and left ventricle is maximal. The volume of regurgitated blood is determined not only by the severity of the aortic valve disease, but also by the compliance of left ventricle and systemic vascular resistance. The left ventricular output may be more than double.

The end diastolic pressure in the aorta is low and the resistance to ejection of blood by the left ventricle is reduced. This reduction in resistance, allied to a large stroke volume, is responsible for the rapid upstroke and wide pulse pressure.

In acute aortic regurgitation the only physical sign may be the early blowing diastolic murmur.

Treatment

Acute aortic regurgitation is a surgical emergency. It is nearly always secondary to infective endocarditis in the presence of acute pulmonary oedema. Vasodilatation, as with acute mitral regurgitation, is the treatment of choice, whilst plans are being made for emergency aortic valve replacement.

Key point

All patients must receive appropriate advice and treatment, where relevant, for infective endocarditis – irrespective of the valvular problem

Acute hypertension

Pathophysiology

Increased left ventricular load, possibly augmented by increased sympathetic nerve activity, is responsible for left ventricular hypertrophy. The consequent increase in muscle mass may be responsible for the development of ischaemia and also ventricular dysfunction, both predisposing to left ventricular failure.

Treatment

Patients with malignant hypertension usually are admitted to a critical care unit for continuous cardiac monitoring and frequent assessment of neurologic status and urine output. An intravenous line is started for fluids and medications. Patients typically have altered blood pressure autoregulation, and overzealous reduction of blood pressure to reference range levels may result in organ hypoperfusion. The initial goal of therapy is to reduce the mean arterial pressure by approximately 25% over the first 24–48 h. An intra-arterial line is helpful for continuous titration of blood pressure.

Sodium and volume depletion may be severe, and volume expansion with isotonic sodium chloride solution must be considered.

Sodium nitroprusside is the agent of choice, as its action can be immediately reversed by stopping the infusion (50 mg of sodium nitroprusside added to 500 ml of 5% dextrose gives a solution of 100 μg/ml). Intra-arterial pressure monitoring is necessary. An infusion of sodium nitroprusside at 10 μg/min (6 ml/h) should be started with increments of 10 μg/min every 5 min, until a maximum dose of 75 μg/kg is reached.

Right ventricular failure

Pathophysiology

The commonest cause of right ventricular failure is inferior myocardial infarction. Failure of the ventricle to contract appropriately reduces forward flow into the pulmonary circulation and hence manifests as low output left ventricular failure. This may be the first clue to the underlying diagnosis. Further signs include tachycardia, hypotension, and a third heart sound. However, there is no pulmonary oedema. Right ventricular dilatation often produces tricuspid regurgitation with a systolic murmur and regurgitant V waves in the JVP. Features of systemic venous hypertension predominate. This clinical picture may initially be confused with a pericardial effusion or constrictive pericarditis, but Kussmaul's sign is negative and there is no pulsus paradoxus.

Treatment

This comprises a fluid challenge to increase the right ventricular filling pressure. Often, inotropes have to be added. Under ideal circumstances, these patients should be monitored on the coronary care unit and their treatment facilitated by readings from a pulmonary arterial flotation catheter.

Key point

Cardiac tamponade and constrictive pericarditis are rare

Summary
Cardiac failure is a common medical emergency. Knowledge of the pathophysiology of the conditions producing heart failure help apply the structured approach to the management of this condition. The underlying cause is usually ischaemic heart disease. A critical feature in the management of these patients is blood pressure. This will dictate whether vasodilatation and/or inotropic support is the management of choice.

BRAIN FAILURE

Introduction

Acute brain failure may be manifested as a decrease in consciousness, pupillary abnormalities or abnormal movements and posture, as described in Chapter 7. However this section will examine the more subtle types of brain failure manifested by intellectual dysfunction, loss of intelligence or loss of intellectual capacity. This condition must be differentiated from learning difficulties, where there is a subnormal intellectual capacity from the outset that is often caused by brain disease acquired during prenatal or early life.

In this context brain failure is not a medical emergency, but it is considered because the differential diagnosis often causes concern (see box).

Differential diagnosis of brain failure

Dementia	
Pseudodementia	**Acute confusional state**
	Inattention
	Depression

All of these conditions will affect the mental state but, in the context of acute medicine, the important diagnosis to establish is that of an acute confusional state. This is the commonest condition that affects the mental state and the commonest form of pseudodementia.

Intellectual functions are best examined using a mental state examination, as described in Chapter 7. In both acute confusion and dementia, there is a **global** impairment in the mental state. However, there are some specific features which will help to distinguish one from the other.

Acute confusional state

In a patient who is acutely confused, the abnormality in mental state is due to reduced cerebral function, commonly secondary to a toxin, hypoxaemia or ischaemia; i.e. the patient has an encephalopathy. Diagnostic clues are the acute onset, a decrease in conscious level and an underlying disease process or exogenous toxin that has precipitated the acute confusion.

Key point

Acute confusional state is associated with a subtly decreased conscious level

The confused patient is unable to maintain a coherent stream of thought or action. This is best assessed by tests of mental attention such as the 'serial sevens'

or '1 tap/ 2 tap' test. Explain to the patient that if you tap once the patient should respond by tapping twice; however, if you tap twice the patient should not tap. A similar test is to ask the patient to recite rapidly all the letters of the alphabet that rhyme with tree.

This ensures that they have to keep the task in mind whilst reciting the appropriate letters.

Dementia

Dementia is characterised by a chronic and often progressive onset of global intellectual dysfunction. Memory impairment is the most commonly found abnormality of cognitive function.

Key point

Do not focus on 'overlearned' knowledge, such as details of the family, as even the most demented patient may still be able to recollect some relevant details

The mini mental state examination is a 30 point score that is easily assessed (see Chapter 7). Search for **Global** impairment of the components (orientation, recent and remote memory, calculation, language). Scattered abnormal results should not be overinterpreted, as mistakes are common. Normal results in these tests are very useful in excluding a diagnosis of dementia.

Dementia is a chronic disabling disease and, therefore, before a firm diagnosis is made it is important not to miss a treatable condition. A full history and physical examination is performed and help should be sought from either a neurologist or a geriatrician.

Some causes of dementia

Common

Alzheimer's disease
Cerebrovascular disease
Alcoholism
Parkinson's disease

Uncommon

Vitamin B_1, B_{12} deficiency
Hypothyroidism
Syphilis
HIV infection
Subdural haematoma
Cerebral tumour
Hydrocephalus
Heavy metal poisoning
Vasculitis
Sarcoidosis
SLE
Huntington's chorea
Multiple sclerosis

A computed tomography scan is usually necessary, as are some laboratory tests, but a battery of screening tests for rare diseases is unrewarding, especially for those that have other clinical manifestations detectable on examination.

Summary

Brain failure commonly presents as an acute confusional state. The differential diagnosis includes dementia, inattention and depression, and is facilitated by a comprehensive medical history and search for an underlying treatable cause. In

the context of acute confusional state, this is usually a toxin, hypoxaemia or ischaemia. In contrast, treatable causes for dementia include meningioma, chronic bilateral subdural haematomata, hydrocephalus and vitamin B_{12} deficiency.

ACUTE RENAL FAILURE/ACUTE KIDNEY INJURY

Introduction

Acute renal dysfunction is common in a sick patient with multiple organ dysfunction and is usually secondary to circulatory failure. As a single organ failure, it is usually due to a nephrotoxin, intrinsic renal disease or post-renal obstruction. Intrinsic renal disease is relatively rare. Approximately 5% of emergency admissions have transient disturbances in renal function.

Definition

The traditional definition of acute renal failure was the abrupt loss of kidney function resulting in the:

- Retention of urea and other nitrogenous waste products
- Dysregulation of electrolytes and extra cellular volume

There have been many problems with this and other definitions. To obviate these problems the term 'acute kidney injury' has been adapted to embrace the spectrum of acute renal failure. The diagnostic criteria are:

- abrupt onset (within 48 h)
- absolute increase in serum creatinine of 26.4 μmol/l above baseline
- an increase of >50% of serum creatinine
- oliguria of less than 0.5 ml/kg/h for more than 6 h.

The SOFA score criteria (see introduction) recognises that acute renal injury is not an all or nothing phenomena.

Causes

Although the causes are divided into three groups (see box) those grouped as prerenal are the commonest. Often, you will suspect that acute kidney injury is likely from either the clinical features or the history. Occasionally, however, it will come to light when laboratory results are examined.

Causes of acute renal failure

Prerenal	**Hypotension, e.g. following shock**
	Hypovolaemia, e.g. gastrointestinal haemorrhage, persistent vomiting or diarrhoea, diuretic or hyperglycaemic states
	Selective renal ischaemia, e.g. hepatorenal syndrome
Intrinsic renal disease	**Glomerular, e.g. primary part of a systemic disease**
	Vascular, e.g. vasculitis, coagulopathy
	Tubular, e.g. acute tubular necrosis (often ischaemic)
	Interstitial, e.g. drug related acute interstitial nephritis
Postrenal	**Urethral obstruction, e.g. prostatic pathology**
	Ureteric obstruction, e.g. carcinoma of the bladder

Primary assessment and resuscitation

The priority in acute kidney injury is to treat life-threatening abnormalities in airway, breathing and circulation. Hypotension, shock and hypovolaemia must

be aggressively corrected. Fluid replacement is usually indicated if pulmonary oedema is absent. If pulmonary oedema is present, it should initially be treated conventionally as detailed above, although diuretics will not usually be effective.

Principles of management of acute kidney injury

- Resuscitate ABC to normal
- Treat life-threatening pulmonary oedema and hyperkalaemia
- Look for and treat other organ failures and infection
- Make a diagnosis of the cause of renal failure and treat accordingly
  - Exclude renal tract obstruction
  - Exclude all potential renal toxins
  - Examine the urine/urinary sediment
- Consider timing of renal replacement therapy if indicated

Secondary assessment and emergency treatment

A full history should consider potential nephrotoxins (see the next box). Clinical examination should always include rectal and pelvic examinations to search for a pelvic tumour.

Common nephrotoxins

- Non steroidal anti-inflammatory drugs
- ACE inhibitors angiotensin receptor antagonists
- Radiocontrast media
- Amphotericin
- Aminoglycosides

The presenting features of renal disease are summarised in the next box. A urinary catheter must be inserted. Multiple organ dysfunction should be sought and managed appropriately.

Symptoms and signs of renal disease presentation

Altered function	Decreased/no urine output
	Flank pain
	Hypertension
	Discoloured urine
Failure	Weakness, lethargy
	Anorexia
	Vomiting
	Oedema
	Confusion, fits
Concurrent disease	Fever
	Arthralgia
	Breathless
Incidental findings (asymptomatic)	Abnormal creatinine
	Urine analysis
	Renal scan

When acute renal injury is suspected, some urgent investigations are required (see the next box).

Urgent investigations in suspected acute renal failure

Plasma sodium, potassium, urea, creatinine and glucose
Urine stick test, microscopy, biochemistry and culture
Arterial blood gases
ECG
Renal ultrasound scan

Hyperkalaemia is a life-threatening emergency and should be carefully managed (see next box).

Emergency Management of hyperkalaemia

Ensure correct fluid balance. Treat or remove the underlying cause. Then consider:

Check K^+ and other electrolytes (1–4 hourly) and monitor ECG:

a **$K^+ > 6.0$ – 100 ml 50% glucose solution with 10 units regular (rapid-acting) insulin IVI**
b **$K^+ > 6.5$ – as for (a), but infuse calcium gluconate, 10 ml 10% solution over 10–15 min first (stabilises cardiac cell membranes) until ECG changes revert to normal**
c **$K^+ > 7.0$ – as for (a) and (b) above, and infuse 50–100 ml 8.5% sodium bicarbonate over 30 min (unless patient in respiratory failure). Repeat glucose and insulin 2–4 hourly prn**
d **Rectal calcium resonium 30 mg once daily to decrease K^+ on a chronic basis**
e **If response to above is inadequate, consider nebulised salbutamol furosemide and haemodialysis.**

Urine analysis is useful in the differential diagnosis of acute renal injury. Urine biochemistry may be helpful, but the values in Table 20.3 are of limited use in the presence of pre-existing renal disease, diuretic therapy, liver disease and intrinsic glomerular disease.

With acute renal injury urine sodium measurement can help distinguish hypovolaemia (pre renal) from acute tubular necrosis. However, urine sodium is

Table 20.3 Urine biochemistry in acute renal injury

	Prerenal	Acute tubular necrosis
Urine sodium (mmol/l)	<20	>40

influenced by urine output. The effect of variations in urine volume can be determined by calculating the fractional excretion of sodium (Fe Na):

$$\text{Fe Na (\%)} = \frac{\text{Urine Na} \times \text{Plasma creatines} \times 100}{\text{Plasma Na} \times \text{Urine creatines}}$$

A value of below 1% = pre renal disease
Above 2% = acute tubular necrosis
(values below 1 and 2 = either disorder)

Urine stick testing and microscopy is more useful. The presence of dysmorphic red cells, red cell casts and proteinuria on urine microscopy is suggestive of acute glomerulonephritis. In contrast, a positive urine stick test for blood, but negative microscopy for red cells, is indicative of rhabdomyolysis. This may be confirmed by the laboratory detection of urine myoglobin and raised blood creatinine kinase. The presence of tubular cell casts, tubular cells and granular casts is highly suggestive of acute tubular necrosis.

It is vital to exclude urinary obstruction as a cause of acute renal failure. Dilation of the renal collecting system, ureters or bladder on renal ultrasound makes obstruction likely and this investigation should be performed urgently in oliguric acute renal failure. The ultrasound will also be useful to confirm number, site and shape of the kidneys including any cortical thinning and scarring, suggestive of pre-existing disease.

Monitoring is usually dictated by any associated multiple organ dysfunction but, as a minimum, should include:

• hourly	urine output and clinical examination
• 4 hourly	pulse, blood pressure
• twice daily	serum, urea, electrolytes and creatinine, arterial blood gases (more frequently if hyperkalaemia or rhabdomyolysis present)

Definitive management

Once resuscitation is complete and acute life-threatening complications have been managed successfully, the next decision is the timing and necessity for renal replacement therapy and the manner in which it is delivered. Low dose dopamine is not thought to be of benefit, on current evidence. There is little evidence for high dose frusemide (e.g. 250 mg over 1 h), but it is often prescribed.

Indications for early renal replacement therapy in acute renal injury

Hyperkalaemia
Pulmonary oedema
Severe metabolic acidosis
Uraemic encephalopathy or pericarditis

Intermittent haemodialysis is the usual renal replacement therapy used in isolated acute renal injury, often within a specialist medical area. The patient with multiple organ failures, especially haemodynamic instability, may benefit from continuous renal replacement therapy, usually in an intensive care unit. All renal replacement therapy will be under the guidance of a nephrologist and/or intensivist.

Summary

Acute renal injury is usually associated with other failing organs. The priorities are to:

- identify all failing organ systems
- treat life-threatening hyperkalaemia and pulmonary oedema
- recognise and treat the underlying cause
- organise dialysis when appropriate.

LIVER FAILURE

Introduction

The incidence of both acute and acute-on-chronic liver failure is increasing. The patient with acute on chronic liver failure is a frequent medical emergency. The immediate management of these two conditions is virtually identical.

Definition

Liver failure is a syndrome that follows severe impairment of hepatocyte function, hence it is also referred to as hepatocellular failure.

Clinical features

These usually develop over several days, but coma onset may occur over hours.

Cardinal signs of acute hepatocellular dysfunction

Jaundice
Hepatic encephalopathy
Ascites (particularly acute on chronic liver failure)
Coagulopathy

Jaundice indicates impaired release of conjugated bilirubin and its intensity is proportional to the extent and duration of hepatocellular necrosis.

Hepatic encephalopathy is manifested as a broad spectrum of neuropsychiatric features that are epitomised by impaired mental state and neuromuscular dysfunction. This form of neurological dysfunction occurs when blood is shunted from the portal venous system into the systemic circulation without hepatic extraction of substances such as ammonia, phenols and GABA (γ-aminobutyric acid)–like glycoprotein. These compounds are believed to act as inhibitory neurotransmitters, depressing both motor function and the conscious level. This may easily be assessed using the Glasgow Coma Score, but hepatologists in particular prefer to use Childs grading.

Childs grading of hepatic encephalopathy

Grade 1	**Prodromal phase – euphoria or irritability**
Grade 2	**Impending coma – drowsiness, lethargy and confusion interspersed with agitated or aggressive behaviour**
Grade 3	**Stupor – somnolent but rousable**
Grade 4	**Coma**

Patients with encephalopathy can present with a variety of neurological signs ranging from flexor, equivocal or extensor plantars (positive Babinski response)

to extrapyramidal features. The classic sign is asterixis; a non-specific 'flapping' tremor associated with liver failure, carbon dioxide retention and uraemia. This is due, in part, to neuromuscular incoordination of the wrist flexors and extensors.

Differential diagnosis of hepatic encephalopathy: three 'H's *and* four 'I's

Hypoxaemia
Hypovolaemia
Hypoglycaemia
Alcohol
Neurodegenerative conditions
Drugs
Infection
Impaction of faeces
Intracranial haemorrhage
Imbalance of electrolytes

Ascites occur primarily due to a raised portal venous pressure, secondary to distortion and destruction of the sinusoids with superadded impaired venous drainage. It is uncommon as a presenting feature in acute liver failure, but common in acute on chronic.

Coagulopathy associated with liver failure is multifactorial. Bleeding tendency is primarily due to impaired synthesis of all coagulation factors (factor VIII is predominantly produced by the endothelium). This is often exacerbated by thrombocytopenia (e.g. secondary to hypersplenism) or platelet dysfunction. Therefore, it is advisable to check both the prothrombin and activated partial thromboplastin times. Occasionally, disseminated intravascular coagulation is present, but this is rarely severe and, if present, often has another cause, e.g. sepsis. Check a blood film and D-dimer or fibrin degradation products.

In addition, two other features worth mentioning are foetor hepaticus and immunocompromise. Foetor hepaticus is a characteristic smell of the patient's breath which is due to sulphur compounds. All patients with liver failure are relatively immunocompromised and severe infection may be present without coexistent pyrexia or leucocytosis.

Critical clinical features

Life-threatening features of acute liver failure

Hypoxaemia	Gastrointestinal haemorrhage
Hypovolaemia	Coma
Hypoglycaemia	Multiple organ failure

- ***Hypoxaemia:*** This is multifactorial in origin and is primarily related to widespread peripheral pulmonary vasodilatation. This results in approximately two thirds of patients becoming hypoxaemic, but the precise cause remains unknown. It is exacerbated by abnormalities in ventilation, perfusion and transfer factor. The hypoxaemia is usually readily reversible with high concentrations of inspired oxygen.
- ***Hypotension:*** This is a manifestation of systemic vasodilatation combined with a hyperdynamic circulation. Patients therefore exhibit a bounding pulse,

prominent left ventricular impulse and a flow murmur. Of interest is the fact that, whilst the systemic blood flow is increased, renal perfusion is reduced along with urine output. Hypotension associated with liver disease is therefore a combination of systemic vasodilatation and hypovolaemia. The situation can be compounded by the fact that patients can have a coexistent dysrhythmia

Key point

Fifty per cent of patients with acute liver disease will have coexistent gastrointestinal haemorrhage

- ***Hypoglycaemia:*** This is extremely important and easy to miss. Hepatic glucose synthesis and release is impaired and this process is exacerbated by raised levels of circulating insulin.

It is important to be aware of the potential for acute hypoglycaemia. Failure to prevent and recognise this condition can lead to irreversible brain damage – unlike the situation with hepatic encephalopathy.

Key point

Hypoglycaemia is common in patients with liver dysfunction and may mimic hepatic encephalopathy

Other key features include:

- ***Cerebral oedema*** is attributed to arterial vasodilatation and failure of cellular osmoregulation with reduction in cerebral oxygen consumption. The crucial factor is how to distinguish cerebral oedema from hepatic encephalopathy. Often this is impossible. However, in patients with grade 4 coma, both cerebral oedema and hepatic encephalopathy coexist. Thus, early discussion with a hepatologist and intensivist is required.
- ***Renal failure*** is very common in patients with liver failure, but only a minority are associated with true hypovolaemia. Most patients have a 'functional' renal failure.
- ***Impaired water clearance***, sodium pump failure, intravenous fluids and diuretics can give rise to hyponatraemia. These may also contribute to hypokalaemia and the coexistent metabolic alkalosis. Other acid–base disturbances include centrally driven respiratory alkalosis associated with hypovolaemia and metabolic acidosis due to anaerobic metabolism from lactate accumulation and tissue destruction.

Key point

In patients with liver failure, remember that urea and creatinine are not reliable indicators of renal function as hepatic synthesis of urea is reduced and tubular excretion of creatinine is increased

Management of liver failure

Primary assessment and resuscitation

The priority is to treat the hypoxaemia, hypovolaemia and hypoglycaemia characteristic of the condition. These are all treated in a conventional manner as detailed

elsewhere in this manual. The presence of gastrointestinal haemorrhage must always be suspected and blood cross-matched urgently. A coagulation screen is requested urgently and this will guide blood product replacement if haemorrhage is present. Coma may require emergency airway management and, once hypoglycaemia is excluded, oral lactulose is started.

Management of liver failure

Universal precautions
High concentrations of inspired O_2
Secure IV access and treat hypovolaemia
Recognise and proactively treat the potential for:
- **Hypoglycaemia**
- **Hepatic encephalopathy**
- **Thiamine deficiency**
- **Underlying infection**

Secondary assessment and emergency management

This part of the assessment focuses on establishing the cause of hepatic failure and detecting treatable sequelae. A full history should include travel and contact with infectious diseases, drug ingestion (recreational and therapeutic, particularly ecstasy, alcohol and paracetamol) and a sexual history. The presence of encephalopathy may make taking a history difficult.

Urgent investigations in liver failure

Full blood count	**Liver enzyme profile**
Clotting times	**Viral serology**
Urea and electrolytes	**Serum/Urine for toxicology**
Glucose	**Urine – Microscopy/Culture**
Arterial blood gases	**Ascites – Microscopy/Culture**
Blood cultures	**Protein/Amylase**
Paracetamol level	**Cytology**
	Cell count

If gastrointestinal haemorrhage is present, this is assumed to be variceal until proven otherwise, particularly in acute on chronic liver failure. The mortality from variceal haemorrhage in these circumstances is around 30–50%, illustrating the need for early expert advice. Shock is treated conventionally, octreotide or vasopressin analogue commenced according to local protocol, and an urgent endoscopy arranged. If variceal bleeding cannot be controlled with endoscopy, a Sengstaken or Minnesota tube can be passed.

Definitive care

Early liaison with a specialist hepatology unit will be guided by the local gastroenterologist. The important issues in this final stage of management are listed in the box.

Important issues in the definitive care of liver failure

Treatment of vasodilatation and increasing oxygen uptake
Prevention or treatment of cerebral oedema
Treatment of coexistent renal failure with haemofiltration
Temporary hepatic support versus emergency transplant

Outcome measures

Many features will dictate the outcome.

- The shorter the interval between onset of jaundice and hepatic encephalopathy, the better the outcome.
- Severe hepatic encephalopathy is associated with a poor prognosis.

Summary

Irrespective of the cause of either acute or acute on chronic liver failure, the initial management is the same. Treat hypoxaemia, hypovolaemia and hypoglycaemia (the three Hs). Early liaison with a gastroenterologist/hepatologist is necessary.

ENDOCRINE FAILURE

Introduction

Of all conditions that are associated with endocrine failure, the two that cause most concern are related to hyperglycaemia and adrenocortical insufficiency. The latter may be related to either a primary adrenal problem or secondary to pituitary pathology.

Hyperglycaemic states: diabetic ketoacidosis

Clinical features of diabetic ketoacidosis

Hyperventilation
Dehydration and shock
Decreased conscious level
Smell of ketones on breath

Primary assessment and resuscitation

The structured approach will ensure that the patient is receiving oxygen and appropriate vigorous fluid therapy, especially if there are signs of either dehydration or shock. Coma will mean that the airway needs protecting. An initial high glucometer reading must be confirmed with a formal blood glucose. Plasma or urine testing for ketones is necessary.

Key points

Consider ketoacidosis in any ill diabetic, especially if there is vomiting and/or tachypnoea
Never diagnose primary hyperventilation until diabetic ketoacidosis has been excluded
Always exclude ketoacidosis in patients who are confused, comatosed or have a metabolic acidosis

As soon as an elevated blood glucose is identified, the patient should receive 10 units of intravenous soluble insulin whilst an infusion is being prepared.

Treatment of shock is a priority. Hyperglycaemia and hypovolaemia are treated simultaneously and consequently the serum potassium may fall sharply. Regimes for fluid, insulin and potassium have reduced the morbidity and mortality from this condition; an example is given in the box below. This may slightly differ from your local policy; so note any changes. Thromboprophylaxis is usually indicated.

Insulin infusion	**Fluids**
10 units IV bolus	**Bolus 1 l normal (N.) saline**
6 units per hour	**1st hour 1 l N. saline**
Reduce to 'sliding scale' when glucose <15 mmol/l	**2nd hour 500 ml N. saline**
	3rd hour 500 ml N. saline
Subcutaneous regime when eating and ketones gone	**Then 250 ml/h N. saline until rehydrated**
	Add 10% dextrose 62.5 ml/h (500 ml in 8 h) when glucose < 15 mmol/L
	If severe hypernatraemia (Na>155 mmol/l), use 0.45% saline and monitor hourly
Potassium	
Urgent U & E	
Serum K^+	***KCl added per litre***
<3.5 mmol/l	**40 mmol**
3.5–5.5 mmol/l	**20 mmol**
>5.5 mmol/l	**None**

Secondary assessment and emergency treatment

Monitoring in an appropriate environment is essential to assess the response to treatment and the development of any complications. Shock, coma, glucose level, acidosis and ketosis should all steadily improve. If ketosis persists, it is important to ensure sufficient glucose is being given with insulin, appropriate to the blood glucose.

Monitoring progress in diabetic ketoacidosis

15 min checks: respiratory rate, pulse, blood pressure and Glasgow Coma score
1 hourly checks: glucometer, urine output
2 hourly checks: blood glucose (until less than 20 mmol/l), serum potassium and sodium

This is the minimum monitoring, along with continuous ECG and SpO_2, if the patient does not improve:

- A clinical search for infection is made and blood and urine cultures taken. The skin is examined thoroughly, including the perianal region and perineum, for signs of infection. If infection is suspected, a broad-spectrum antibiotic such as Tazocin is given.

- Other causes of ketoacidosis should be considered, such as non-compliance with insulin, myocardial infarction.
- Sodium bicarbonate use in severe acidosis is controversial, but if pH <6.9, 50 mmol of sodium bicarbonate may be infused and rechecked, targeting a pH >6.9 (rather than normal levels).

Key point

Patients with ketoacidosis can have a neutrophil leucocytosis without any evidence of an infection. Do not treat with antibiotics

Definitive management

The patient with ketoacidosis will usually need either high dependency or intensive care.

The insulin infusion should be continued until either plasma urinary ketones are negative and the venous bicarbonate is normal. Most patients will be tolerating a normal diet by this stage and it is, therefore, safe to convert to subcutaneous insulin. However, the infusion should be continued for approximately 60 min after the first subcutaneous dose. In the newly diagnosed diabetic, start with short acting soluble insulin three times per day before meals. After 24 h, it should be possible to estimate the total daily insulin dose. This should then be given as two thirds of the daily dose before breakfast and the remainder before supper. Each dose should comprise half of soluble insulin and half of intermediate acting insulin. Glucometer readings should be taken before breakfast, lunch and dinner, and the insulin should be adjusted accordingly. Other long-term regimes may be recommended by a diabetologist.

In contrast, known diabetic patients can be restarted on their normal insulin regime; however, they should be monitored in case this has to be amended.

Hyperosmolar non-ketotic hyperglycaemia

Key points

This diagnosis should be considered in any patient with severe hyperglycaemia, dehydration and drowsiness

Hyperosmolar non-ketotic coma is differentiated from diabetic ketoacidosis by:

- **blood glucose greater than 30 mmol/l, but only 1+ or absence of ketonuria**
- **plasma osmolality greater than 350 mosmol/kg**

The patient with hyperosmolar non-ketotic hyperglycaemia is usually elderly, but the management should follow the guidelines for diabetic ketoacidosis with the following exceptions:

- Half normal saline is used for fluid replacement if the plasma sodium is greater than 150 mmol/l. Frequent checks of serum sodium are important to ensure serum sodium does not fall too quickly. Insulin sensitivity is greater in the absence of severe acidosis, therefore, the infusion should be started at 3 ml/h.

- The risk of thromboembolism is high; therefore, the patient should be fully anticoagulated with heparin according to local policy unless there are contraindications.
- Total potassium is low and plasma level is more variable.

Acute adrenal insufficiency

The causes of adrenal insufficiency are listed in the box.

Causes of acute adrenal insufficiency

Rapid withdrawal of corticosteroids after chronic therapy
Sepsis or surgical stress in patients with chronic adrenal dysfunction from:
chronic corticosteroid therapy
autoimmune adrenalitis
Rare causes such as tuberculosis, age related infection with cytomegalovirus and adrenal metastases
Bilateral adrenal haemorrhage (rare) secondary to fulminant meningococcal sepsis or anticoagulant therapy
Sepsis or surgical stress in patients with hypopituitarism
Septic shock may produce 'relative' adrenal insufficiency

Key point

This diagnosis should be considered in any patient with:
unexplained hypotension
mild hyponatraemia
corticosteroid therapy
pigmentation
preceding anorexia, vomiting, diarrhoea and weight loss

Emergency management issues

The patient will be treated appropriately by the structured approach according to their presenting symptoms, especially if they are comatosed, hypotensive or confused. Remember that the patient may be hypoglycaemic. As soon as the diagnosis of acute adrenal insufficiency is suspected, draw blood for a random cortisol and adrenocorticotrophic hormone (ACTH) measurement. If the patient has an impaired conscious level or shock give hydrocortisone 100 mg immediately followed by 100 mg three times per day.

The urgent investigations will not differ from those normally requested in the primary assessment. The results of the cortisol and ACTH estimations above may provide supportive evidence of the clinical diagnosis. A short Synacthen® test should be done to confirm the diagnosis once the patient has improved begin treatment. Change hydrocortisone to an appropriate dose of dexamethasone before testing with using 250 μg of Synacthen® after baseline cortisol levels. The cortisol levels should be repeated after 30 min and then 1 h.

The typical biochemical findings in acute adrenal insufficiency are low sodium (120–130 mmol/l), raised potassium (5–7 mmol/l), raised urea (>6.5 mmol/l) and low glucose.

Intravenous fluid replacement, dextrose and hydrocortisone should continue until the patient is asymptomatic. Maintenance therapy usually comprises hydrocortisone 20 mg in the morning and 10 mg at night. Fludrocortisone is not always necessary and will be co-prescribed according to local policy or if the patient has postural hypotension.

Key point

Patients with acute adrenal insufficiency do not always exhibit classic biochemical features

Summary

Diabetic emergencies are common in medical practice. Consider hyperglycaemia in all patients who are hyperventilating, confused, comatosed or acidotic. Fluid replacement and intravenous insulin are the essential therapy.

Acute adrenal insufficiency should be suspected in any patient who has unexplained hypotension, mild hyponatraemia, corticosteroid therapy, pigmentation or preceding anorexia, nausea, vomiting and weight loss. The mainstay of therapy is to provide adequate inspired oxygenation and fluid replacement while increasing the serum glucose (if required) and providing intravenous hydrocortisone replacement.

Time Out 20.2

List the causes of:

- **(i)** respiratory failure
- **(ii)** cardiac failure
- **(ii)** brain failure
- **(iv)** renal failure
- **(v)** liver failure
- **(vi)** endocrine failure

For each cause list the underlying problems, e.g. hypoxaemia, hypovolaemia.
Note how common problems occur, irrespective of the cause, and how and when these problems will be treated in the initial assessment.

SUMMARY

Organ failure is a common medical emergency. Initial treatment is directed at the manifestations of failure, rather than at the underlying cause.

PART V
Special Circumstances

Acute Medical Emergencies: The Practical Approach, Second Edition
Edited by Advanced Life Support Group
 ISBN: 978-0-727-91854-3

Advanced Life Support Group

CHAPTER 21
The elderly patient

OBJECTIVES

After reading this chapter you will be able to understand:

- why age does not influence the structured approach to patient assessment
- how age influences homeostasis
- how age influences disease pathophysiology
- the special considerations that are needed when assessing and managing an acutely ill elderly patient.

INTRODUCTION

Defining 'elderly' is not easy. Some patients in their 60s are physiologically older than those in their 80s. In general, 'elderly' characteristics become more prevalent after the age of 75 years.

When the elderly become acutely unwell, assessment and treatment will follow the structured approach previously described. However, the elderly differ in a number of ways, which may affect presentation, assessment and treatment.

MULTIPLE CONDITIONS

The prevalence of most diseases increases with increasing age. The elderly, therefore, tend to have multiple conditions. In addition to any acute presenting problem, there are usually other coexisting chronic disorders. These make assessment more difficult and may influence prognosis and management.

Key point

Multiple pathology is the rule in the elderly

NON-SPECIFIC/ATYPICAL PRESENTATION

Illness in the elderly often presents with confusion, falls, immobility or incontinence, rather than the typical pattern seen in a younger population, e.g.:

- pneumonia is equally as likely to present with either confusion or pleuritic pain and breathlessness
- cardiac failure may present with confusion or falls rather than breathlessness.

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The reasons for this are multiple. The physiological and pathological changes associated with ageing produce a reduction in physical and mental reserves. Under normal circumstances, the elderly person is able to function satisfactorily with these limited reserves, remaining mobile, continent and mentally clear. However, with the additional stress of an acute illness these abilities may be overcome. Consequently, confusion, falls, immobility and incontinence are common presenting features.

Acute myocardial infarction, pleurisy or acute abdominal emergencies may not present with pain in the elderly. Possible reasons for this include:

- reduced perception of visceral pain
- multiple pathology – diminished awareness of a symptom amongst a complex of symptoms
- associated mental impairment or communication difficulties.

Key point

Any acute medical problem in the elderly can present with confusion, falls, immobility or incontinence. An acute abdomen or acute myocardial infarction may be painless

POLYPHARMACY AND ALTERED DRUG HANDLING

Adverse drug reactions are an important cause of morbidity, even mortality, in older people.

Key point

Approximately one in ten older patients will experience an adverse drug reaction. This may either precipitate their admission to or follow treatment in hospital

Factors underlying adverse drug reactions are complex (see Fig. 21.1). Old people usually have multiple conditions which give more opportunity for prescribing and may lead to polypharmacy.

The elderly may have impaired hearing, eyesight, memory and manual dexterity which can affect their ability to follow a prescribed drug regime (reduced compliance).

Polypharmacy, especially if the drug regime is complex, is also associated with reduced compliance.

A number of changes in pharmacokinetics and pharmacodynamics occur with increasing age:

- Reduced renal clearance – increases the risk of toxicity for water-soluble drugs excreted by the kidney (especially digoxin, gentamicin).
- Reduced hepatic clearance – reduces first-pass metabolism for certain drugs, increasing the likelihood of adverse effects (important for propranolol and morphine). Reduced activity of hepatic mixed-function oxidase causes a decline in clearance of diazepam and chlordiazepoxide.

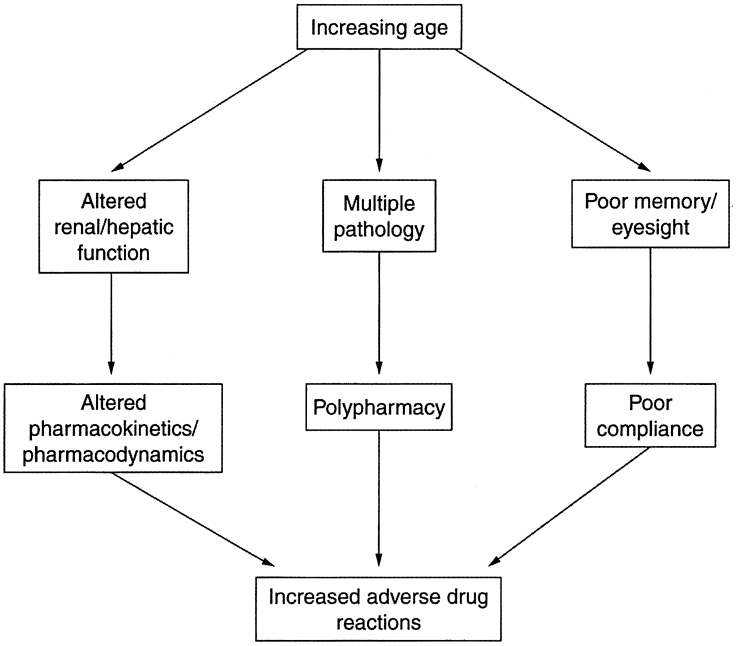

Fig. 21.1 Factors underlying adverse drug reactions in the elderly.

- Altered body composition – there is a relative increase in body fat and a reduction in body water (associated with a reduction in lean body mass) with increasing age. This results in a reduced volume of distribution for water-soluble drugs and an increased volume of distribution for lipid soluble drugs. Therefore, following a given dose of digoxin (based on body weight), a higher serum level is achieved in the elderly compared with the young. The increased volume of distribution for lipid soluble drugs (diazepam, chlordiazepoxide, thiopental) increases the amount of drug bound in body fat and increases the half-life of these drugs.
- Reduced protein levels – may be important with some drugs which bind to albumin.
- Increased sensitivity – has been demonstrated with opiates and benzodiazepines. The elderly require lower doses of warfarin to achieve anticoagulation, without any demonstrable changes in warfarin pharmacokinetics.

Some drugs which are commonly associated with adverse effects in the elderly include the following.

Digoxin

There is an age-related decline in renal function. Therefore, the body is less able to excrete digoxin and hence levels may rise. The levels required to produce both a therapeutic effect and toxicity are very close. Thus, there is a significant risk of toxicity with increasing age and any intercurrent illness.

The adverse effects of digoxin are multiple and may be life-threatening, e.g.:

• cardiac	arrhythmias
• gastrointestinal	anorexia
	nausea/vomiting
	diarrhoea
• neurological	confusion/agitation
	visual disturbance

Key point

Be suspicious of digoxin toxicity in any patient on digoxin who develops anorexia, vomiting or confusion

As a general rule, the elderly require a reduced loading dose of digoxin, because of the reduced volume of distribution (0.5 mg compared with 1.0 mg in younger patients). They also require a reduced maintenance dose (125 or 62.5 μg daily, compared with 250 μg in a younger patient), because of the reduced renal clearance. Estimation of serum digoxin levels is helpful.

Diuretics

Diuretics are commonly used in the elderly, often inappropriately (for leg oedema associated with venous stasis). Homeostatic mechanisms are less efficient (see later) than in younger patients. Thus, elderly patients taking diuretics are more prone to dehydration, metabolic disturbances and postural hypotension, especially when there is intercurrent illness.

Antihypertensives

The use of antihypertensives is increasing in the elderly. These drugs are more likely to cause adverse effects, particularly postural hypotension, because of impaired homeostasis.

Non-steroidal anti-inflammatory drugs

Non-steroidal anti-inflammatory drugs are responsible for a quarter to a third of hospital admissions for gastrointestinal bleeding in older people. They can cause renal failure, fluid retention and worsening of heart failure.

Sedatives and hypnotics

Drowsiness and falls are caused by sedatives and hypnotics. Their half-life tends to be prolonged (due to increased volume of distribution and reduced hepatic clearance of diazepam and chlordiazepoxide). The elderly are more sensitive to the effects of sedatives and hypnotics. Withdrawal reactions are common if these drugs are stopped abruptly. An acute confusional state a couple of days after admission may be the first indication that the patient had been taking sleeping tablets (or alcohol).

Antidepressants

Common side effects from tricyclic antidepressants include postural hypotension, confusion, dry mouth and urinary retention in males. The newer selective seretonin reuptake inhibitors antidepressants are less likely to cause side effects.

Major tranquillisers

Phenothiazines and haloperidol commonly cause side effects including drowsiness, unsteadiness, postural hypotension, constipation and drug-induced Parkinsonism.

Treatment for Parkinson's disease

Levodopa preparations are associated with a high prevalence of side effects, mainly postural hypotension, confusion and dyskinesia. Anticholinergic drugs are even more likely to cause confusion.

Key points

An accurate drug history is essential
Always be suspicious that any new problem may be a side effect of an existing medication, rather than an indication for additional drug therapy
The drug regime should be as simple as possible (in terms of the number and the frequency of dosing), to improve compliance and reduce the risk of adverse reactions
When introducing new drugs, start with a low dose and increase slowly. Review regularly. Always question whether a particular drug is needed. If it is, what would be the correct dose?

IMPAIRED HOMEOSTASIS

This is important in a number of ways. Postural hypotension due to impaired blood pressure control is mentioned in Chapter 12. Two other important areas are fluid and electrolyte imbalance and temperature homeostasis.

Fluid and electrolyte imbalance

A number of changes which affect sodium and water homeostasis occur with increasing age, increasing the risk of fluid and electrolyte disturbance. These changes include:

- a reduction in renal blood flow
- loss of nephrons
- impaired ability to:
  excrete an extreme sodium load
  conserve sodium and other electrolytes
  concentrate urine
  excrete a water load
- a decrease in total body water with age (associated with the reduction in lean body mass)
- decline in thirst with age
- reduction in the levels of plasma renin and aldosterone (although no changes in angiotensin II concentrations have been found). This probably contributes to the reduced ability to preserve sodium when necessary.

Fluid and electrolyte imbalance can be exacerbated by increased fluid loss (diarrhoea and/or vomiting) and reduced fluid intake (which is more likely to cause serious metabolic disturbance). Water replacement is less effective because of the reduced sensation of thirst. The elderly have impaired water conservation. In addition, the reduced total body water means the patient starts from a lower baseline and that dehydration occurs more readily.

Temperature homeostasis

Core body temperature is held within a narrow range around 37°C. Heat is generated in most tissues of the body and lost by radiation, convection, conduction and evaporation. The balance between heat production and heat loss is regulated by the hypothalamus.

If the core temperature rises, the hypothalamus is perfused by 'heated' blood and responds by causing cutaneous vasodilatation and sweating. This allows increased heat loss.

In contrast, if the core temperature falls, the hypothalamus increases core heat production (increased muscle tone with shivering) and reduces heat loss from the skin (cutaneous vasoconstriction). In the elderly, these processes are impaired because of:

- a delayed vasoconstrictor response to cold
- a smaller increase in metabolic heat production (probably due to the reduced muscle bulk)
- a decline in the perception of cold, which affects behavioural responses, e.g. wearing extra clothes, seeking shelter.

Key point

Homeostatic mechanisms are less efficient in the elderly. The stress of an acute illness or the effects of drugs are more likely to be associated with postural hypotension, fluid and electrolyte disturbance and disturbances of temperature control

The main clinical manifestation of impaired temperature homeostasis seen in the UK is hypothermia.

HYPOTHERMIA

Introduction

Hypothermia is defined as a core body temperature of 35°C or less. The true prevalence is unclear. Mortality is high, especially in the elderly, with estimates varying from 30 to 75%.

Multiple factors usually contribute to hypothermia. The elderly are at particular risk because of impaired temperature homeostasis. Other important factors include:

- physical – poor mobility, risk of falling
- social – living alone, inadequate heating
- medical conditions – affecting heat production, heat loss or temperature control
- drugs/alcohol – phenothiazines cause vasodilatation and act directly on the temperature control centre in the hypothalamus. Benzodiazepines, antidepressants and opioids act centrally and may increase the risk of falling
- Alcohol will predispose to hypothermia by inhibiting shivering, impairing hepatic gluconeogenesis and inducing peripheral vasodilatation.

The causes of hypothermia are listed in the next box.

Pathophysiology

Mild hypothermia (35–32°C)

The initial response to a fall in temperature is to increase the metabolic rate by shivering and to reduce heat loss by peripheral vasoconstriction. Even at this early stage, psychomotor function can be impaired especially in the elderly manifested by confusion, dysarthria and incoordination.

Causes of hypothermia

Excessive heat loss
- **environmental exposure**
- **increased cutaneous blood flow**

Inadequate heat production
- **malnutrition**
- **hypoglycaemia**
- **hypothyroidism**
- **diabetic ketoacidosis**
- **adrenal insufficiency**
- **hepatic failure**
- **uraemia**

Altered thermoregulation
- **hypothalamic dysfunction**
- **spinal cord injury (T1 or above)**

Drugs
- **(i) Central effects**
  - **alcohol**
  - **phenothiazines**
  - **barbiturates**
  - **opioids**
  - **benzodiazepines**
- **(ii) Peripheral vasodilatation**
  - **alcohol**
  - **phenothiazines**

Moderate hypothermia (32–28°C)

As the core temperature falls below 32°C, cardiac conduction becomes impaired, heart rate falls and cardiac output decreases. Atrial fibrillation with a slow ventricular response is common. In addition, shivering stops and is replaced by hypertonia. Coma may develop.

Severe hypothermia (below 28°C)

There is a high risk of ventricular fibrillation. As the temperature continues to fall, hypotension results and eventually asystole occurs. In severe hypothermia, coma may be associated with a flat electroencephalogram, but this is not indicative of brain death and may be reversible.

In addition to these cardiovascular and neurological effects, hypothermia has other important effects.

Respiratory

Respiratory effects include tachypnoea in the early stages, followed by hypoventilation as hypothermia becomes more severe. Loss of cough and gag reflexes predispose to aspiration pneumonia.

Renal

A 'cold diuresis' occurs, due to increased central blood volume (peripheral vasoconstriction shunting blood from peripheral to central circulation). There may be additional sodium and water losses due to impaired function of epithelial

transport mechanisms in the kidney. The result is severe volume depletion and hypotension.

Gastrointestinal

Hypomotility of the gastrointestinal tract is common. Gastric dilatation may occur, with increased risk of aspiration. Hypothermia may cause acute pancreatitis and acute peptic ulceration with haematemesis.

Haematological

The haemoconcentration associated with the reduced plasma volume may predispose to thrombotic complications. In addition, there may be bleeding problems. Clotting factors work less efficiently at lower temperatures and thrombocytopenia may occur due to sequestration of platelets.

Impaired oxygen delivery

As the temperature falls, the haemoglobin oxygen dissociation curve shifts to the left. Thus oxygen delivery to hypothermic tissues is impaired. However, hypothermia also reduces the tissues oxygen requirements.

Hyperglycaemia

Moderate to severe hypothermia inhibits the action of insulin. This leads to reduced glucose utilisation and hyperglycaemia.

Assessment

Primary assessment and resuscitation

Airway

- Airway may be obstructed due to depressed conscious level.

Breathing

- Respiratory rate is reduced with moderate/severe hypothermia.
- May be evidence of aspiration pneumonia (although slow shallow breathing may make clinical signs difficult to detect).

Circulation

- Check blood pressure. Treat hypotension due to hypovolaemia ('cold diuresis'). Fluid replacement has to be carefully controlled, as the cold myocardium does not tolerate excessive fluid loads.
- Check core temperature with a low reading thermometer or rectal thermocouple probe. Initiate rewarming measures.
- Check pulse for 60 s.
- ECG monitoring (risk of atrial fibrillation, ventricular fibrillation, asystole).

Disability

- Neurological dysfunction may be either the cause or the effect of hypothermia.
- Check glucose.

Rewarming

Rewarming techniques can be active or passive, and active rewarming can be external or internal. Which method is most appropriate will depend on a number of considerations including the degree of hypothermia, the rate of development of hypothermia, the age of the patient and the patient's cardiovascular status. A young person who is hypothermic due to cold exposure will usually tolerate rapid, active, surface rewarming. In contrast, this will lead to circulatory collapse in an elderly patient. An ideal rate of rewarming is 0.5°C/h. In the presence of

cardiac arrest, core temperature must be raised as rapidly as possible. However, if the patient does not have a life-threatening arrhythmia, interventions aimed at rapid rewarming should be used with caution to minimise the risk of precipitating arrhythmias.

Key point

The hypothermic myocardium is very sensitive. Any physical manipulation of the patient (central lines, nasogastric tubes, endotracheal tubes, rapid rewarming techniques) increases the risk of developing ventricular fibrillation. This is resistant to defibrillation until the core temperature has risen to 32°C

Passive rewarming

This uses the patient's own heat production to raise core temperature. Any wet clothing is removed and the patient is dried and then insulated with blankets. It is important to keep the head covered as up to 30% of body heat can be lost from this site. Warm humidified oxygen minimises respiratory heat loss.

Active external rewarming

Immersion in warm water (40°C) can be appropriate in conscious uninjured patients with a core temperature of greater than 30°C, where hypothermia has been of short duration and rapid onset. However, it is inappropriate and impractical for the majority of hypothermic patients. Circulating water blankets, electric blankets, warm air blankets and heating cradles are less efficient than warm water immersion but more practical. There are a number of potential dangers with active external rewarming.

When hypothermia has developed slowly and has been prolonged, there is hypovolaemia due to 'cold diuresis' and profound acidosis in the underperfused peripheral tissues. The vasodilatation caused by external heating may therefore cause hypotension and a metabolic acidosis. Active external rewarming also causes a significant 'afterdrop' in core temperature that can potentially trigger arrhythmias.

Active internal rewarming

Intravenous fluids should be heated to 40°C but their small volume means that they have a minimal effect on core temperature. Inspired humidified air heated to 42°C minimises respiratory heat loss but contributes little to active rewarming. Irrigation of hollow organs (stomach, bladder) and body cavities (pleura, peritoneum) with warm fluid (40°C) can be used in extreme conditions, such as cardiac arrest, but may need to be continued for several hours. Irrigation fluids should be isotonic and potassium free. Haemodialysis and cardiopulmonary bypass can bring about rapid rewarming but they require specialised skills and availability is limited.

Management of arrhythmias

Atrial arrhythmias are common and usually reversible with rewarming alone; specific antiarrhythmic therapy is rarely needed. Ventricular arrhythmias in the hypothermic patient are usually refractory to drugs and defibrillation. Antiarrhythmic drugs should not be used until the body temperature is normal. Below 32°C, defibrillation is unlikely to succeed. Therefore, the initial three cycles of shocks of the ventricular fibrillation algorithm should be given but, if

unsuccessful, further attempts should be withheld until the temperature is greater than 32°C. Repeated defibrillation in the hypothermic patient will simply cause myocardial damage.

It is difficult to distinguish reversible from irreversible hypothermia. Apnoea, asystole and absence of brain activity are usually signs of death but can also be present in severe reversible hypothermia. Patients should continue with cardiopulmonary resuscitation until a deep body temperature of at least 32°C has been achieved or the temperature has failed to rise despite effort. Only then can a definite diagnosis of death be made.

Key point

The patient is not dead until both warm and dead

Secondary assessment

In all hypothermic patients chest X-ray and 12-lead ECG are essential. ECG may show 'J' waves although they have no prognostic significance. Recheck urea and electrolytes, amylase and glucose, together with thyroid function, a drug screen and alcohol estimation. Arterial blood gases will need to be corrected for the low core temperature. Take blood cultures and start empirical broad-spectrum antibiotic therapy, as the usual signs of infection may be masked.

Once rewarming has been initiated and the patient stabilised, reassess for any underlying condition that may have precipitated the hypothermia.

Time Out 21.1

The percentage of elderly hospital admissions associated with drug side effects is:
a 1%
b 4%
c 10%

Key points

Hypothermia is a life-threatening condition
Assessment and treatment follows the structured approach previously described
The rate of rewarming needs to be adjusted according to the clinical situation
Ventricular fibrillation or circulatory collapse can be precipitated by rapid rewarming techniques
The patient is not dead until both warm and dead

SUMMARY

In the elderly patient:
- assessment follows the structured approach
- multiple conditions are common
- disturbances in mobility, mental function and continence are common presentations of many conditions.

Advanced Life Support Group

CHAPTER 22

Transportation of the seriously ill patient

OBJECTIVES

After reading this chapter you will be able to:

- discuss the principles necessary for the safe transfer and retrieval of critically ill patients
- describe the systematic 'ACCEPT' approach for managing such patients.

Key point

Transport is a potential period of instability

INTRODUCTION

There is an increasing need to transfer patients who are medically ill. Historically, patients are often transferred from home to hospital by ambulance and this will continue. However, with changes in the provision of health care more patients are being transferred because of the following:

- reduction in number of, and increased pressure on, hospital beds
- transfer for tertiary care, e.g. neurosurgery, cardiothoracic surgery
- the need for intensive care treatment, either supra-specialist care or because of a local shortage of beds.

Thus, it is common for patients to be transferred between hospitals, because of a bed shortage in one region or throughout the United Kingdom. It is important to remember that the transfer distance is irrelevant to the need for meticulous preparation before transfer. Movement from one ward to another is just as important, and can be associated with just as many problems as a transfer over 500 miles. Commonly reported problems are shown in the next box.

Most commonly reported adverse events

No capnography available (when clinically indicated, with potential for raised intracranial pressure)
Cardiovascular problems during transfer
Tachyarrhythmias
Bradycardias
Hypotension
Hypertension

Continued

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Most commonly reported adverse events (Continued)

Hospital equipment problems
- **Monitor failure**
- **Pump failure**
- **Equipment not available**
- **Mechanical ventilator not available**

Significant hypoxaemia
Ambulance breakdown/lost en route
Cardiac arrest in ambulance
Death during transfer

PRINCIPLES OF SAFE TRANSFER

The aim of a safe transfer policy is to ensure that there is continuing medical treatment for the patient without any detrimental effect. To achieve this, the **right** patient has to be taken at the **right** time, by the **right** people, to the **right** place by the **right** form of transport. This requires a systematic approach that incorporates a high level of planning and preparation before the patient is moved.

The systematic approach to patient transfer

A – assess the situation
C – control the situation
C – communication
E – evaluate the need for transfer
P – package and prepare
T – transportation

By following the ACCEPT approach, appropriate procedures are done in the correct order and are not forgotten. The acronym also emphasises the need for a great deal of preparation before the patient is transported.

ASSESSMENT

The initial clinician involved with patient management does not always accompany the patient during transfer. It is therefore important that the transportation procedure begins with assessing the situation. This is helped by answering several key questions.

Assessment questions

What are the patient's basic details?
What is the problem?
What has been done?
What was the effect?
What is needed now?

The clinician should also determine the lines of responsibility for the patient not only before transfer, but also during any future transportation. In practice,

this responsibility is usually held jointly by the referring consultant clinician, the receiving consultant clinician and the transfer personnel. However, there should be a named person with overall responsibility to organise the transfer.

Conditions requiring transfer

There is an increasing need to transfer patients who require active resuscitation. Some of the more frequent medical conditions needing transfer between hospitals are listed in Table 22.1 (see over).

Potential problems during transfer

From the above list of conditions requiring transfer, one can predict potential problems that may arise – as in any clinical practice. For reference, these will be listed in the following seven boxes.

Potential respiration problems

Airway	**Hypoxaemia**
Breathing	**Hypercarbia**
	Severe bronchospasm
	Acute/chronic respiratory failure
	Respiratory arrest
Circulation	**Cardiac arrest**

Potential circulation problems

	Primary
Airway	**Hypoxaemia**
Breathing	**Hypercarbia**
Circulation	**Myocardial infarction**
	Dysrhythmia
	Cardiac failure (either left, right or biventricular)
	Rupture of papillary or ventricular muscle
	Cardiac tamponade
	Pulmonary embolus
Disability	**Deterioration in Glasgow Coma Score**
	Secondary
	Pain
	Peripheral embolus

Most cardiac patients requiring transfer will receive appropriate analgesia and anticoagulation or thrombolysis before transfer. The two most likely problems during transfer are either dysrhythmia, including cardiac arrest, or cardiac failure. Management of any dysrhythmia should follow the guidelines specified by the European and United Kingdom Resuscitation Councils (see Chapter 6). In contrast, the presence of cardiac failure would require treatment either with venodilators or inotropes (left or biventricular failure) or with a fluid challenge (right ventricular failure).

It is unusual for patients to develop these problems during transfer, as the potential for deterioration should have been recognised. Thus most patients at risk of these complications are sedated, paralysed and ventilated prior to transfer – but this is not always the case.

Table 22.1 Conditions requiring transfer

Respiration (AB)	Respiratory failure (to a respiratory physician, intensivist or *rarely* to an extracorporeal membrane oxygenation unit) • Acute severe asthma, chronic obstructive airways disease • Severe pneumonia • Adult respiratory distress syndrome
Circulation (C)	Critical ischaemic heart disease (to a cardiologist or cardiac surgeon) • Unstable angina, myocardial infarction • Heart failure, cardiogenic shock • Arrhythmia • Valvular or septal rupture Other cardiac disease (to a cardiologist) • Cardiomyopathy, myocarditis, pericarditis Critical vascular insufficiency (to a vascular surgeon) • Aortic aneurysm or dissection • Limb ischaemia
Nervous system (D)	Central nervous system failure (to a neurologist, neurosurgeon, spinal surgeon, stroke unit or psychiatric unit) • Intracerebral haemorrhage or infarction • Intracranial abscess, encephalitis or meningitis • Intracranial tumour or hydrocephalus • Spinal cord compression • Acute psychosis or suicidal behaviour Peripheral nervous system failure (to a neurologist) • Myasthenia gravis • Guillain-Barré syndrome (post-infectious polyneuropathy)
Metabolism/Excretion	Metabolic failure (to a renal, hepatic, gastrointestinal or endocrine unit) • Renal failure • Acute liver failure • Variceal or other gastrointestinal haemorrhage • Diabetic ketoacidosis or lactic acidosis • Thyrotoxicosis or other endocrine/metabolic derangement
Host defense	Infection (to an infectious diseases unit or ICU) • Septic shock including meningococcaemia • Specific infections Immune failure (to a specialist immunological unit, haematologist or oncologist) • Severe allergy or autoimmune process • Immune deficiency or marrow suppression Intoxication (to an ICU or specific poisons unit, e.g. hyperbaric unit) • Poisoning/Overdose • Carbon monoxide poisoning Immersion/Other environmental injury (to an ICU) • Near drowning • Hypothermia

Patients with neurological conditions are commonly transferred to a tertiary referral centre, as most hospitals do not have the relevant facilities. The potential problems encountered are listed in the box.

Potential neurological problems

Airway	Hypoxaemia
Breathing	Hypercarbia
	Respiratory arrest
Circulation	Cardiac arrest
	Hypoglycaemia
	Hyponatraemia
Disability	Deterioration in Glasgow Coma Score
	Fit
	Subarachnoid haemorrhage

Similarly, most hospitals do not have facilities for managing patients with renal or hepatic disease. The relevant potential problems are listed in the next two boxes.

Potential renal problems

Airway	Hypoxaemia
Breathing	Hypoxaemia
Circulation	Hypovolaemia
	Fluid overload
	Hypertension
	Dysrhythmia
	Metabolic acidosis
	Hyperkalaemia
Disability	Deterioration in Glasgow Coma Score
	Fit

Potential hepatic problems

Airway	Hypoxaemia
Breathing	Hypercarbia
Circulation	Hypovolaemia: vasodilation
	Hyponatraemia
	Hypokalaemia
	Hypoglycaemia
	Lactic acidosis
	Dysrhythmia
	Haemorrhage
Disability	Deterioration in Glasgow Coma Score
	Fit

Two other groups of patients that often require transfer are those who have taken an overdose or who are septic and the associated problems are listed in the next two boxes.

Potential problems associated with a septic patient

Airway	Hypoxaemia
Breathing	Pulmonary oedema
Circulation	Hypovolaemia
	Haemorrhage
	Hypoglycaemia
	Disseminated intravascular coagulation
Disability	Reduced Glasgow Coma Score
	Fit

Potential problems associated with overdose

Airway	Hypoxaemia
Breathing	Hypercarbia
Circulation	Hypovolaemia
	Haemorrhage
	Hypokalaemia
	Hypotension
	Dysrhythmia
Disability	Reduced Glasgow Coma Score
	Fit

Although there is a broad spectrum of clinical conditions, there are clearly defined common potential problems, notably hypoxaemia, fluid balance, fitting, changes in Glasgow Coma Score and electrolyte disturbances. Thus, once the patient's condition has been stabilised, there are only a limited number of common complications. These will be prevented or reduced by ongoing treatment and monitoring. There are three major principles:

1 Do no further harm
2 Ensure ABCDE are maintained during transport
3 The most important assessment is the reassessment.

CONTROL

This comprises:

- identify the team leader
- identify the tasks to be done
- allocate tasks.

The person in charge needs to take control of the situation following the primary assessment (Chapter 3).

All immediately life-threatening conditions need to be identified and treated and the patient monitored. The responsible clinician should also decide the most appropriate place for further management, e.g. whether in the resuscitation room or ward, or whether to move the patient to an area in the hospital with greater resources.

A common example of this is moving a patient from the ward to a high dependency unit.

The secondary assessment includes a head-to-toe survey, perusal of the medical notes and formulating a management plan by considering the clinical findings, response to treatment, and the results of any investigations. At the end of this phase, you should know whether transportation will be necessary and if so the ultimate destination.

COMMUNICATION

Moving critically ill patients from one place to another obviously requires the cooperation and involvement of several people. Therefore, key personnel need to be informed when transportation is being considered, as shown in the next box.

Communication

The patient's consultant
Your consultant (if different from above)
The intensive care unit consultant, where appropriate
The patient's relatives
The accepting consultant
Ambulance control
Special transportation controls (when appropriate)

It can be quite time consuming if all communication is delegated to one person. Therefore, delegate the tasks to appropriate people, taking into account their expertise and the local policies. In all cases, it is important that information is passed on clearly and unambiguously. This is particularly true when talking to people over the telephone. A useful tip is to plan what you wish to say before telephoning and use the systematic summary shown in the box.

Communication plan

Who you are
What is needed from the listener
What are the patient's basic details
What is the problem
What has been done
What was the response
What is needed

The second statement is repeated at the end to help summarise the situation and inform the listener what is required. The response to all these points should be documented in the patient's notes. The person in overall charge can then assimilate this information so that a proper evaluation of the patient's requirements for transportation can be made. In doing this, the clinician has to balance the risks involved in transfer against the risks of staying and the potential benefits of treatment from the receiving unit.

EVALUATION

Critically ill patients require transfer because of the need for:
- specialist treatment, such as haemodialysis
- specialist investigations unavailable in the referring hospital
- intensive care or high dependency unit facilities
- a bed.

Having identified the need for transfer, the responsible clinician has to triage the patient, considering their priority in relation to other patients on the intensive care or high dependency unit, the urgency of transfer and the nature of the medical support required. Following acute life-threatening illnesses or injuries, the patient may require urgent transfer after resuscitation, e.g. the movement of a patient with a subarachnoid haemorrhage to the neurosurgical centre. In contrast, patients with organ failure may require less urgent transfer to a tertiary hospital. Under these circumstances, it may be possible to use the transfer team from the specialist centre. On occasions, when empty beds are scarce, it is possible to transfer a less critically ill patient rather than the one currently being dealt with. This obviously requires formal triage of the patients involved and it needs to be done by the consultants in charge of their care.

Triage – categories of clinical urgency

Intensive
Time critical
Ill and unstable
Ill and stable
Unwell
Well

PREPARATION AND PACKAGING

Preparation: principles
stabilise patient
obtain and check all equipment
fully prepare transport personnel

Patient preparation

Key point

Inadequate resuscitation will result in problems during transfer

To avoid complications during any journey, meticulous resuscitation and stabilisation should be done **before** transfer. This may involve procedures requested by the receiving hospital or unit. The transferring team must also ensure that the patient's airway is assessed for patency and protection and appropriate respiratory support is being provided. In many cases, this will mean intubating the patient if it has not been done previously. Blood gases should be taken following this procedure, or after a change in ventilator setting, to make sure that the patient is maintaining an adequate PaO_2 (ideally more than 13 kPa) and $PaCO_2$ of 4.0–4.5 kPa.

Key point

Remember a patient with pulmonary pathology may take up to 15–20 min to stabilise on a new ventilator or ventilator setting

The ventilator obviously needs to be portable. In addition, it must be able to provide the functions the patient requires. This includes variable FiO_2, inspiratory/expiratory ratio, respiratory rate, tidal volume and positive end expiratory pressure. For safety, there should also be a disconnect alarm and an ability to measure airway pressure. Those requiring intubation should be connected to an end tidal carbon dioxide monitor in addition to the basic monitoring equipment required for all critically ill patients.

Basic monitoring equipment

Pulse oximetry
Suction
ECG, defibrillator
Blood pressure – preferably direct intra-arterial monitoring
Thermometer
Urinary catheter
Naso/orogastric tube

Chest drains should be secured and unclamped with any underwater attachment replaced by a Portex drainage bag.

Key point

Before transfer, chest drains need to be inserted prophylactically if the patient has a simple pneumothorax or is at risk of developing one as a result of fractured ribs

Venous access is essential and preferably should be by two large bore cannulae. The patient must receive adequate fluid resuscitation to ensure optimal tissue oxygenation. Preferably, the haematocrit should be over 30%. In some patients inotropic support may also be necessary. Before transfer, invasive central monitoring may have been used to optimise volume replacement. During transfer, however, these lines become unreliable.

Appropriate drugs must be available to maintain the patient's airway, breathing and circulation. This may require infusion pumps (with a backup power source).

A urinary catheter is frequently necessary to monitor urinary output and for patient comfort.

The transfer team should confirm that all equipment is functioning, including battery charge status and oxygen availability, against what is calculated as necessary. The oxygen supply should be sufficient to last the maximum expected duration of the transfer, with a reserve of 1–2 h. There should also be a non-invasive blood pressure device and a self-reinflating bag (such as an Ambu Bag), so that resuscitation can be maintained in the event of either a power or gas failure. A member of the team should also be given the task of ensuring that all the patient's documents are taken.

These include case notes, results of any investigations and the transfer form. All lines and drains should then be secured to the patient and the patient secured to the trolley. The trolley should then be secured to the ambulance and positioned such that all monitors are visible and lines are accessible.

Transport – time/mode

The choice of transport needs to take into account several factors.

Factors involved with transport

Nature of illness
Urgency of transfer
Mobilisation time
Geographical factors
Weather
Traffic conditions
Cost

Road ambulances are by far the most common means used in the United Kingdom. They have a low overall cost, rapid mobilisation time and are less affected by weather conditions. They also give rise to less physiological disturbance. Air transfer is used for journeys over 50 miles or 2 h in duration, or if road access is difficult.

Although this mode of transport is fast, this has to be balanced against the organisational delays and the inter-vehicle transfer at the beginning and end of the journey. Helicopters are used for distances of approximately 50–150 miles and are particularly useful when road or fixed winged air ambulances are not possible. They are, however, often cramped, noisy and uncomfortable. Fixed winged aircraft should ideally be pressurised and are used for transfers of distances greater than 150 miles.

Depending on the geographical location, other forms of transport are used, particularly outside the UK. These include anything from a boat to horseback.

Personnel

In addition to the ambulance crew, a minimum of two attendants should accompany a critically ill patient. One attendant should be an experienced medical practitioner who is competent in resuscitation and organ support. Ideally this doctor should have received training in intensive care and transportation medicine. The Intensive Care Society (ICS) recommend at least 2 years' experience in anaesthesia, intensive care medicine or other equivalent specialties. The clinician must be accompanied by another experienced attendant, who is usually a nurse. This person should be qualified with, ideally, 2 years' intensive care experience. All personnel should be competent in the transfer procedure and familiar with the patient's clinical condition. They should have adequate insurance to cover both death and disability occurring during transfer.

Personal equipment

In addition to the medical equipment described previously, the transfer team needs their own personal equipment.

Personal equipment

P – phone
E – enquiry number and name
R – revenue
S – safe clothing
O – organised route
N – nutrition
A – A–Z
L – lift home

This equipment will ensure that the journey is more comfortable and that a number of contingencies are available should problems occur. The telephone will enable direct communication with both the receiving and home unit. However, they should be given contact names and numbers before leaving. All personnel require appropriate clothing to ensure safety and enough money to enable them to get home should the ambulance be re-diverted to other duties. They also require a planned route and food if a long journey is envisaged.

TRANSPORTATION

Physiological problems during transfer can arise from both the patient's condition as well as the effects of movement. The latter include tipping, vibration, acceleration and deceleration forces, as well as barometric pressure and temperature changes seen with air transport. Adequate preparation can minimise many of these effects.

The standard of care and monitoring before transfer needs to continue. This will include SpO_2, ECG and arterial pressure monitoring. The end tidal carbon dioxide recording needs to be maintained in all patients who are intubated. Non-invasive arterial pressure monitor is sensitive to motion artefacts and, therefore, the intraarterial route is recommended. As mentioned previously, many of the central monitoring devices, such as central venous pressure or pulmonary artery wedge pressure, may be inaccurate due to movement of the ambulance.

The patient should be well covered and kept warm during the transfer. With ground transfer, road speed decisions depend both on clinical urgency and the availability of limited resources, such as oxygen (although the oxygen requirement should have been calculated as outlined above). Ambulance staff should therefore be advised whether a particularly smooth ride is required or a short journey time is important.

With adequate preparation, the transportation phase is usually incident free. Occasionally, untoward events occur; thus, the patient must be reassessed using the structured approach. Appropriate corrective measures should then be taken. This reassessment has to be thorough and cannot be done when faced with excessive vehicular motion. Therefore, in the case of land vehicles, ask the driver to stop at the first available place. Following such events, it is important to communicate with the receiving unit. They can then be adequately prepared and may be able to provide ongoing advice. Again this communication should follow the systematic summary described previously. A continuous record of the patient's condition during the transfer should be made. This can be helped by having monitors with memory functions, which can be accessed later.

At the end of the transfer, the team should make direct contact with the receiving team. A verbal, succinct, systematic summary of the patient can then be provided. This must be accompanied by a written record of the patient's history, vital signs, treatment and significant clinical events during transfer. All the other documents which have been taken with the patient should also be handed over. Whilst this is going on, the rest of the transferring team can help move the patient from the ambulance trolley to the receiving unit's bed. A copy of the transfer sheet should then be handed over, with one copy retained by the transfer team who can then retrieve all their equipment and personnel for the return journey.

The data collection sheets should be subjected to regular audit by a designated consultant in each hospital. This will ensure transfers are appropriate and to the correct standards. Problems can also be addressed and corrected.

Time Out 22.1

A 27-year-old mechanic presented with an occipital headache. A clinical diagnosis of subarachnoid haemorrhage is confirmed by CT scan and lumbar puncture. The local neurosurgical centre is 30 miles away by road. The patient's vital signs are:

A – patent (FiO_2 0.85)
B – rate 14/min – no focal signs
C – sinus tachycardia 110/min BP 120/70
(IV access secured)
D – GCS 15/15; PERLA, no lateralising signs glucose 7.0 mmol/l

Write down an outline of how you, as the doctor in charge, would arrange this patient's transfer to the neurosurgical centre.

SUMMARY

The safe transfer and retrieval of a patient requires a systematic approach. By following the ACCEPT method, important activities can be done at the appropriate time (see the STaR Transfer Master below).

STaR Transfer Master

Assessment	Problem (Sound bite) Action Effect Next	
Control	Team members	Task
	1	Team leader
	2	Look after patient
	3	Communications
	4	Equipment collection
	5	Additional tasks

(*Continued*)

(*Continued*)

Communicate	Who are you What is needed Basic details The problem (Sound bite) Action (what has been done) Effect (is it effective) What is needed (repeated)	
Evaluate	Is the need agreed? Triage = when (how soon) + mode + who (competencies)	
	M I N T	
	M & **N**	**I** = (Equipment)
Preparation and package	PERSONAL **P**hone **E**nquiry number **R**evenue **S**afe clothing **O**rganised route **N**utrition **A** to Z **L**ift home	A B C D E
Transport	min. . .**T**	Handover (CLEAR) **C**ase notes **L**aboratory **E**valuation **A**udit **R**eturn equipment

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CHAPTER 23
The pregnant patient

OBJECTIVES

After reading this chapter you will be able to understand:
- the anatomical and physiological changes that occur during pregnancy
- how these changes may influence your initial assessment.

INTRODUCTION

Acute medical problems in the pregnant patient are rare. For that reason, the physician may feel apprehensive about the management of the acutely ill pregnant patient. This chapter will demonstrate that whilst there are significant physiological changes occurring in virtually every system of the body, pregnancy does not alter the initial assessment of the mother. These anatomical and physiological changes will be described and how these changes may influence the presentation, clinical features and detection of underlying conditions. A thorough understanding of the relationship between the mother and her fetus is essential.

Key point

Optimum treatment of the mother provides the optimum treatment for the fetus

ANATOMICAL AND PHYSIOLOGICAL CHANGES DURING PREGNANCY

These changes will be described and linked to the stages of the initial assessment.

Primary assessment

Airway–

The airway may be difficult to control due to neck obesity, breast enlargement and possible supraglottic oedema. There is an increased risk of aspiration, because of:
- increased incidence of gastro-oesophageal reflux
- delayed gastric emptying
- the pressure on the stomach from the gravid uterus.

Breathing–

Oxygen consumption is increased due to the metabolic demands of pregnancy. There is a physiological tachypnoea. As a result, hypocapnia is common. In particular in late pregnancy, diaphragmatic elevation results in a reduced functional residual capacity. This is offset by an increase in inspiratory capacity.

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Circulation–

The heart rate increases gradually throughout pregnancy by approximately 10–15 beats/min, reaching a maximum in the third trimester. In contrast, the blood pressure falls by 10–15 mm Hg during the second trimester, returning to normal levels preterm.

Supine hypotension can occur as described above aggravated by the gravid uterus causing reduced venous return by compression of the vena cava. By the end of the first trimester, the cardiac output has increased by approximately 1.5 l/min, due to:

- an increase in plasma volume
- decreased vascular resistance of the uteroplacental unit.

Cardiac output may be significantly reduced in the supine position for reasons described earlier.

The effects of pregnancy on the results of initial investigations

The chest X-ray can show diaphragmatic elevation with increased lung markings and prominent pulmonary vasculature.

The ECG may appear unchanged during pregnancy, or the axis can be deviated to the left. Inferior T waves may appear flattened or inverted.

Plasma volume increases throughout pregnancy, with a smaller increase in red cell volume, thus there is a physiological anaemia. The white blood cell count can rise during pregnancy.

Fibrinogen levels are mildly elevated, but thrombin and activated partial thromboplastin times can be shortened. Antithrombin III levels are reduced while factors VII, VIII and IX are increased.

Levels of creatinine and urea are significantly reduced, because of an increase in glomerular filtration rate and renal blood flow. Glycosuria is common during pregnancy.

THE STRUCTURED APPROACH TO THE PREGNANT PATIENT

The initial assessment of the pregnant patient remains unchanged but particular attention has to be paid to prompt resuscitation, as the integrity of the fetus depends on optimum assessment and management of the mother. There are specific components that need special attention, especially after considering the changes described above. These are detailed below:

- Cricoid pressure during assisted ventilation and subsequent intubation reduces the risk of aspiration of gastric contents.
- Endotracheal intubation may be more difficult. The combination of neck obesity and breast enlargement may prevent the insertion of the laryngoscope. This problem can be overcome by detaching the blade, which is then placed in the patient's mouth and reattached to the handle. As always, early recognition of the likely need for urgent intubation allows time to alert an expert in advanced airway management, thus reducing the need for immediate intervention in a desperate situation that may have been avoidable.
- Be wary of using the physiological tachypnoea as a marker of shock, in particular in the third trimester.
- Be wary of hypotension in the second trimester. Supine hypertension (described above) can be rapidly relieved by placing a wedge under the patient's right hip manually displacing the uterus to the left, off the vena cava.

SPECIFIC MEDICAL EMERGENCIES IN PREGNANCY

Asthma

Asthma is the commonest chest disease in pregnancy. The acute management of this condition has already been described (Chapter 8) and no modification of this treatment is required in the acute phase. Both a pneumothorax and pneumomediastinum are rare complications of pregnancy, but are more common than in the non-pregnant state. They occur more commonly in labour, presumably due to raised intrathoracic pressure during straining, especially if there is an underlying lung condition.

The physical signs of a pneumothorax have already been described along with its subsequent management.

In contrast, a pneumomediastinum presents with chest pain and, if extensive, the patient may be shocked, due to tension pneumomediastinum. Most leaks are believed to follow rupture of an emphysematous bulla with air tracking into the mediastinum and subcutaneous tissues.

Chest X-ray features are diagnostic. Most leaks resolve spontaneously. Rarely, in the case of large airway leaks, thoracotomy may be required if the shocked patient does not respond to appropriate treatment.

Acute respiratory distress syndrome

This is a major cause of maternal mortality as it is the final common pathway for many obstetric complications. The overlap between the adult acute respiratory distress syndrome and the systemic inflammatory response syndrome has been described earlier in Chapter 10. The particular obstetric causes of this syndrome are listed in Table 23.1.

The management of these conditions has been described earlier, but often there is no treatment specific to the particular condition, e.g. amniotic fluid embolism.

Table 23.1 Obstetric causes of systemic inflammatory response syndrome

Aspiration of gastric content		Daily
Shock	Antepartum haemorrhage or postpartum haemorrhage	Weekly
Disseminated intravascular coagulation	Severe pre-eclampsia, amniotic fluid embolus, dead fetus syndrome, gram negative septicaemia,	Monthly
Infection	Acute pyelonephritis	Monthly
Hydatidiform mole		Annually

Pulmonary embolus

This is a major cause of maternal mortality. The diagnosis and management has been described in Chapter 8. However, particular points that are pertinent to the pregnant patient include the following:

- Pregnancy is a risk factor for venous thromboembolism.
- Stasis – the venous stasis in the lower limbs is caused by compression of the enlarging uterus. Increasing age and parity are important risk factors, as is caesarean section.
- Previous thromboembolism increases the risk of a similar problem during pregnancy.

Other risk factors have been previously been described in Chapter 8.

The diagnosis of pulmonary embolus may be obvious clinically and be supported by appropriate clinical features, blood gas analysis and imaging.

However, massive pulmonary embolus has to be included in the differential diagnosis of shock. This has to be differentiated from an intra-abdominal cause of bleeding, especially if it occurs at the time of delivery. The latter will usually present with classic hypovolaemic shock, but remember that the relevant signs may be late in developing as the mother maintains her circulation at the expense of the fetus. The placenta is exquisitely sensitive to circulating catecholamines and rapid shut down of the uteroplacental unit will occur, jeopardising the fetus. Abdominal signs may also be present.

In contrast, the clinical features of pulmonary embolus may include signs of right-sided cardiac compromise, including a raised JVP, parasternal heave and a widely split second sound. According to the clinical picture, other less common conditions have to be considered in the shocked pregnant patient. These include dysrhythmia, myocardial infarction, pneumothorax/pneumomediastinum, aspiration of gastric contents and amniotic fluid embolism.

The management of pulmonary embolus in the pregnant patient does not differ from that described earlier. However, the clinical condition of the patient will dictate whether anticoagulation with heparin is appropriate. In the shocked patient, local facilities will dictate whether the management is thrombolysis or surgery. Subsequent treatment for the remainder of the pregnancy will be dictated by local protocol and discussion with the obstetrician and haematologist. Similarly, in patients who have known risk factors for thromboembolism, prophylaxis should be considered after discussion with a haematologist.

Heart disease in pregnancy

New onset of heart disease during pregnancy is rare. Patients with congenital heart disease will have been counselled carefully before pregnancy about potential problems and their solutions. Other cardiological conditions, including valvular disease, pre-existing cardiomyopathy and cardiac dysrhythmias are usually known before pregnancy and a careful management strategy is planned electively.

Occasionally, however, mitral stenosis may be detected for the first time during pregnancy. Management is unchanged from that described in Chapter 20. Digoxin and β blockers can be effective in controlling the ventricular rate. While diuretics can be used to reduce pulmonary oedema, it is important to avoid hypovolaemia.

Ischaemic heart disease is rare during pregnancy, but has a high mortality. Thrombolytic drugs are contraindicated in this situation.

Hypertension in pregnancy (and up to 2 weeks after delivery)

Hypertension in pregnancy can be caused by:

- pre-eclampsia syndrome due to the pregnancy
- pregnancy-induced hypertension which is transient, appears after midterm and resolves following delivery
- acute or chronic, pre-existing hypertension – classically towards the end of pregnancy
- due to an underlying medical condition not related to the pregnancy.

Thus all forms of hypertension in pregnancy, in particular those occurring after 20 weeks, should always be taken seriously. Diagnosis of the syndrome of

pre-eclampsia can be difficult. The risk factors for this condition are listed in the box below.

Risk factors for pre-eclampsia

Pregnancy
Multiple pregnancy
Age under 20 or over 35 years
Personal or family history of pre-eclampsia
Migraine
Pre-existing hypertension
Pre-existing renal disease

Clinical features of pre-eclampsia

- Hypertension
- Excessive weight gain
- Generalised oedema
- Ascites
- Epigastric pain
- Vomiting
- Hypertensive encephalopathy
- Cortical blindness

A patient with pre-eclampsia is usually asymptomatic. Epigastric pain suggests liver involvement. HELLP syndrome is a useful acronym to remember the components of haemolysis, elevated liver enzymes and low platelet count. However, none of the symptoms or signs are specific. Thus, the combination of hypertension, proteinuria and excessive weight gain should raise the suspicion of the pre-eclampsia syndrome. Not all of the clinical features listed above have to be present. Paradoxically, even hypertension and proteinuria do not appear to be essential components.

Clinical suspicion of pre-eclampsia syndrome can be supported by the results of the laboratory investigations, as listed in the box below.

Laboratory features of the pre-eclampsia syndrome

Proteinuria
Hyperuricemia
Hypocalcaemia
Thombocytopenia
Raised von Willebrand factor concentration
Reduced antithrombin III concentration
Haemolysis
Elevated liver enzymes – often with normal bilirubin

There are several important sequelae to the pre-eclampsia syndrome, most notably, tonic-clonic seizures or eclampsia(hence the use of the term pre-eclampsia). Other conditions are listed in the next box.

Sequelae to Pre-eclampsia

Tonic-clonic seizures
Cerebral haemorrhage
Cerebral oedema
Cortical blindness
Pulmonary oedema
Peripheral oedema
Acute respiratory distress syndrome
Disseminated intravascular coagulation
HELLP syndrome
Hepatic rupture or infarction
Renal cortical necrosis
Renal tubular necrosis
Fetal hypoxaemia/death

Key point

The sequelae to pre-eclampsia can arise before or after delivery

The management of pre-eclampsia syndrome is:

- Delivery – The placenta is the cause of the problem and therefore its removal is necessary.
- Control hypertension – this alone does not prevent the development of the pre-eclampsia syndrome. A variety of drugs are used according to local protocol. These may include hydralazine, labetalol and nifedipine. The complex nature of this syndrome often requires the involvement of multiple specialist colleagues.

Renal disease

This is uncommon in pregnancy. Acute renal injury is rare. It should be managed according to conventional guidelines, as described in Chapter 20. In late pregnancy in particular, this is usually associated with an underlying condition such as severe pre-eclampsia, eclampsia, prolonged intrauterine fetal death, amniotic fluid embolism or acute fatty liver.

Neurological problems

There is an increased incidence of ischaemic stroke during pregnancy and the puerperium. This is attributed to the mild hypercoagulable state that develops during the few weeks before and after delivery. This hypercoagulable state is attributed to the increased clotting factors, reduced level of antithrombin III and decreased fibrinolysis. However, it is important to exclude underlying procoagulant conditions, such as antithrombin III, protein C and protein S deficiencies, antiphospholipid antibodies, haemoglobinopathies and an underlying vasculitis. In young patients, an underlying cardiac cause, including embolic disease, has to be excluded.

In contrast, cerebral venous thrombosis occurs classically after delivery. The patient experiences a headache with progressive neurological deficit, fits and papilloedema. MRI is the investigation of choice.

SUMMARY

Normal pregnancy results in changes in airway, breathing (manifest as tachypnoea) and circulation/cardiovascular (manifest as transient hypotension and tachycardia).

Haematological (physiological anaemia and a prothrombotic state) values also change.

Pregnancy is also a risk factor for conditions such as pre-eclampsia, eclampsia and amniotic fluid embolism. The gravid uterus and changes in the mother's body mass can cause mechanical complications, such as vena caval compression and airway management problems, which must be anticipated.

The potential effects of certain drugs on the fetus may need to be borne in mind when considering the risk-benefit ratio of some treatments.

However, in general, the principles of treatment remain the same as for the non-pregnant patient. Optimum treatment of the mother provides the optimum treatment for the fetus.

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CHAPTER 24

The immunocompromised patient

OBJECTIVES

After reading this chapter you will be able to describe:

- why patients may have a compromised immune system and the infections they may incur
- a clinical approach to the immunocompromised patient.

SUSCEPTIBILITY TO INFECTION

Immunocompromised patients have alterations in phogocytic, humoral or cellular immunity that increase the risk of infection and reduce the ability to combat infection. Patient immunity may be impaired either temporarily or permanently due to:

- Immunodeficiency — congenital; acquired (HIV)
- Immunosuppression — disease; drugs; radiation

The risk of infection is related to the:

- Cause (see box below)
- Duration
- Absolute neutrophil count (highest risk if neutrophils $<500/mm^3$).

High risk	**Haematological malignancies**
	AIDS patients with low CD4+ counts
	Bone marrow transplants
	Splenectomy/Splenic dysfunction
	Genetic disorders (e.g. severe compound immunodeficiency)
Intermediate risk	**Solid tumours/Chemotherapy**
	HIV/AIDS
	Solid organ transplant
Low risk	**Long-term corticosteroid use**
	Diabetes mellitus

CAUSE OF INFECTION

This will vary according to:

- Type of immunosuppression
- Degree of immunodeficiency
- Duration of immunodeficiency

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Remember that as the immune system is impaired more than one organism/infection may be present. Hence a wide range of organisms can cause infection including:

- opportunistic organisms
- commensals
- *Candida albicans*
- *Cyclomegalovirus*
- nosocomial infections.

PRIMARY ASSESSMENT

This follows the structured ABC approach.

Particular importance must be placed on:

- universal precautions
- hypoxaemia due to pulmonary infections such as Pneumocystis Jiroveci
- shock due to volume depletion (e.g. increased insensible losses), poor diabetic control, fever, sepsis, haemorrhage
- confusion/focal neurological signs due to causes listed above
- sepsis, hypoglycaemia, intra-cranial infections
- fever. This is often the only symptom of infection and there is no pattern/periodicity.
- a thorough search of the skin and mucous membranes is essential for any clues/evidence of a rash.

Key point

A severely immunocompromised patient can have overwhelming infection without a fever

In addition to oxygen, fluids and other components of resuscitation, the following should be considered:

- manage in isolation
- intravenous broad-spectrum antibiotics – according to local policy
- antifungal agents to either treat or prevent secondary infection
- early liaison with specialists in microbiology, haematology, infectious diseases according to the clinical picture.

Investigations

The history and examination findings will govern the samples required including:

• Blood	for bacterial cultures, atypical and viral serology, fungal cultures
• Urine	gram stain culture, virology, antigens (e.g. legionella)
• Stool	microscopy, cysts, ova, parasites
• Nasal/throat swabs	culture
• Sputum	culture, microscopy
• Cerebrospinal fluid	culture, microscopy, cell count, PCR

SECONDARY ASSESSMENT

This should follow the usual format of:

1 'Phrased' history with particular reference to:
   - underlying diseases

- previous surgery
- use of illicit drugs
- sexual history
- travel history
- alcohol use
- prescribed drugs
- nutrition
- immunisations
- chemoprophylaxis
- family history.

2 Thorough physical examination to identify:
- site of infection
- clues to the underlying cause.

> **Key point**
>
> **The immunocompromised patient should be examined thoroughly every day**

SUMMARY

The immunocompromised patient often presents as a medical emergency. Assessment should follow the structured approach. The common cause is immunosuppression (including chronic disease, malignancy and associated chemo/radiotherapy) and acquired immunodeficiency (including HIV). Severe neutropenia is the greatest risk factor. Early empirical treatment with antibiotics and antifungals is required as is early specialist consultation.

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CHAPTER 25

The patient with acute spinal cord compression

OBJECTIVES

After reading this chapter you will be able to:

- understand why the diagnosis of acute spinal cord compression is delayed
- how to make this diagnosis earlier
- list the clinical features and underlying conditions associated with spinal cord compression
- describe the principles of management.

INTRODUCTION

This common condition often results in significant morbidity as the diagnosis is often delayed. A high index of suspicion is needed especially for patients with malignancy and back pain. The prognosis for recovery depends on the:

- severity of neurological deficit
- duration of deficit.

PATHOPHYSIOLOGY

The spinal cord is enclosed by a protective ring of bones, the vertebral column. Each bone comprises the vertebral body anteriorally and the lamina, pedicles and spinous process posteriorally. The spinal cord, covered by the thecal sac, extends from the base of the skull to the level of the first lumbar vertebra where it continues as the cauda equina, which is surrounded by cerebrospinal fluid. The thecal sac comprises the outermost layer is the dura and between the dura and the bone is the epidural space which normally contains fat and the venous plexus.

Causes of spinal cord compression

- Neoplastic — secondary = commonest
- Degenerative
- Trauma
- Inflammation — rheumatoid disease
- Infection — vertebral osteomyelitis (intraspinal)
- Haematoma — rare

Malignant spinal cord compression from secondary deposits is the commonest cause and is also a common complication of cancer affecting 5% of patients (bronchus, breast, renal, gastrointestinal, prostate, myeloma). The thoracic spine is most frequently involved (60%; lumbar-sacral 30%; cervical 10%) and arterial seeding of the bone probably accounts for most cases.

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CLINICAL FEATURES

Key point

Early, prompt recognition is crucial to improve outcome

- Pain
- Weakness
- Numbness
- Loss of bladder and bowel function
- Ataxia

Pain is usually the first symptom in over 90% of patients and precedes neurological symptoms by 2 months. The pain is initially local, then radicular and is often worse on sitting.

Weakness is present in approximately 75% of patients. The pattern of weakness reflects the level of cord compression, e.g. above the conus will be pyramidal and symmetrical (affecting the corticospinal tract) involving preferentially, the flexors in the legs (and extensors in the arms if above the thoracic spine).

With cauda equina lesions the reflexes in the legs are reduced. Occasionally an isolated motor radiculopathy occurs with a lateral epidural deposit.

Numbness is ascending and nearly as common as weakness. When a sensory level is present it is often one to five levels below the level of the cord compression. Sensory loss in a 'saddle' distribution is common in cauda equina lesions. In contrast, lesions above the cauda equina result in sacral sparing (to pinprick). Sensory loss can also occur in a radicular distribution.

Loss of bladder and bowel function is usually a late finding due to an autonomic neuropathy.

Ataxia consider spinal cord compression in any patient with symptoms and signs of suspected or diagnosed malignancy who has back pain and an ataxic gait.

PRIMARY ASSESSMENT AND RESUSCITATION

Airway and cervical spine, are assessed and managed according to the details in Chapter 3, 'C' spine immobilisation is of paramount importance when pathology is suspected. If in doubt, always seek expert help from colleagues in emergency medicine and orthopaedics.

Breathing is rarely a problem unless the cord compression is marked and either above C6, potentially effecting the diaphragm (C3, 4, 5 – phrenic nerve), or proximal to the thoracic spine affecting intercostal muscles.

Circulation is assessed as described in Chapter 3. Particular potential 'C' problems include sepsis, autonomic dysfunction associated with malignancy and the numerous issues associated with 'trauma'. Although this topic is far too extensive to be considered in this manual, the management principles for shock are the same as those described in Chapters 3 and 9.

Disability is rarely a problem unless the following occur:

- Coexisting intracranial pathology (e.g. metastases)
- Metabolic sequelae to malignancy (e.g. hypercalcaemia, hyponatraemia, hypoglycaemia)
- Drug effects.

Exposure problems are rare, but pressure sores and associated sepsis should be actively sought.

The other important management issue, although not resuscitation, is adequate analgesia. This not only is humane but will facilitate clinical examination and subsequent treatment.

The initial investigations have already been considered in detail (Chapters 3, 7, 9, 12 and 19). Magnetic resonance imaging is the modality of choice. CT with CT myelography is quicker but does not demonstrate clearly the spinal cord or epidural space. In contrast, MR produces excellent, accurate images of the cord and intramedullary pathology. It is also more sensitive than radioisotope bone scans at detecting bone metastases. However, MR is less likely to be well tolerated due to the duration of the scan. These tests are complimentary and the choice will depend on clinical need, patient preference, expert advice and the presence of 'metal implants'.

DIFFERENTIAL DIAGNOSIS

- Musculoskeletal pain
- Disc disease
- Spinal stenosis
- Spinal epidural abscess
- Vertebrae metastases without epidural extension
- Intramedullary metastases
- Malignant meningitis
- Malignant polyneuropathy

Management

Early referral to neurosurgery is essential. The precise management plan will vary according to the underlying cause, duration of symptoms/signs and the results of imaging.

There are some general principles:

- Pain management – usually opiates. Steroids may be beneficial in patients with malignancy
- Bed rest
- Anticoagulation – especially for patients with reduced mobility and cancer.
- Prevention of constipation – constipation can be due to limited mobility, analgesia, hypercalcaemia and autonomic dysfunction.

SUMMARY

Acute spinal cord compression is a relatively common condition. The diagnosis is often delayed. A high index of suspicion is needed in susceptible patients – especially those with malignant disease. Back pain is the cardinal feature, along with weakness, numbness, loss of bowel and bladder function. Early referral to a neurosurgeon is crucial to improve outcome.

PART VI

Interpretation of Emergency Investigations

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CHAPTER 26

Acid–base balance and blood gas analysis

OBJECTIVES

After reading this chapter you will be able to:

- describe the meanings of the common terms used in acid–base balance
- describe how the body removes carbon dioxide and acid
- explain the causes of an increased anion gap
- understand the system for interpreting a blood gas result.

TERMINOLOGY

It is important to understand the meaning of the terms commonly used when discussing acid–base balance.

Acids and bases

Key point

An acid is any substance which is capable of providing hydrogen ions (H^+)

Originally the word **'acid'** was used to describe the sour taste of unripe fruit. Subsequently many different meanings have led to considerable confusion and misunderstanding. This was not resolved until 1923 when the following definition was proposed.

A strong acid is a substance that will readily provide many hydrogen ions and conversely, a weak acid provides only a few. In the body we are mainly dealing with weak acids such as carbonic acid and lactic acid.

The opposite of an acid is a **base** and this is defined as any substance that 'accepts' hydrogen ions. One of the commonest bases found in the body is bicarbonate (HCO^{3-}).

The pH scale, acidosis, acidaemia, alkalosis and alkalaemia

The concentration of hydrogen ions in solution is usually very small, even with strong acids. This is particularly true when dealing with acids found in the body where the hydrogen ion concentrations are in the order of 40 nmol/l.

Key point

A nanomole = 1 billionth of a mole

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To place this low concentration in perspective compare it with the concentration of other commonly measured electrolytes. For example, the plasma sodium is around **135 mmol/l**, i.e. 3 million times greater!

Dealing with such very small numbers is obviously difficult and so in 1909 the pH scale was developed. This scale has the advantage of being able to express any hydrogen ion concentration as a number between 1 and 14 inclusively. The pH of a normal arterial blood sample lies between 7.36 and 7.44 and is equivalent to a hydrogen ion concentration of 44–36 nmol/l respectively.

It is important to realise that when using the pH scale, the numerical value **increases** as the concentration of hydrogen ions **decreases** (Fig. 26.1). This is a consequence of the mathematical process that was used to develop the scale. Therefore an arterial blood pH below 7.36 indicates that the concentration of hydrogen ions has increased from normal. This is referred to as an **acidaemia**. Conversely, a pH above 7.44 would result from a reduction in the concentration of hydrogen ions. This condition is referred to as an **alkalaemia**.

In contrast, acidosis and alkalosis are terms used to denote the initial acid base disturbance at cellular level that if uncorrected would result in acidaemia and alkalaemia respectively.

Another important consequence of the derivation of the pH scale is that **small changes in pH mean relatively large changes in hydrogen ion concentration**; e.g. a fall in the pH from 7.40 to 7.10 means the hydrogen ion concentration has risen from 40 to 80 nmol/l; i.e. it has doubled.

Key point

Small changes in the pH scale represent large changes in the concentration of hydrogen ions

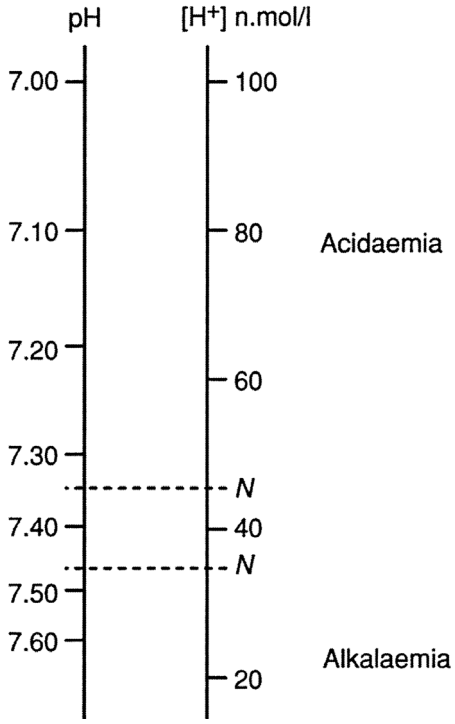

Fig. 26.1 The hydrogen ion scale.

Summary

- Hydrogen ions are only present in the body in very low concentrations.
- As the hydrogen ion concentration increases the pH falls.
- As the hydrogen ion concentration falls the pH rises.
- An acidaemia occurs when the pH falls below 7.36 and an alkalaemia occurs when it rises above 7.44.

Buffers

Many of the complex chemical reactions occurring at a cellular level are controlled by special proteins called enzymes. These substances can only function effectively at very narrow ranges of pH (7.36–7.44). However, during normal activity the body produces massive amounts of hydrogen ions, which if left unchecked would lead to significant falls in pH. Clearly a system is required to prevent these hydrogen ions causing large changes in pH before they are eliminated from the body. This is achieved by 'buffers'. They 'take up' the free hydrogen ions in the cells and blood stream, thereby preventing a change in pH.

There are a variety of buffers in the body. The main intracellular ones are proteins, phosphate and haemoglobin. Extracellularly there are also plasma proteins and bicarbonate. Proteins 'soak up' the hydrogen ions like a sponge and transport them to their place of elimination from the body, mainly the kidneys. In contrast, bicarbonate reacts with hydrogen ions to produce water and carbon dioxide.

$$H^+ + HCO_3^- \Leftrightarrow H_2O + CO_2$$

The carbon dioxide is subsequently removed by the lungs.

With these common terms defined, let's consider why people can become acidaemic and how this can be corrected by the body.

ACID PRODUCTION AND REMOVAL

All of us, whether we are healthy or ill, produce large amounts of water, acid and carbon dioxide each day. A healthy adult will normally produce 1,5000,000 nmol of hydrogen ions each day as waste products generated when food is metabolised to release energy. This process occurs at a cellular level where these products initially accumulate. If this were left unchecked irreparable cellular damage would result.

The first acute compensatory mechanism is the intracellular buffering system. As described previously, this provides the cell with a temporary way of minimising the fluctuations in acidity. Subsequently, these waste products (i.e. carbon dioxide and hydrogen ions) are excreted into the blood stream where they are taken up by the extracellular buffers (Fig. 26.2).

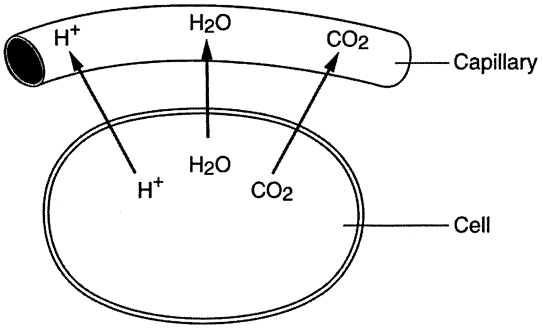

Fig. 26.2 Removal of waste products from cells.

However, this is only a temporary solution because there is only a limited amount of buffer. If this was the sum total of the body's defence to carbon dioxide and acids then the buffers would soon be saturated. Hence, a system is needed to remove these harmful substances from the body so that they do not reach toxic levels and, at the same time, regenerate the buffers. Fortunately, the body can eliminate these waste products removed mainly by the lungs and the kidneys.

Carbon dioxide removal (the respiratory component)

Carbon dioxide (CO_2) released from cells is transported in the blood to the lungs after diffusing into the alveoli is removed from the body during expiration (Fig. 26.3).

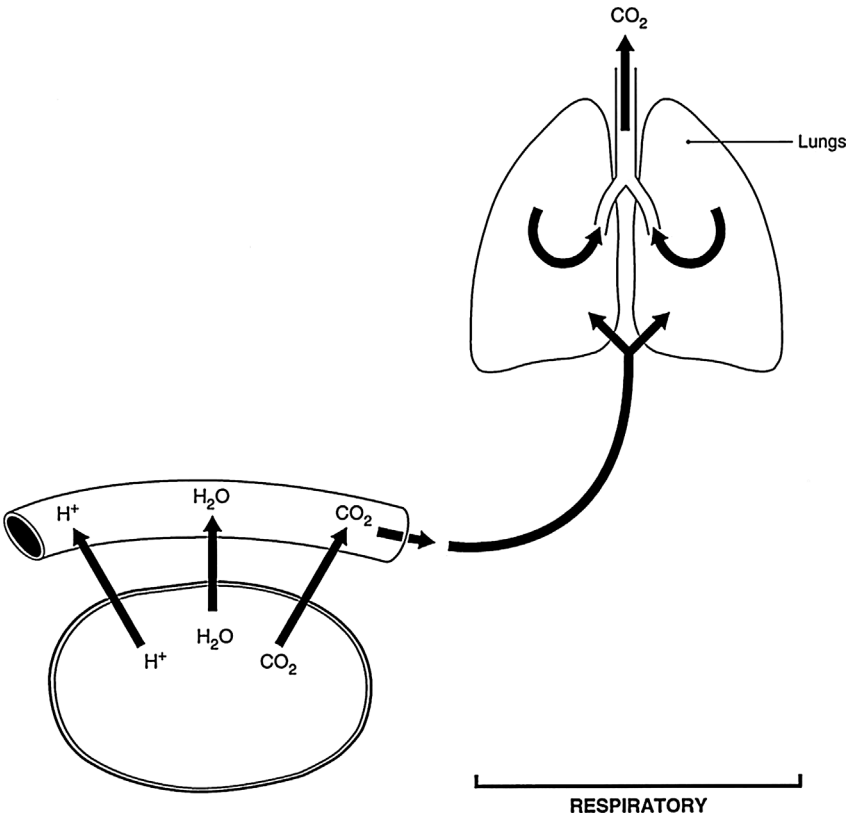

Fig. 26.3 Removal of carbon dioxide by the lungs.

If carbon dioxide production is faster than elimination or there is a blockage to its removal, then it will accumulate in the blood stream. In the plasma CO_2 reacts with water to produce hydrogen ions (H^+) and bicarbonate (HCO_3^-):

$$CO_2 + H_2O \Leftrightarrow H^+ + HCO_3^-$$

The greater the amount of carbon dioxide, the more hydrogen ions are produced. If this increase in plasma concentration of hydrogen ions causes the pH to fall below 7.36 then an acidaemia occurs. As the cause of the **acidaemia** in this case is a problem in the respiratory system, it is known as a **respiratory acidaemia**.

If a sample of arterial blood was taken immediately this occurred then the result given in Table 26.1 would be obtained.

Table 26.1 Effect of a respiratory acidosis on blood gas analysis

	Normal	Respiratory acidaemia
pH	7.36–7.44	↓
$PaCO_2$	4.7–6.0 kPa 35–45 mm Hg	↑
Actual HCO_3^-	21–28 mmol/l	↑

Immediately after a rise in carbon dioxide the pH falls as H^+ ion concentration rises. There is a small rise in the concentration of actual bicarbonate.

As a by-product of the reaction between carbon dioxide and water, the actual bicarbonate concentration also increases by the same amount as the hydrogen ions. However, this increase is usually in the order of several nanomoles. As the normal concentration is 21–27 mmol (i.e. 21,000–27,000 nmol) the net increase in actual bicarbonate is very small. Consequently these changes in concentration are enough to change the pH scale but are not large enough to alter significantly the plasma bicarbonate concentration.

In a normal person at rest, the respiratory component will excrete at least 1,2000,000 nmol of hydrogen ions per day. It is therefore easy to see that there can be a rapid onset of acidosis during episodes of hypoventilation.

Acid removal (the metabolic component)

Acids are continuously produced as a result of cellular metabolism. The amount produced from normal metabolism is approximately 3,000,000 nmol/day. This acid load is soaked up by buffers in the blood stream so that they can be transported safely for elimination (Fig. 26.4).

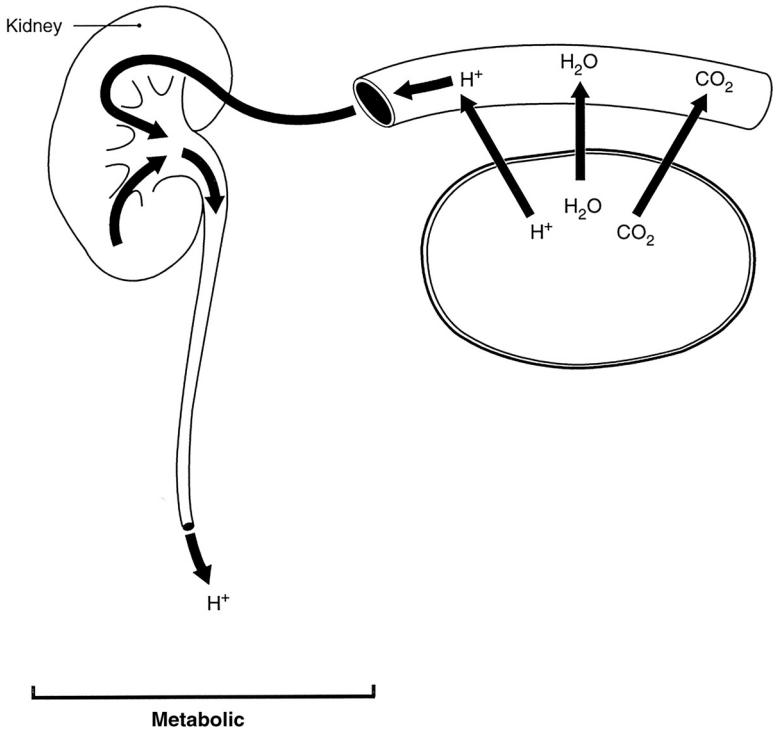

Fig. 26.4 Removal of hydrogen ions by the kidney.

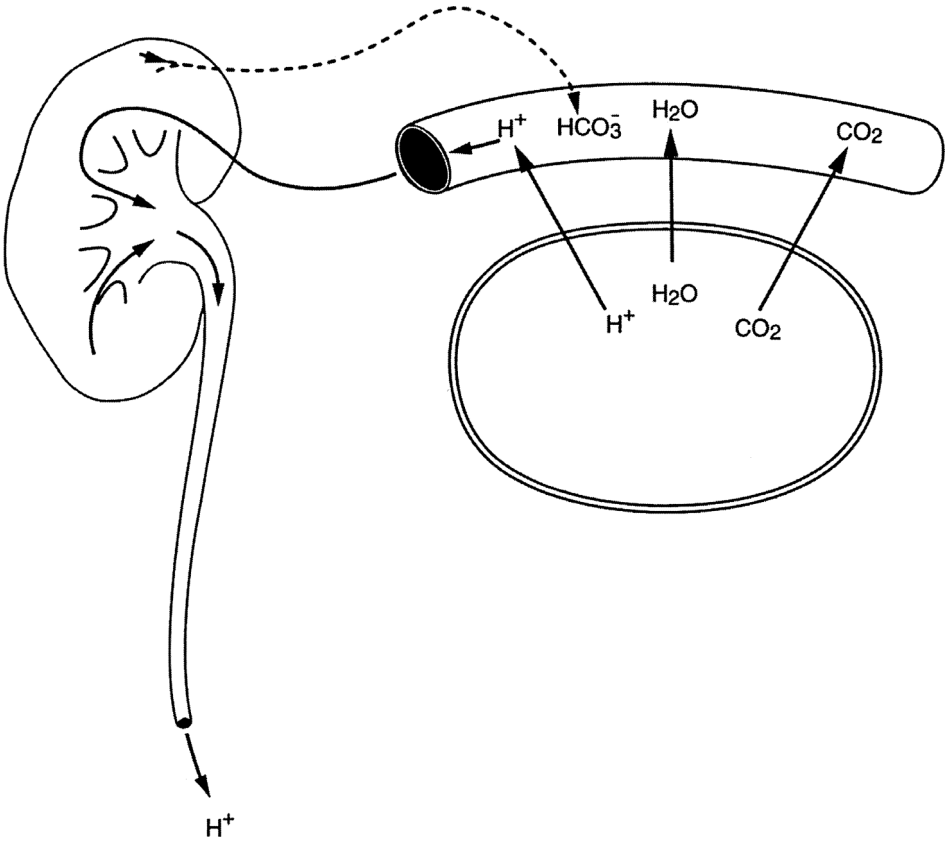

Fig. 26.5 Release of bicarbonate into the blood.

One of the buffers is bicarbonate. This is generated by the kidneys and released into the blood stream where it reacts with free hydrogen (Figure 26.5).

In certain circumstances, so much acid is produced by the cells that it exceeds the capacity of the protein buffers and bicarbonate. If this results in an accumulation of free hydrogen ions in the plasma so that the pH falls below 7.36 then an **acidaemia** has been produced. As this is a result of a defect in the metabolic system, it is termed a **metabolic acidaemia**.

If a sample of arterial blood was taken when this occurred then the result given in Table 26.2 would be obtained.

Table 26.2 Effect of a metabolic acidaemia (1)

	Normal	Metabolic acidaemia
pH	7.36–7.44	↓
$PaCO_2$	4.7–6.0 kPa 35–45 mm Hg	
Actual HCO_3^-	21–28 mmol/l	↓

An increase in carbon dioxide is too small to be observed because it is of the same order of magnitude as the increase in free hydrogen ion concentration, i.e. in nmol/l.

The actual bicarbonate level has fallen as a consequence of reacting with the free hydrogen ions to produce carbon dioxide and water. However, as noted

previously, this concentration is also affected by respiration. It follows that this concentration represents the effects of both the metabolic and respiratory systems. What is required therefore is a measure of the bicarbonate concentration resulting from only the metabolic system. This is why the blood analyser will also give a standard bicarbonate concentration as well (see results in Table 26.3).

Table 26.3 Effect of metabolic acidaemia (2)

	Normal	Metabolic acidaemia
pH	7.36–7.44	↓
$PaCO_2$	4.7–6.0 kPa	4.7–6.0 kPa
	35–45 mm Hg	35–45 mm Hg
Actual HCO_3^-	21–28 mmol/l	↓
Standard HCO_3^-	21–27 mmol/l	↓

The standard bicarbonate is an estimate of what the bicarbonate concentration would be, if the $PaCO_2$ were in the middle of the normal range. In other words, the contribution of any respiratory abnormality to HCO_3^- ion concentration is removed.

Base excess and base deficit

Both base excess and deficit are equivalent to the standard bicarbonate as another estimate of the contribution of the non-respiratory component to acid–base regulation; i.e. how much excess base (alkali) would be in the body if the $PaCO_2$ were in the middle of the normal range.

However, the difference from standard bicarbonate is that base excess and deficit take into account all the buffers in the blood sample and are considered a more accurate assessment of the metabolic component of acid–base status.

It follows that a **base excess** of 3 mmol/l means that 3 mmol of a strong acid had to be added to each litre of the original sample to get the pH to 7.4, with temperature kept at 37°C, and $PaCO_2$ kept at 5.3 kPa (40 mm Hg). Conversely a **base deficit** of 3 mmol/l means that 3 mmol of a strong base had to be added to each litre of the original sample to get the pH to 7.4 under the same conditions mentioned above.

For simplicity, many laboratories only use the term 'base excess'. A **negative** base excess is equivalent to base deficit. With the above examples, a base deficit of 3 mmol/l would be reported as a base excess of −3 mmol/l, and a true base excess of 3 mmol/l would be reported as a base excess of +3 mmol/l. The normal range of values for a base excess described in this way is −2 to +2 mmol/l.

A base excess below −2.0 mmol/l indicates that there is a metabolic acidosis in the metabolic component to the acid–base balance.
A base excess above +2.0 mmol/l indicates that there is a metabolic alkalosis in the metabolic component to the acid–base balance.

Considering these points it is possible to understand the results from the blood gas analysis of the respiratory and metabolic acidaemia cases discussed previously:

Respiratory acidaemia: see Table 26.4

Table 26.4 Effect of respiratory acidaemia

	Normal	Respiratory acidaemia
pH	7.36–7.44	↓
$PaCO_2$	4.7–6.0 kPa	↑
	35–45 mm Hg	
Actual HCO_3^-	21–28 mmol/l	↑
Standard HCO_3^-	21–27 mmol/l	–
Base Excess	−2 to +2 mmol/l	–

Metabolic acidaemia: see Table 26.5

Table 26.5 Effect of metabolic acidaemia

	Normal	Metabolic acidaemia
pH	7.36–7.44	↓
$PaCO_2$	4.7–6.0 kPa	4.7–6.0 kPa
	35–45 mm Hg	35–45 mm Hg
Actual HCO_3^-	21–28 mmol/l	↓
Standard HCO_3^-	21–27 mmol/l	↓
Base Excess	−2 to +2 mmol/l	↓

Summary

- CO_2 and metabolic acids are continuously produced by cell metabolism.
- The body has two methods of removing these waste products of metabolism and thereby maintain acid–base balance.
- Removal of CO_2 by the lungs regulates the respiratory component of the body's acid–base status.
- Excretion of H^+ ions and generation of HCO_3^- ions by the kidney are the main regulators of the metabolic component of acid–base balance.
- The actual bicarbonate concentration depends on both the respiratory and metabolic systems. In contrast, the standard bicarbonate and base excess are dependent only on the metabolic system.

The respiratory–metabolic link

The body therefore has two distinct methods of preventing the accumulation of hydrogen ions and the subsequent development of an acidaemia. As a further protection these two components are in balance (or equilibrium) so that each can **compensate** for a derangement in the other.

This link between the respiratory and metabolic systems is due to the presence of **carbonic acid** (H_2CO_3) (Fig. 26.6). The ability for each system to compensate for the other becomes more marked when the initial disturbance in one system is prolonged.

The production of carbonic acid depends on an enzyme called carbonic anhydrase that is present in abundance in the red cells and the kidneys. It is therefore ideally placed to facilitate the link between the respiratory and the metabolic systems.

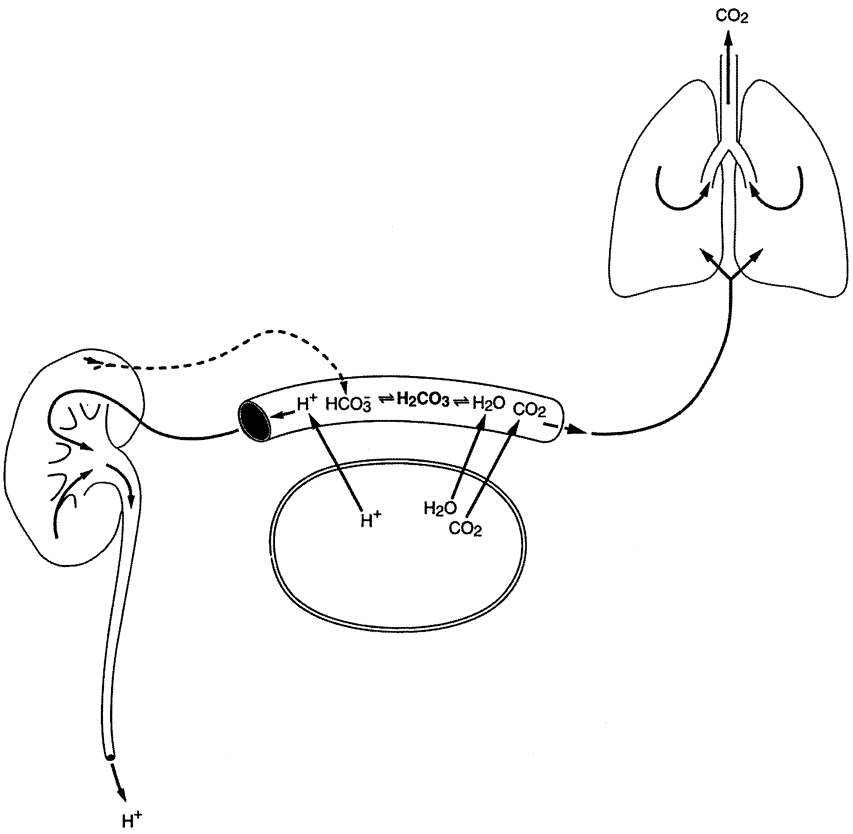

Fig. 26.6 Carbonic acid–bicarbonate buffers: acid production and its removal.

Let us consider how this link can help the body respond to an excess of either carbon dioxide or acid.

Example 1

In a patient with inadequate alveolar ventilation, e.g. chronic bronchitis, carbon dioxide accumulates. This will tend to cause a respiratory acidosis. Rather than the body existing in a chronic state of acidosis, the metabolic system can help compensate by increasing bicarbonate production by the kidneys. Using the carbonic acid link enables the removal of some of the excess carbon dioxide (Fig. 26.7 – see page 396). However, this takes several days to become effective as it depends on the increased production of enzymes in the kidney.

It is important to realise that in the acute situation **the body does not fully compensate**. Consequently, if an arterial blood sample is taken at this time, it will demonstrate that there is still a persistent but slight underlying acidaemia (Table 26.6).

Table 26.6 Underlying acidaemia

	Normal values	Effect of a respiratory acidaemia	Effect of metabolic compensation
pH	7.36–7.44	↓ ↓	↓
$PaCO_2$	4.8–5.3 kPa 36–40 mm Hg	↑	↑
Standard HCO_3^-	21–27 mmol/l	N	↑ ↑
Base excess	± 2 mmol/l	N	↑ ↑

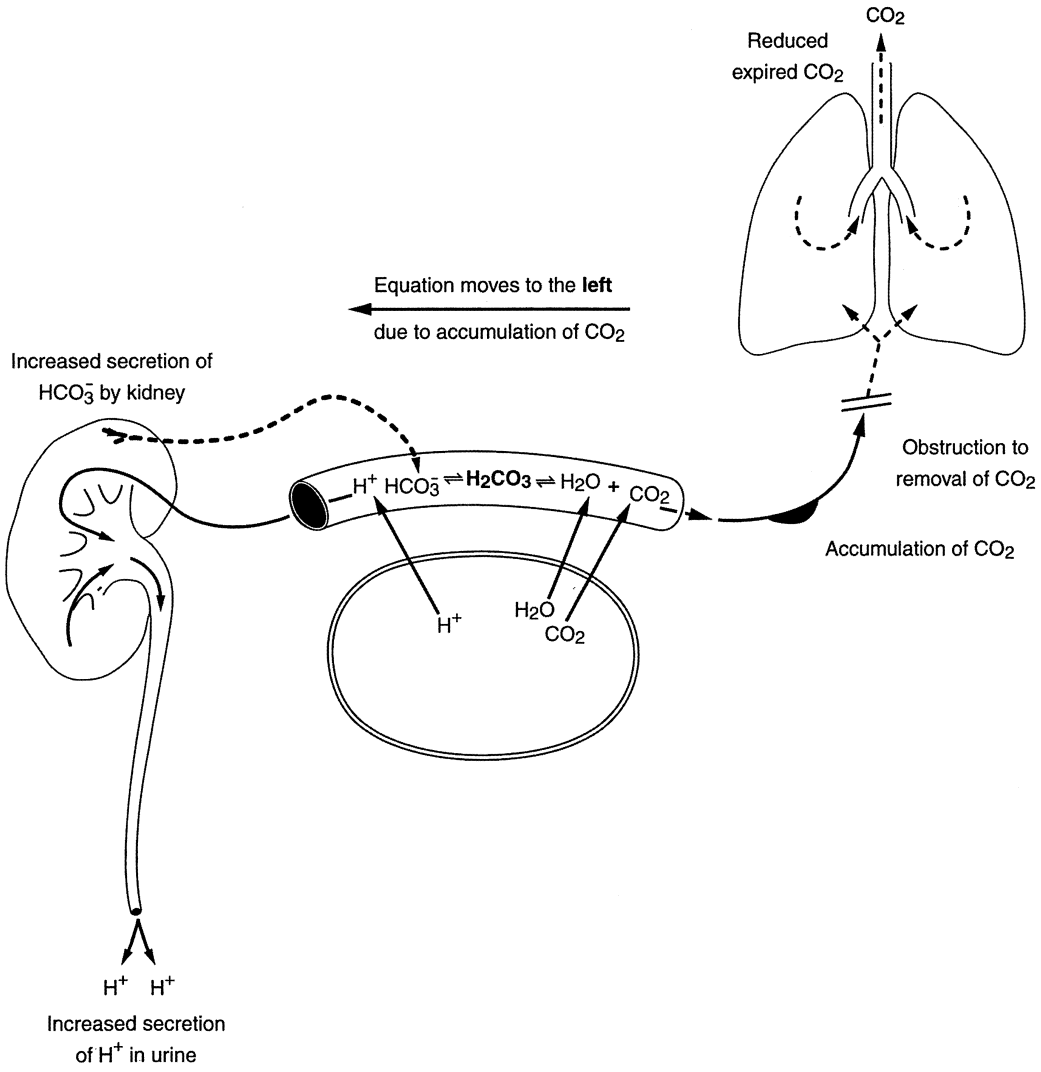

Fig. 26.7 Metabolic compensation for a respiratory acidosis (i.e. the metabolic system is compensating for the respiratory system).

Example 2

Diabetic patients sometimes develop a state of excess acid production known as **diabetic ketoacidosis**. The excess cellular acid is released into the plasma to be transported to the kidney for excretion. However, the kidneys are only able to excrete the additional acid load slowly and a metabolic acidosis develops. The kidneys are slowly stimulated to increase bicarbonate production. This will counteract the acidaemia but it takes several days. In the meantime, because of the carbonic acid link, some of the excess acid can be converted to carbon dioxide and eliminated by the respiratory system (Fig. 26.8).

This compensation occurs quickly because excess hydrogen ions are detected by special receptors in the brain which, in turn, increase the respiratory rate and depth within minutes (compare this with the slow response of the kidneys). This process enables the body to eliminate the extra carbon dioxide, providing that there is no obstruction to ventilation. The lowering of carbon dioxide levels in the blood encourages further free acid to be converted into carbonic acid and eventually carbon dioxide.

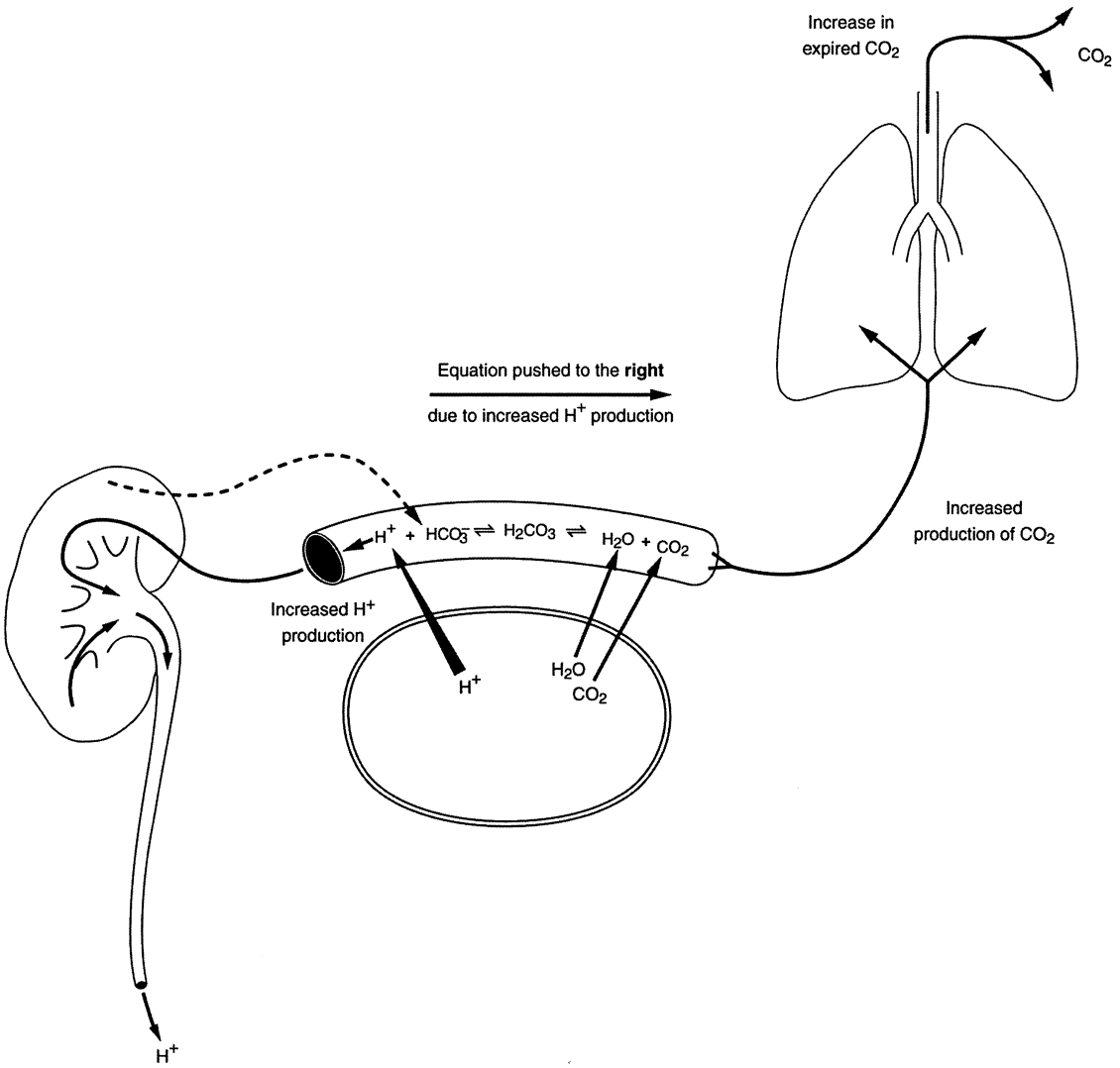

Fig. 26.8 Respiratory compensation for a metabolic acidaemia (i.e. the respiratory system is compensating for the metabolic system).

However, the body does not fully compensate in the acute situation. Therefore even after several hours, respiratory compensation will only be partial and the patient will still be slightly acidaemic (Table 26.7).

It must also be remembered that the degree to which the respiratory system can compensate depends on the work involved in breathing and the systemic effects of a low arterial concentration of carbon dioxide.

Table 26.7 Slight acidaemia

	Normal values	Effect of a metabolic acidosis	Effect of respiratory compensation
pH	7.36–7.44	↓ ↓	↓
$PaCO_2$	4.8–5.3 kPa 36–40 mm Hg	4.8–5.3 kPa 36–40 mm Hg	↓ ↓
Standard HCO_3^-	21–27 mmol/l	↓ ↓	↓
Base excess	± 2 mmol/l	↓↓	↓

Summary

- The metabolic component of the body's acid elimination mechanism can compensate for a respiratory acidosis by increasing the production of bicarbonate by the kidneys.
- Compensation by the metabolic component usually takes days to achieve.
- The respiratory component of the body's acid elimination mechanism can compensate for a metabolic acidosis by increasing ventilation of the lungs and hence eliminating carbon dioxide.
- Compensation by the respiratory component usually takes place within minutes.
- In the acute situation the body never fully compensates; therefore, the underlying acidaemia will remain.

Combined metabolic and respiratory acidaemia

Should both the metabolic and respiratory systems be defective or inadequate for the body's needs, then the accumulation of acid and carbon dioxide will be unchecked. An example of this particularly dire situation is seen in patients following a cardiorespiratory arrest. This results in the cells of the body producing lactic acid because they are being starved of oxygen. In addition, carbon dioxide accumulates in the cells and blood because it can no longer be excreted by the lungs due to the failure of ventilation (Fig. 26.9 – see page 399).

An arterial blood sample taken at this time would therefore demonstrate a combined respiratory and metabolic acidaemia (Table 26.8).

Table 26.8 Combined respiratory and metabolic acidaemia

	Normal values	Effect of respiratory and metabolic acidaemia
pH	7.36–7.44	↓ ↓ ↓ ↓
$PaCO_2$	4.8–5.3 kPa 36–40 mm Hg	↑ ↑
HCO_3^-	21–27 mmol/l	↓ ↓

CENTRAL VENOUS AND ARTERIAL BLOOD SAMPLES

So far we have concentrated on arterial blood analysis. This is blood that has had the benefit of passing through the lungs, where carbon dioxide can be eliminated and oxygen taken up. In contrast, central venous blood (i.e. blood in the right atrium) represents a mixture of all the blood returning to the heart from the body's tissues. It therefore has a high concentration of the body's waste products and low levels of oxygen (Table 26.9).

Compare these results with those following a cardiorespiratory arrest. In the absence of cardiopulmonary resuscitation blood will not flow through unventilated lungs. Therefore, the arterial sample and central venous sample will be **approximately the same**.

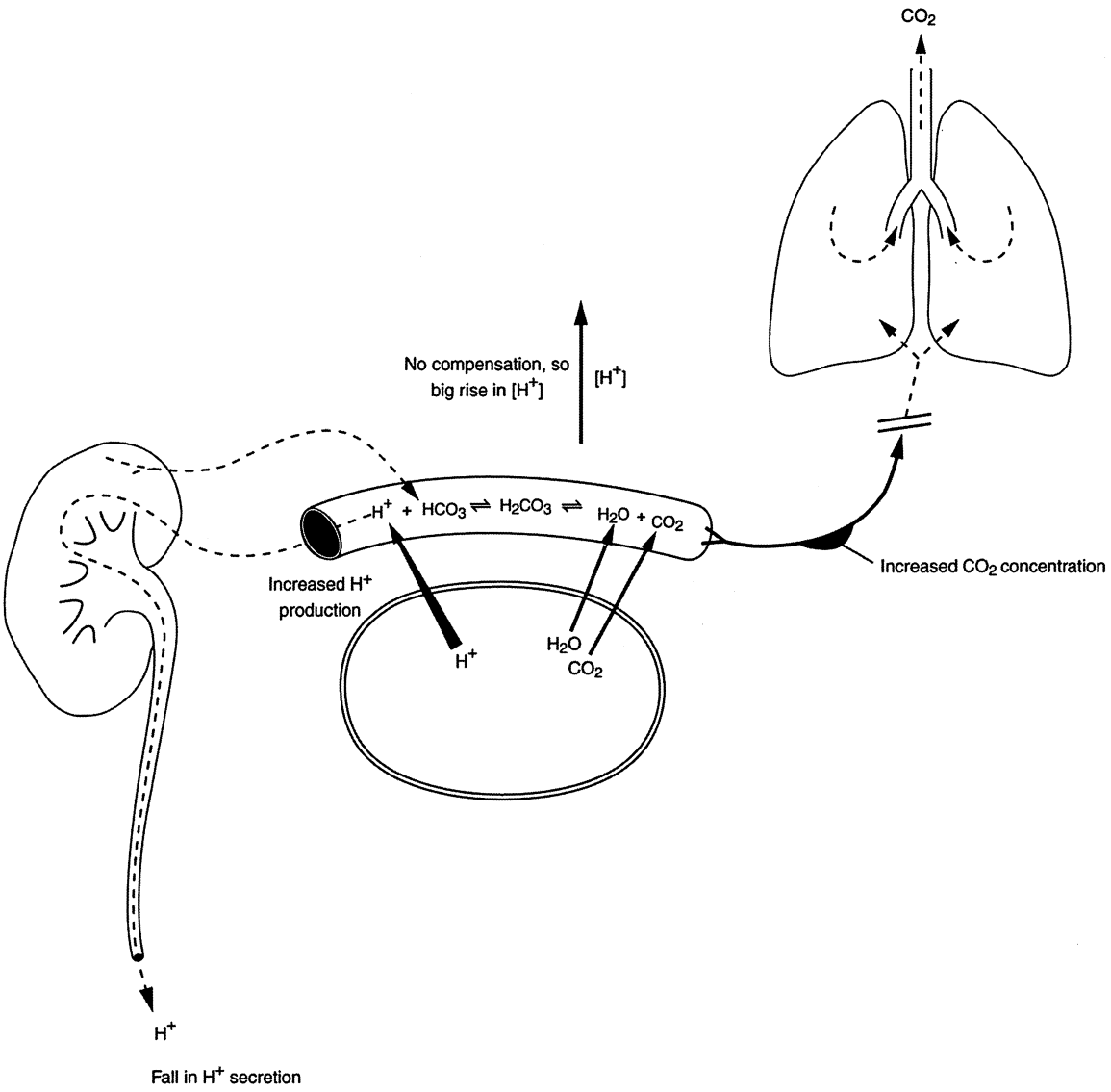

Fig. 26.9 Combined metabolic and respiratory acidosis.

In contrast, following endotracheal intubation, artificial ventilation and external chest compression, the carbon dioxide delivery to the alveoli is resumed. This is easily cleared by mechanical ventilation and some oxygen is taken up. The removal of carbon dioxide can be so effective that there is a marked reduction in

Table 26.9 Comparison of the composition of arterial and central venous blood

	Arterial blood	Central venous blood
pH	7.36–7.44	7.31–7.40
$PaCO_2$	4.8–5.3 kPa	5.5–6.8 kPa
	36–40 mm Hg	41–51 mm Hg
HCO_3^-	21–27 mmol/l	25–29 mmol/l
PaO_2 on air	Over 10.6 kPa	5.1–5.6 kPa
	Over 80 mm Hg	38–42 mm Hg

arterial carbon dioxide and the development of a paradoxical respiratory alkalosis (i.e. low arterial carbon dioxide despite high venous carbon dioxide and acidosis). Consequently, the arterial pH can be neutral, mildly acidotic or even alkalotic depending on how much carbon dioxide is being removed. In contrast, severe arterial acidosis in a patient receiving cardiopulmonary resuscitation indicates that resuscitation is inadequate, i.e. there is either inadequate blood flow to the lungs or inadequate ventilation or a combination of both.

The arterial sample taken during the resuscitation of a patient with a cardiorespiratory arrest is simply demonstrating the clinician's ability to remove carbon dioxide and add oxygen. The patient's true 'acid' state (i.e. pH, carbon dioxide and bicarbonate) is more accurately assessed by analysis of central venous blood.

A SYSTEMATIC APPROACH FOR ANALYSING A BLOOD GAS SAMPLE

There are many similarities between analysing a blood gas result and interpreting a rhythm strip. In both cases it is important to assess the patient first and to be aware of the clinical history and current medications. A review of the other laboratory investigations is also helpful. In the emergency situation, however, these data may not be immediately available. Consequently, you will have to interpret the initial results with caution and follow trends whilst the rest of the information is being obtained.

The system

History
- **Any symptoms due to the cause of an acid–base disturbance?**
- **Any symptoms as a result of an acid–base disturbance?**

Results
- **Is there an acidaemia or alkalaemia?**
- **Is there evidence of a disturbance in the respiratory component of the body's acid–base balance?**
- **Is there evidence of a disturbance in the metabolic component of the body's acid–base balance?**
- **Is there a single or multiple acid–base disturbance?**
- **Is there any defect in oxygen uptake?**

Integration
- **Do the suspicions from the history agree with the analysis of the results?**

Is there an acidaemia or alkalaemia?

In most patients there will be an acute single acid–base disturbance. In these circumstances the body rarely has the opportunity to completely compensate for the alteration in hydrogen ion concentration. Consequently the pH will remain outside the normal range and thereby indicate the underlying acid–base disturbance.

pH less than 7.36 = underlying acidaemia

pH greater than 7.44 = underlying alkalaemia

In acute, single acid–base disturbances the body usually does not have time to fully compensate. The pH will therefore indicate the primary acid–base problem.

Nevertheless a normal pH does not necessarily mean the patient does not have an acid–base disturbance. In fact there are three reasons for a patient having a pH within the normal range:

- There is no underlying acid–base disturbance.
- The body has fully compensated for a single acid–base disturbance.
- There is more than one acid–base disturbance with equal but opposite effects on the pH.

Using your knowledge of the patient's history and examination you will have a good idea which of these options is the true answer. However, to confirm or refute your suspicions you will need to see if there are alterations in the respiratory and metabolic components of the body's acid–base balance. This entails reviewing the $PaCO_2$ and standard bicarbonate (or base excess) respectively.

Is the abnormality due to a defect in the respiratory component?

The $PaCO_2$ gives a good indication of ventilatory adequacy because it is inversely proportional to alveolar ventilation. When combined with pH it can be used to determine either if there is a problem with the respiratory system or if the respiratory component is simply compensating for a problem in the metabolic component.

For example: an arterial sample with a pH of 7.2 and a $PaCO_2$ concentration of 8.0 kPa (60 mm Hg). A pH of 7.2 indicates that there is an acidaemia. As the $PaCO_2$ is raised, this indicates that there is a **respiratory acidosis**. Consider now a patient with a similar pH but a $PaCO_2$ of 3.3 kPa (25 mm Hg). There is still an acidaemia but as the $PaCO_2$ is lowered, it would imply there is **respiratory compensation to a metabolic acidosis**. To confirm this, the metabolic component would need to be assessed.

Is the abnormality due to a defect in the metabolic component?

To determine the metabolic component, the concentration of bicarbonate is measured. In a similar situation to that described earlier, when the bicarbonate concentration is combined with pH one can determine if there is either a primary metabolic or compensatory metabolic problem.

Using the second example above, the standard bicarbonate was found to be 9.5 mmol/l and the base excess –10 mmol/l. Consequently, a pH of 7.2 and a $PaCO_2$ concentration of 3.3 kPa (25 mm Hg) is in keeping with the idea that this patient has a **respiratory compensation to a metabolic acidosis**.

Is there a single or multiple acid–base disturbance?

To narrow down the diagnosis even further we need to consider how much the $PaCO_2$ and bicarbonate (base excess) concentration has changed. If these changes fall within certain limits then there is usually only a single acid–base disturbance. Alternatively if they are outside this range then it is likely that the patient has more than one acid–base disturbance.

Take a moment to familiarise yourself with the layout of Flenley's graph (Fig. 26.10). In particular note the following:

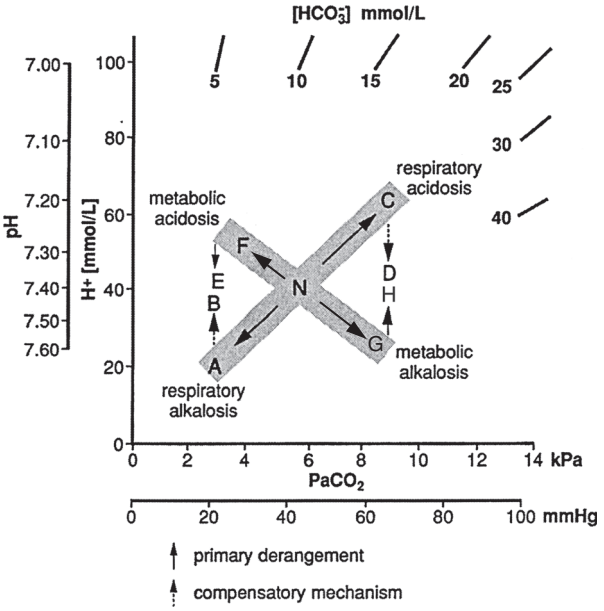

Fig. 26.10 Flenley's graph.

- The graph is showing how the pH alters with changes in $PaCO_2$.
- Cutting diagonally across the graph are lines which indicate the concentration of bicarbonate. These are known as isopleths.
- As the concentration of standard bicarbonate increases the gradient of the isopleths falls.
- The square box indicates the normal range for pH, $PaCO_2$ and actual bicarbonate concentration (N).
- Fanning out from this box are the possible ranges of normal responses you could expect with single acid–base disturbances.
- The bands representing the acute respiratory disturbances run approximately parallel to the isopleths. Respiratory acidaemia (C) and alkalaemia (A) will alter the pH and $PaCO_2$ but have little effect on the bicarbonate concentration. These bands do not include patients who have had long enough to develop metabolic compensation and so alter their bicarbonate concentrations. Such patients are represented in the chronic respiratory acidosis group (B and D).
- The band representing the metabolic disturbances runs across the isopleths. Therefore metabolic acidaemia (F) and alkalaemia (G) will alter the bicarbonate concentration as well as the pH and $PaCO_2$. These bands include the patients who are using respiratory compensation to counteract the pH changes. However, it does not include those patients who have had long enough to develop metabolic compensation (i.e. those who have a chronic metabolic disturbance (E and H)).

Using this graph you can plot the results from the blood gas analysis. If it lies within one of these bands then there is likely to be only one acid–base disturbance. However, if the results lie outside these normal ranges then there is likely to be more than one acid–base disturbance.

You have now finished the interpretation of the parameters in the blood gas analysis which provide information on the patient's acid–base balance. There is, however, one more important value which needs to be assessed in an arterial sample and that is the partial pressure of oxygen.

This is important because a failure to take up oxygen can lead to many adverse conditions including hypoxaemia. With regard to the acid–base balance,

hypoxaemia can give rise to metabolic acidosis because it causes the cells to change to anaerobic metabolism and so produce excessive quantities of lactic acid.

Defect in oxygen uptake

By knowing the FiO_2 it is possible to predict what the PaO_2 would be if the patient was ventilating normally.

Since atmospheric pressure is 100 kPa (approximately 760 mm Hg), 1% is 1 kPa or 7.6 mm Hg. This would mean that inspiring 30% oxygen from a facemask would produce an inspired partial pressure of oxygen of 30 kPa (268 mm Hg). This should lead to an arterial concentration of around 20–25 kPa (152–257.6 mm Hg) because there is a normal drop of about 7.5 kPa (57 mm Hg) between the partial pressure of oxygen inspired at the mouth and that in the alveoli. A drop of significantly greater than 10 kPa (76 mm Hg) would imply that there is a mismatch in the lungs between ventilation of the alveoli and their perfusion with blood.

For example, an arterial PaO_2 of 32.9 kPa (250 mm Hg) in a patient breathing 40% oxygen is within normal limits. In contrast, an arterial PaO_2 of 23.7 kPa (180 mm Hg) in a patient breathing 50% oxygen indicates that there is a defect in the take-up of oxygen. An inspired oxygen of 50% will have a partial pressure of approximately 50 kPa. This would mean the expected PaO_2 would be at least $50 - 10 = 40$ kPa.

Example

Using this system for interpreting blood gases let us now consider the following case.

History

A 17-year-old girl who is normally in good health is found at home by her parents in a restless and confused state. She is pale, sweaty and hyperventilating.

Results

Whilst she was breathing room air, an arterial sample was taken for blood gas analysis. The results are given in Table 26.10.

Table 26.10 Example arterial blood sample

	Normal values	Patient's values
pH	7.36–7.44	7.10
$PaCO_2$	4.8–5.3 kPa	2.4 kPa
	36–40 mm Hg	18 mm Hg
Standard HCO_3	21–27 mmol/l	5.5 mmol/l
Base excess	±2 mmol/l	−14 mmol/l
PaO_2	Over 12.0 kPa	14 kPa
	Over 90 mm Hg*	105 mm Hg*

*On room air.

Analysis

History

Hyperventilation may be a primary problem (e.g. anxiety) or compensation for an underlying metabolic acidaemia. You would therefore suspect from the history

that there could be either a respiratory alkalaemia or a metabolic acidaemia with respiratory compensation. With regard to acid–base balance you can also deduce from the history that this is an acute event. It is therefore unlikely that there would be sufficient time for any metabolic compensation in either of the possible acid–base disturbances suspected.

Systematic analysis of the blood gas results

Is there an acidaemia or alkalaemia?

- As the pH is below 7.36 there is an acidaemia.

Is there evidence of a disturbance in the respiratory component of the body's acid–base balance?

- Yes, the $PaCO_2$ is low. In the light of the pH, this indicates there is either respiratory compensation to a metabolic acidosis or a combination of a big metabolic acidosis and smaller primary respiratory alkalosis.

Is there evidence of a disturbance in the metabolic component of the body's acid–base balance?

- Yes, the bicarbonate concentration is low and the base excess is very negative. In the light of the pH and $PaCO_2$ this supports the two possibilities suggested in the previous question.

Is there a single or multiple acid–base disturbance?

- Using Fig. 26.10 (see page 402) you can see that the results lie within the metabolic acidaemia band. This would imply that the patient has a metabolic acidaemia with respiratory compensation and has not had time to develop metabolic compensation.

Is the PaO_2 uptake abnormal?

- The expected PaO_2 when breathing room air is over 12.0 kPa (over 90 mm Hg). There is, therefore, no evidence of any problem in oxygen uptake in this patient.

Integrate the clinical findings with the data interpretation

The clinical and data analyses tally. This girl has a metabolic acidaemia with respiratory compensation. Your next move would be to determine what is the cause of the acidaemia. This involves further tests which are selected in the light of the patient's history and physical examination. A useful initial screen is to determine the anion gap.

ANION GAP

Derivation

The body needs to ensure that electroneutrality is maintained because an imbalance would impair cellular function. This means the total number of negatively charged particles (anions) and positively charged particles (cations) must be equal.

We can therefore write that in the circulation:

Total concentration of anions = total number of cations

You will be aware that only some of the anions and cations are routinely measured. We can therefore adjust the above equation to:

Total concentration of measured anions + Total concentration of unmeasured anions
=
Total concentration of measured cations + Total concentration of unmeasured cations

If we use the symbol [ ] to indicate concentration, and insert the anions and cations which are routinely measured, the equation becomes:

$$[\text{Actual } HCO_3^-] + [Cl^-] + [\text{Total unmeasured anions}] = [Na^+] + [H^+] + [K^+] + [\text{Total unmeasured cations}]$$

This equation can be rearranged to read:

$$[\text{Total unmeasured anions}] - [\text{Total unmeasured cations}] = ([Na^+] + [H^+] + [K^+]) - ([\text{Actual } HCO_3^-] + [Cl^-])$$

The equation is rearranged this way because the total number of unmeasured anions is usually bigger than its cation counterpart. This difference is known as the anion **gap**. Consequently the equation becomes:

$$\text{Anion gap} = ([Na^+] + [H^+] + [K^+]) - ([\text{Actual } HCO_3^-] + [Cl^-])$$

Thinking back you will remember that the actual concentration of hydrogen ions is tiny compared to the other electrolytes. In addition, the concentration of potassium in plasma is small because it is mainly located inside the cells of the body. We can therefore remove these two concentrations from the equation without producing any significant errors. As a result the equation becomes:

$$\text{Anion gap} = [Na^+] - ([\text{Actual } HCO_3^-] + [Cl^-])$$

Key point

The anion gap is the difference in concentration between the unmeasured anions and cations

The normal range for the anion gap is 6–18 mmol/l

This value is obviously very dependent on the method used to measure the electrolytes. Chloride assessment in particular is being changed. As a result some departments will work on the newer normal range of anion gap which is 7 ± 4 mmol/l. It is therefore important that you find out from your laboratory what they consider the normal range to be.

Types of anion gap

Considering how the anion gap is derived it is easy to understand how it can be altered by changes in the concentration of unmeasured anions, unmeasured cations or a combination of the two.

Increase in the unmeasured anions	Decrease in the unmeasured cations
Metabolic acidosis	Hypokalaemia
Hyperalbuminaemia	Hypomagnesaemia
Marked alkalosis	Hypocalcaemia
Therapy with substances which produce unmeasured anions (e.g. sodium citrate, lactate or acetate. It also applies to excessive doses of penicillins particularly carbenicillin and ticarcillin)	

Key point

The most common cause of an increase in the anion gap is metabolic acidosis

Anion gap and metabolic acidosis

Metabolic acidoses can be divided into those with a wide anion gap or a normal anion gap. Widening of the anion gap results from an increase in the acid load on the body. In contrast, metabolic acidoses with a normal anion gap are produced by conditions leading to either the loss of bicarbonate or where the kidneys are unable to excrete the normal daily acid load.

Key points

Conditions with a widened anion gap include metabolic acidoses due to increases in the acid load on the body.

Conditions with a normal anion gap include metabolic acidoses due to either a loss of bicarbonate or impaired elimination of a normal acid load

METABOLIC ACIDOSIS WITH A WIDE ANION GAP

The most likely metabolic acidosis you will come across is one giving rise to an increase in the anion gap. In these conditions a metabolic acidosis is produced because the actual bicarbonate concentration falls:

$$\text{Anion gap} \uparrow = [Na^+] - ([HCO_3^-] \downarrow + [Cl^-])$$

Nevertheless, it is important to remember that metabolic acidosis represents only one of several causes of an increase in anion gap. In most cases though, these non-acidotic causes produce only a small increase in the anion gap.

Key point

An increased anion gap needs to be put into clinical context because this will enable the most appropriate investigations to be selected to confirm the diagnosis

Table 26.11 Causes of metabolic acidosis with an increased or normal anion gap

Increased anion gap		Normal anion gap	
M	Methanol	D	Diuretic – potassium sparing
E	Ethylene glycol	I	Intestinal fistula
D	Diabetic ketoacidosis	A	Acetozolamide
I	Isoniazid	R	Renal tubular acidosis (I and II)
C	Cachexia	R	Renal failure
A	Alcohol	H	Hypo-aldosteronism
L	Lactic acidosis	O	Oral resin (cholestryamine)
T	Toluene	E	Entero-ureterostomy
R	Renal failure/rhabdomyolysis	A	Alimentary feeding
I	Iron		
P	Paraldehyde		
S	Salicylate		

Causes

A metabolic acidosis with a widened anion gap is common because it is produced by several common clinical conditions. These can be remembered by the mnemonic 'medical trips' (Table 26.11). In contrast, a metabolic acidosis with a normal anion gap is uncommon. The causes can be remembered with the mnemonic 'diarrhoea'.

The young girl's blood sample was analysed further by measuring the appropriate electrolyte concentrations. In this case these were found to be:

- Na^+ 135 mmol/l
- K^+ 5.0 mmol/l
- HCO_3 10 mmol/l
- Cl^- 95 mmol/l

Using this information it is possible to determine the anion gap:

$$\begin{aligned}\text{Anion gap} &= [\text{Sodium}] - [\text{Bicarbonate} + \text{Chloride}] \\ &= [135] - [10 + 95] \\ &= 30 \text{ mmol/l}\end{aligned}$$

Therefore the 17-year-old patient has a metabolic acidaemia with a wide anion gap.

The most likely cause of a metabolic acidosis with an increased anion gap in a previously healthy adolescent is an overdose or diabetic ketoacidosis. Consequently the salicylate and blood sugar levels must be checked in this patient.

SUMMARY

The body's system for removing the carbon dioxide and acid produced by metabolism has both a respiratory and metabolic component. These are linked by the effects of carbonic acid that enables one component to compensate for a defect in the other.

In acute medical emergencies one or both of these systems are often defective. Using a systemic approach to blood gas analysis you can determine where the problem lies. In the common structure of a metabolic acidosis, the use of the anion gap can also be helpful in identifying a specific cause.

Advanced Life Support Group

CHAPTER 27
Dysrhythmia recognition

OBJECTIVES

After reading this chapter you will be able to:

- understand the origin and pathways of electrical activity in the heart
- recognise which patients require this activity to be monitored and how it should be done
- understand the system for analysing electrical activity recorded on a rhythm strip.

CARDIAC ELECTRICAL ACTIVITY: ITS ORIGIN AND ORGANISATION

The sinoatrial node (SAN) is a specialised area of cardiac muscle that generates spontaneously a continuous sequence of regularly timed waves of electrical activity known as depolarisations. These radiate through both atria, inducing contraction. The ability of the SAN to spontaneously depolarise is termed pacemaker activity, and may also be seen in other areas of the heart, usually at slower rates. As the wave of depolarisation spreads through the atria it gives rise to the 'P' wave on the electrocardiogram (ECG). Normally the P wave has a duration of 0.08–0.12 s, (80–120 ms).

Atrial depolarisation normally finishes by converging on a specialised collection of cells called the atrioventricular node located at the base of the right atrium. This delays the transmission of the depolarising wave to the ventricles and gives rise to a significant proportion of the PR interval. The latter is measured on the ECG tracing from the start of the P wave to the first deflection of QRS complex (0.12–0.20 s, 120–200 ms).

After the atrioventricular node, the wave of depolarisation is conducted through the fibrous atrio-ventricular barrier via the bundle of His. At the proximal part of the muscular intraventricular septum this splits into the right and left bundle branches, with the latter subsequently separating into anterior and posterior divisions (fascicles). These, in turn, terminate in small (Purkinje) fibres which transmit the electrical impulse to the non-specialised ventricular myocardium. The passage of the impulse through the ventricular conduction system is rapid compared with the atrioventricular node and is represented by the QRS complex ($<$0.12 s, $<$120 ms) on the ECG.

Following stimulation, the myocardial cells recover their normal resting electrical potential in an active biochemical process called repolarisation. The atrial repolarisation wave is usually obscured by the QRS, but the ventricular repolarisation gives rise to the T wave. For most of the period of repolarisation the ventricles remain unresponsive (or 'refractory') to further electrical stimulation. A diagram of the conducting system and its relationship to the ECG is shown in Fig. 27.1.

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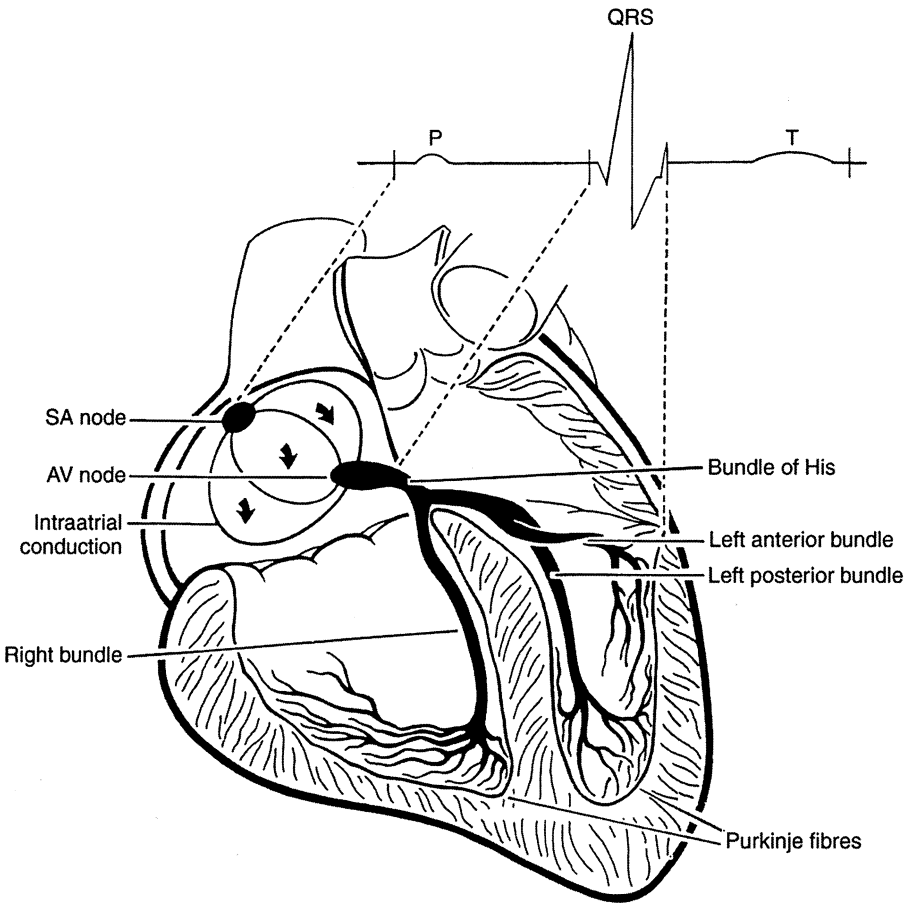

Fig. 27.1 The conduction system of the heart and its relationship to the electrocardiogram.

The T wave is sometimes followed by a U wave, which is a rounded deflection in the same direction as the T wave. Its exact genesis is unclear but it becomes more prominent in hypokalaemia, hypercalcaemia and may be inverted in ischaemic heart disease.

The QT interval is measured from the beginning of the QRS complex to the end of the T wave. Consequently it represents the total time for ventricular depolarisation and repolarisation. Nevertheless, prolongation of this interval is mainly associated with clinical conditions that delay ventricular repolarisation. This increases the period of time where the ventricles are susceptible to lethal dysrhythmias (see later).

It is important to realise that the duration of the QT interval is directly dependent on age and sex and inversely related to the heart rate. The latter is calculated by most ECG machines and expressed as the corrected QT interval so the heart rate has been considered (normal range 0.35–0.42 s, 350–420 ms).

PATHOLOGY OF THE CONDUCTING SYSTEM

The origin and spread of depolarisation through the heart can be affected by ischaemia, drugs, trauma and abnormal metabolic conditions. This can lead to spontaneous depolarisation originating from abnormal areas of the heart and spreading by an abnormal route. The ECG can be used in these situations to help locate the affected sites.

Many parts of the specialised conducting system have the ability to initiate a wave of depolarisation, and thus have potential pacemaker activity, but do so at varying rates. The eventual heart rate is determined by the part of the conducting system that has the fastest intrinsic rate of depolarisation, normally the sinoatrial node. After the sinoatrial node, the next fastest part is usually the junctional tissue near the atrioventricular node, which will then take over as the cardiac pacemaker, followed in turn by the bundle of His and ultimately the terminal fibres of the His-Purkinje system in the ventricular myocardium.

Occasionally a pathological focus develops in the heart which has a faster intrinsic rate of depolarisation when compared with the sinoatrial node. As a consequence, this focus will replace the sinoatrial node as the cardiac pacemaker.

CAUSES OF DYSRHYTHMIA

Increasing the heart rate

An increase in heart rate occurs normally as a result of emotion, exercise and fear. The effect is mediated by the sympathetic nervous system which acts on the sinoatrial node to increase its rate of depolarisation. However, an increase in the heart rate can also result from the following pathological reasons:

- automaticity
- re-entry
- both.

Automaticity

Automaticity is the ability to depolarise spontaneously. This is a common feature of cells in the conducting system and certain areas of myocardium. As mentioned previously, the sinoatrial node usually has the fastest rate of spontaneous depolarisation and therefore acts as the dominant pacemaker.

Re-entry

Re-entry occurs when there is a dual conducting system between the atria and the ventricles. This can be either within the atrioventricular node or bypassing it (e.g. Wolff–Parkinson–White and Lown–Ganong–Levine syndromes). One pathway (A) only allows conduction in a single direction (unidirectional block or a longer refractory period) and the other (B) has a slow conduction rate (Fig. 27.2). In response to a premature beat, the impulse has to go down the slow pathway (B). This is because the fast pathway (A) has not repolarised from the previous beat and is therefore unable to conduct the electrical impulse. This increase in transit time allows A to repolarise so that the impulse can be conducted opposite to the normal direction of flow. This gives B sufficient time to repolarise and so be able to be stimulated by the retrograde impulse which has travelled along A. Consequently a self-sustaining cycle of electrical impulses is created.

Both

Automaticity and re-entry can act together.

Slowing of the heart

A reduction in the heart rate is a normal physiological response during sleep, at rest, and in the athletic individual. The heart rate can also fall in certain pathological conditions when the rate of depolarisation of the intrinsic cardiac pacemaker and/or the conducting system has been reduced. Degenerative fibrosis is the most

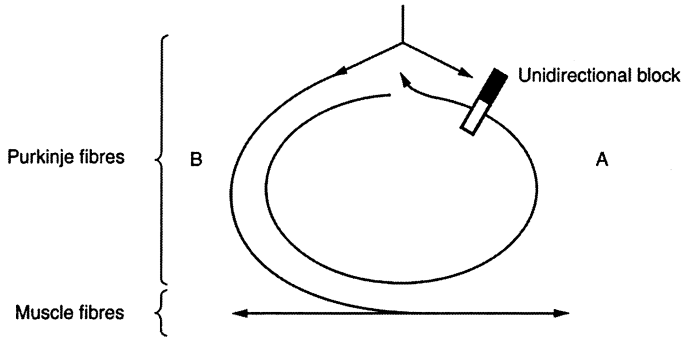

Fig. 27.2 The origin of a circus movement.

common cause but ischaemic heart disease, drugs and other diseases (e.g. hypothyroidism, jaundice) can also be responsible.

MONITORING CARDIAC ELECTRICAL ACTIVITY

In the acute situation cardiac electrical activity is usually assessed continuously by an ECG monitor connected to the patient by a standard system of electrical leads (Fig. 27.3).

Cardiac ECG monitors

Although there are many different types of cardiac monitor, the majority have common features including a screen for displaying the cardiac rhythm and a way to print a copy of it. This is commonly known as the 'rhythm strip'. Most models also incorporate a heart rate meter which is triggered by the QRS complexes and a device to automatically store a record of the ECG should the heart rate fall

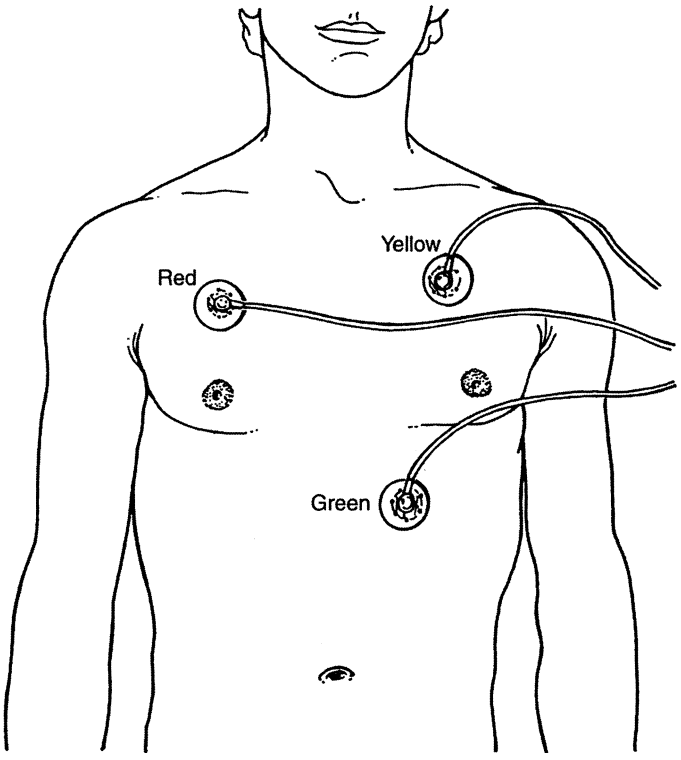

Fig. 27.3 Monitoring by the electrocardiogram.

outside certain preset limits. Lights and audible signals may also provide additional indications of the heart rate.

Modern monitoring systems are usually digital. This enables the machine to perform complex functions such as computer aided rhythm analysis, automatic and semiautomatic defibrillation, and electronic storage of the signal for review and analysis.

Leads

Lead I measures the voltage between the right and left shoulder. It gives a good view of the left lateral aspect of the heart and the QRS complex but does not necessarily give a good picture of the P wave (Fig. 27.4).

Lead II measures the voltage between the right shoulder and left leg. It is the most commonly used lead for monitoring the cardiac rhythm. As it is in line with the mean frontal cardiac axis, it gives a good view of both QRS complexes and P waves. (Fig. 27.4).

Lead III measures the voltage between the left shoulder and left lower chest (or leg). It is rarely an advantage in dysrhythmia recognition but it does give a good view of the inferior aspect of the heart (Fig. 27.4).

The remaining leads are not used during routine monitoring or the initial management of a cardiac arrest. They are, however, required for definitive dysrhythmia analysis and determining the position of the cardiac axis. The MCL1 lead measures the voltage between the right pectoral area (VI position) and the left shoulder. This gives a good view of the QRS complex and the P wave, but it is not commonly used.

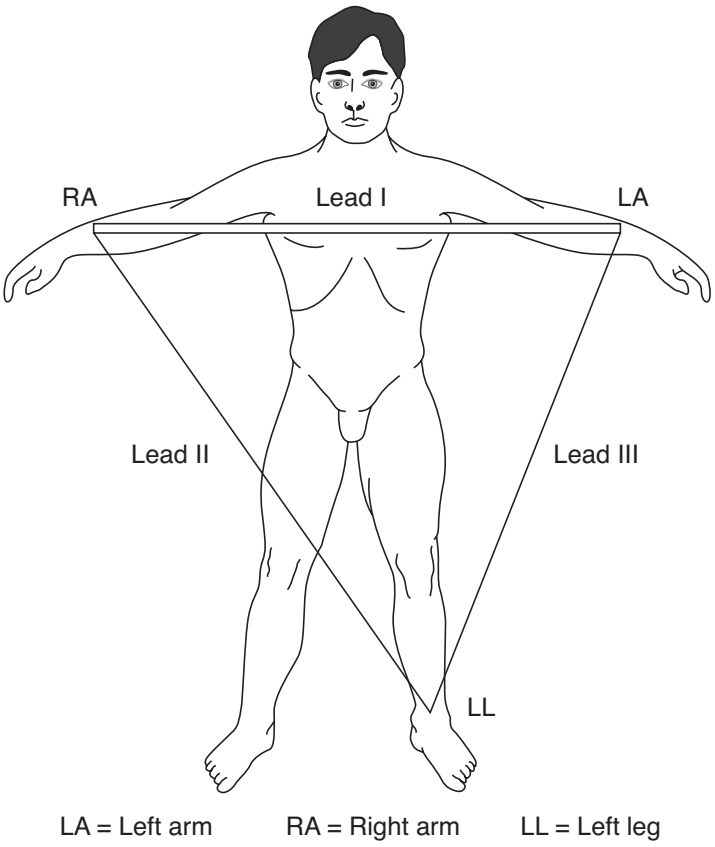

Fig. 27.4 The bipolar limb leads.

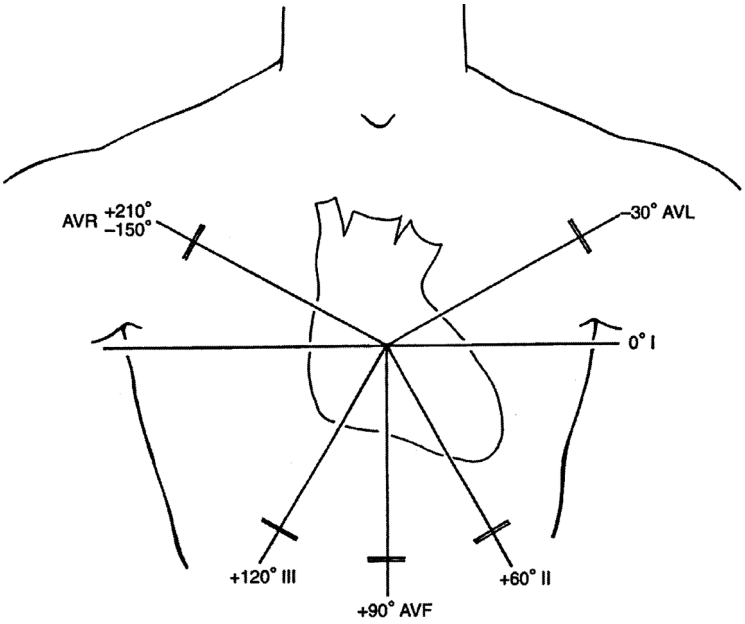

Fig. 27.5 The standard lead view of the heart.

Leads I, II, III and AVR, AVL, and AVF look at the heart in the vertical plane (Fig. 27.5).

Leads V1–6 view the heart in the horizontal plane such that V1 and V2 look at the right ventricle, V3 and V4 the interventricular septum and V5 and V6 mainly the left ventricle (Fig. 27.6).

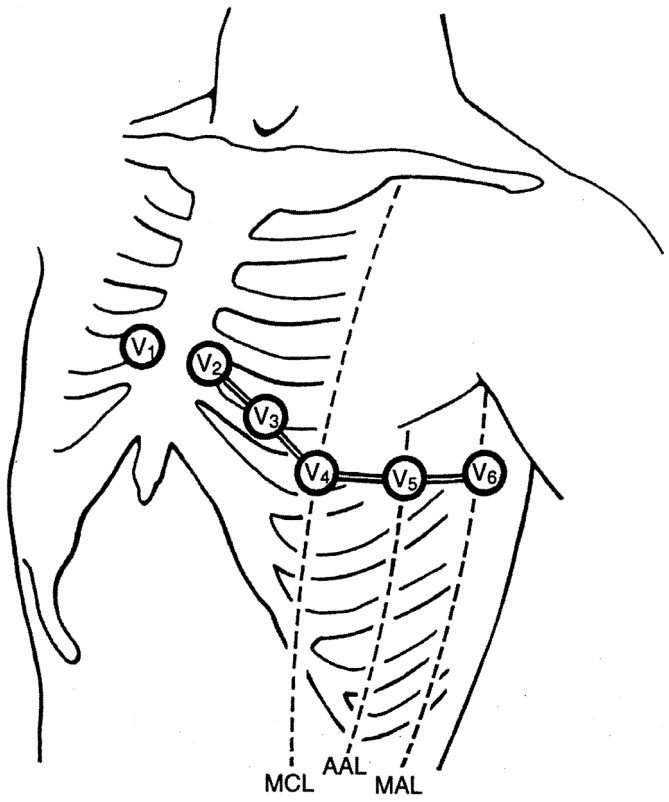

Fig. 27.6 The chest leads.

Practical points

ECG monitoring should be used on all patients presenting with chest pain, syncope, dizziness, collapse, hypotension, palpitations and cardiac arrest.

ECG machines record at a standard speed of 25 mm/s. Calibrated recording paper is used so that each large square (5 mm) is equivalent to 0.2 s (200 ms) and each small square (1 mm) is equivalent to 0.04 s (40 ms). The amplitude of the trace is standardised at 1 mV/cm and most machines have the capability of testing this (Fig. 27.7).

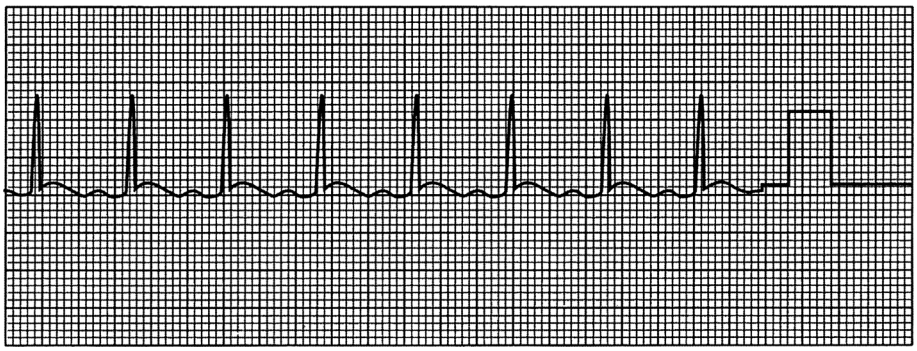

Fig. 27.7 Voltage calibration of the electrocardiogram.

To minimise electrical interference the electrodes should be all of the same type, applied over bone rather than muscle and positioned such that they are equidistant from one another. Hair should be removed from the areas where the electrodes are to be attached and the skin cleaned with alcohol to dissolve surface oil. The electrodes should be positioned on the patient's chest so that they will not interfere with any other activities, such as external cardiac massage.

Adhesive silver/silver chloride electrodes give the best signal and, if readily available, are preferable to defibrillation paddles even for the first 'quick look' in cardiac arrest. Another advantage is that paddles will only give a reading when they are in position and, therefore, are not practical for continuously assessing the rhythm. They also may fail to transmit the ECG signal following defibrillation and this may give a false impression of asystole. Nevertheless, if paddles are used, it is essential that they be placed over gel pads to ensure good electrical contact.

Ensure that the QRS height is sufficient to stimulate the rate meter by adjusting the gain control. However, this should not be so excessive as to cause artefacts on the monitor. The user needs to be aware how much the monitor trace is larger or smaller than normal.

Any activity, such as drug use or carotid sinus massage, should be recorded on the rhythm strip as it happens. This helps greatly in the later analysis of the dysrhythmia.

A common problem is artefact produced by the patient's movements, strenuous respiratory effort or if subjected to external cardiac compression. As the latter is usually sufficient to completely mask the patient's own cardiac rhythm, it must be stopped for 3–5 s so that the cardiac arrest rhythm can be analysed (see later).

It is important to realise that the leads used to monitor dysrhythmias are not the optimum ones for recording changes in the ST segment and the T wave. A 'diagnostic' setting may be required to reproduce ST displacement accurately but this produces more baseline wandering.

Ideally, old notes should be obtained and previous ECGs studied. The recording of a full 12-lead ECG should be done with any arrhythmia which does not cause loss of consciousness as this recording may provide vital evidence for future management.

A SYSTEMATIC APPROACH TO INTERPRETING A RHYTHM STRIP

Avoid the temptation to simply 'eye ball' the rhythm strip produced by the ECG monitor. Instead, develop a system so that clues and multiple problems are not missed.

Systematic approach to interpreting a rhythm strip

How is the patient?
Is there any electrical activity?
- **No** — **Asystole**
- **Yes** — **Not asystole**

Are there recognisable complexes?
- **No** — **Ventricular fibrillation**
- **Yes** — **Not ventricular fibrillation**

What is the ventricular rate?
What is the rhythm?
- **Regular**
- **Regular irregularity**
- **Irregular irregularity**

Are the P waves uniform?
- **Shape**
- **Timing – Early or later than normal**

Is there atrial flutter? (flutter waves, ventricular rate 150/min with 2:1 block)
Are there the same number of P waves as QRS complexes?
- **Yes** — **What is the PR interval?**
- **No** — **Is the PR interval constant?**
  Is the RR interval constant?

Is the QRS duration normal (less than 3 small squares, 0.12 s, 120 ms)?
- **Yes** — **Normal ventricular conduction**
- **No** — **Abnormal ventricular conduction**
  - **Shape**
  - **Timing – Early or later than normal**
  - **Frequency**

  Ventricular tachycardia:
  - **Supraventricular tachycardia with aberrant ventricular conduction**
  - ***Torsade de pointes***

  Idioventricular rhythm:
  - **Agonal rhythm**

Basic principles

It is helpful to remember the following basic principles when you are interpreting the rhythm strip.

If the depolarisation wave is moving towards the electrode then an upward (positive) deflection is seen on the monitor.

If the depolarisation wave is moving away from the electrode then a downward (negative) deflection is seen on the monitor.

The next box contains one of the many systems that have been developed to enable health care workers to interpret a rhythm strip from lead II in the acute situation. It is an effective system based on a series of questions which pick out the most life-threatening dysrhythmias first.

1. How is the patient?

Key point

Always remember: Treat the patient, not the rhythm

It is extremely important to assess the patient before making a diagnosis and suggesting a treatment from a single rhythm strip. For example, a patient who is not breathing and has no palpable pulse is suffering from a cardiorespiratory arrest irrespective of what the monitor shows. For example, if the arrest occurs despite normal (or near normal) electrical activity then pulseless electrical activity (PEA; previously called electro-mechanical dissociation) exists.

2. Is there any electrical activity?

If there is no electrical activity, check:

- connections to monitor
  to patient
- QRS gain
- leads I and III.

If there is still no electrical activity diagnose asystole (Fig. 27.8). However, beware that a completely flat tracing, without any baseline wandering, is usually caused by not connecting the patient's leads.

Occasionally P waves can be detected, indicating that atrial activity is still present (ventricular standstill, P wave asystole). This may occurs, transiently, shortly after the onset of ventricular asystole, or during Stokes–Adams syncope, and is associated with a better prognosis than when P waves are absent.

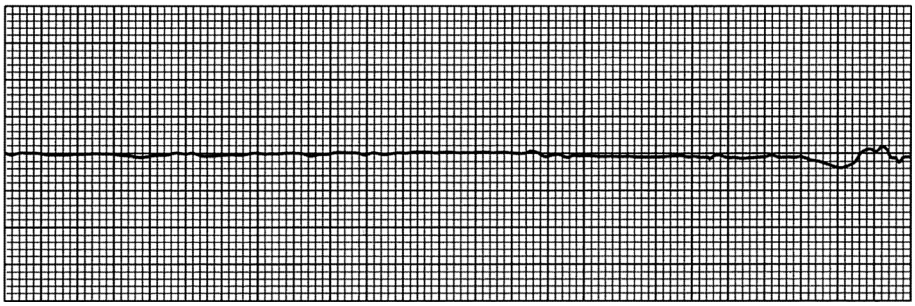

Fig. 27.8 Asystole.

3. Are there any recognisable complexes?

If there are no recognisable complexes, diagnose ventricular fibrillation (Fig. 27.9).

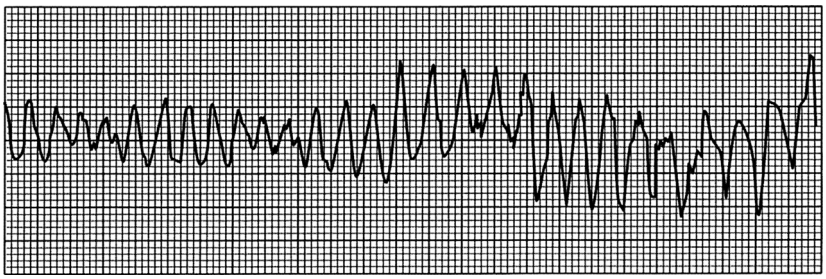

Fig. 27.9 Ventricular fibrillation.

Ventricular fibrillation is a totally chaotic rhythm because small areas of the myocardium depolarise in a random fashion. Initially, the amplitude of the waveform is large and the dysrhythmia is known as 'coarse ventricular fibrillation'. Over time 'fine ventricular fibrillation' develops because the amplitude diminishes and the tracing becomes flatter. Eventually asystole results.

It may be difficult to determine when the patient has made the transition from fine ventricular fibrillation to asystole. This is made more difficult by the presence of any baseline wandering and electrical interference. In such cases the rhythm should be reassessed taking the precautions listed for asystole. In addition, all contact with the patient should cease briefly (less than 5 s) so that a reliable tracing can be gained without interference.

Key point

The presence or absence of the commonest cardiac arrest rhythms will have been determined by answering these first three questions. As these require immediate treatment further interpretation of the ECG assumes that these rhythms have been excluded

4. What is the ventricular rate?

This can be calculated as follows:

Ventricular rate = 300/number of large squares between consecutive R waves

Fig. 27.10 demonstrates a ventricular rate of 75/min.

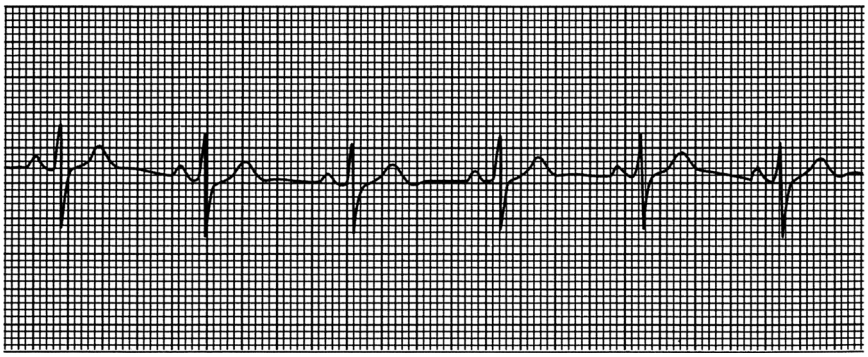

Fig. 27.10 Sinus rhythm.

Any rhythm that has a ventricular rate greater than 100 is called a 'tachycardia'. The most common tachycardia is sinus tachycardia which has, by definition, one P wave before each QRS and usually has a rate of 100–130 beats/min (Fig. 27.11) and is due to physiological causes. In contrast, a ventricular rate less than 60/min is called a bradycardia. Sinus bradycardia is a common type of bradycardia that has a P wave before each QRS.

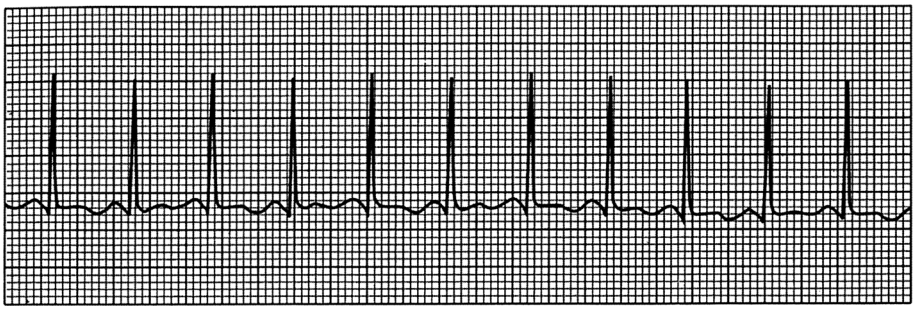

Fig. 27.11 Sinus tachycardia.

Supraventricular tachycardias (SVT) (Fig. 27.12) can be divided into two groups depending on whether it results from re-entry or enhanced automaticity. However, in the absence of finding an atrial premature beat (see later) before the tachycardia starts, it is not possible on routine ECG monitoring to distinguish between these two mechanisms.

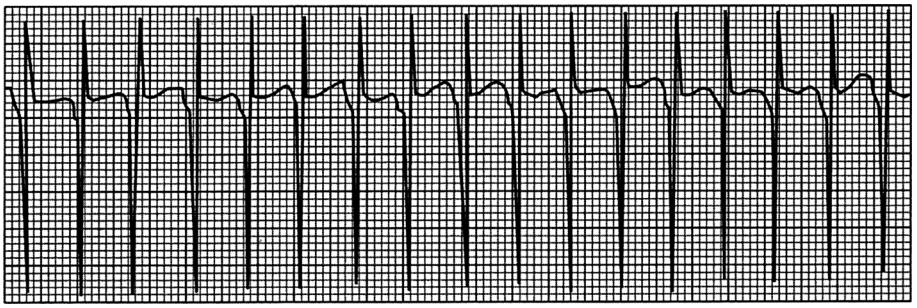

Fig. 27.12 Supraventricular tachycardia.

In a supraventricular tachycardia, the QRS complexes are narrow unless there is an aberrant conduction through the ventricles producing widening of the QRS complex (see later).

5. What is the rhythm?

To answer this question correctly it is important to inspect carefully an adequate length of the rhythm strip tracing. In this way it will be possible to detect subtle variations in rhythm.

Assessment of the regularity of the rhythm is made by comparing the RR intervals of adjacent beats at different places in the tracing. Callipers or dividers are very useful for this but it is also possible to obtain an accurate result by marking the peaks of four adjacent R waves on a piece of paper. This must be done

precisely because rhythm irregularity becomes less marked as the heart rate increases. The paper is then moved along the strip to see if the RR gaps correspond. If they do then the rhythm is regular. As interpretation of a fast heart rate can be difficult, a further rhythm strip recorded during carotid sinus massage may help by temporarily slowing the heart rate.

Sinus rhythm is diagnosed when:

- the P waves have a normal duration (2–3 small squares, 0.08–0.12 s or 80–120 ms)
- the PR interval has a normal and consistent duration (3–5 small squares, 0.12–0.20 s or 120–200 ms)
- the heart rate is between 60 and 100/min
- a P wave precedes each QRS complex.

Usually successive RR intervals are constant but occasionally, in healthy young individuals, the RR interval varies with respiration. Nevertheless the P wave shape and PR interval remain the same. This variation in the RR interval is called sinus arrhythmia and results from the inhibition of the cardioinhibitory centre during inspiration causing the heart rate to increase. The opposite occurs during expiration.

If the RR interval is irregular, it is important to decide whether there is either an 'irregular irregularity' with no recognisable pattern or RR intervals of 'regular irregularity', when the variation in the RR intervals repeats in a regular fashion. In the latter case the relationship between the P waves and the QRS waves assumes special importance and will be discussed in greater detail later.

When an irregular irregularity in the RR interval is associated with a constant QRS shape, the likely diagnosis is atrial fibrillation (AF) (Fig. 27.13). Atrial fibrillation is due to atrial depolarisation in a disorganised fashion at a rate of 350–600/min with conduction through the atrioventricular node occurring at an irregular rate. There are no P waves with AF, but the baseline may vary between fine and course fluctuations in different parts of the strip.

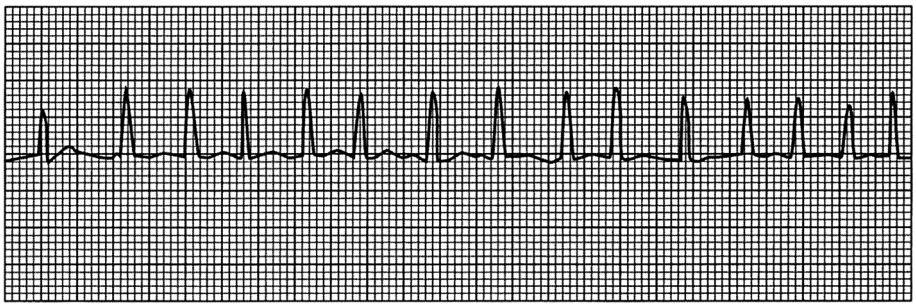

Fig. 27.13 Atrial fibrillation.

6. Are the P waves uniform?

Normal P waves have a duration of 2–3 small squares (0.08–0.12 s or 80–120 ms.) and a vertical deflection of less than 2.5 mm. They can be distinguished from the larger T waves.

It is important to check the whole strip for P waves because they may be hidden in the QRS complex or T wave, producing inconsistent and abnormal 'lumps and bumps' (Fig. 27.14). Repeating the tracing using a different lead (V1, MCL1 or III) can also help identify apparently missing P waves.

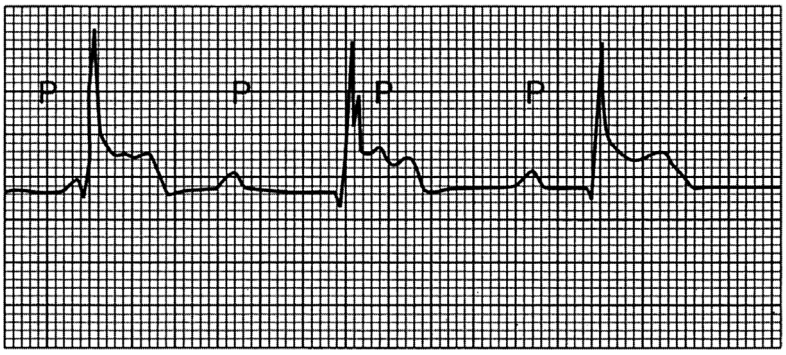

Fig. 27.14 P waves hidden in the QRS complex.

Occasionally, hidden P waves can be revealed in patients with a regular tachycardia by slowing the ventricular rate by either vagal stimulation (e.g. carotid sinus massage) or drugs (see atrial flutter)

Abnormal shaped P (i.e. 'ectopic') waves indicate that the direction of depolarisation through the atria is abnormal and consequently has not been initiated by the sinoatrial node. They have two possible sources.

Premature beats: As these usually originate in the atria and, rarely, from the atrioventricular junction they are known as atrial and junctional premature beats, respectively.

A distinguishing feature of a premature beat is the coupling interval, i.e. the time period between the normal P wave and the abnormal one (P'). This is shorter than that between two normal P waves (PP) because the myocardial focus giving rise to the premature atrial beat, depolarises before the sinoatrial node (Fig. 27.15). The coupling interval is constant if the premature beat is always produced from the same focus.

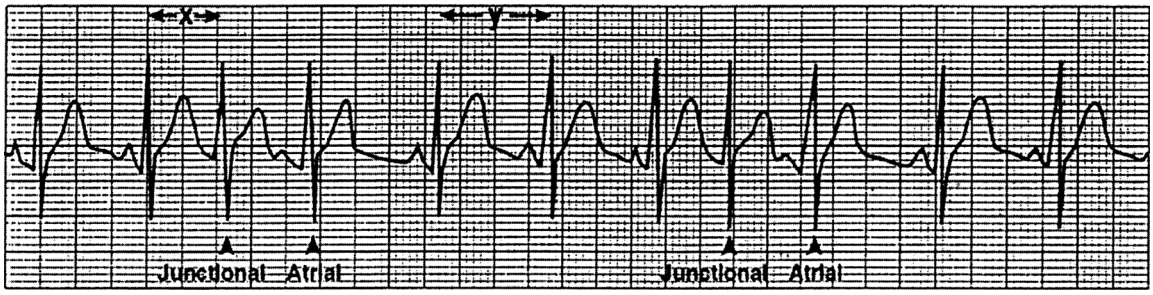

Fig. 27.15 Ectopic P waves.

The premature P wave (P') (Fig. 27.15) blocks the SAN from discharging and so disturbs the subsequent rhythm of P wave production. This can be demonstrated on the rhythm strip by noting the interval between the normal P waves on either side of the ectopic beat. This distance (*X*) is less than twice the normal PP interval (*Y*).

The abnormal focus may produce single or multiple premature beats. A tachycardia is defined as having three or more such beats occurring in rapid succession. If they occur in discrete self-terminating runs, they are described as being paroxysmal. When they occur in longer runs, the abnormal focus may take over completely and not allow any normal (SAN-generated) P waves to occur for a prolonged period of time. In these cases it is important to study the whole rhythm strip to determine if a normal PP interval can be found.

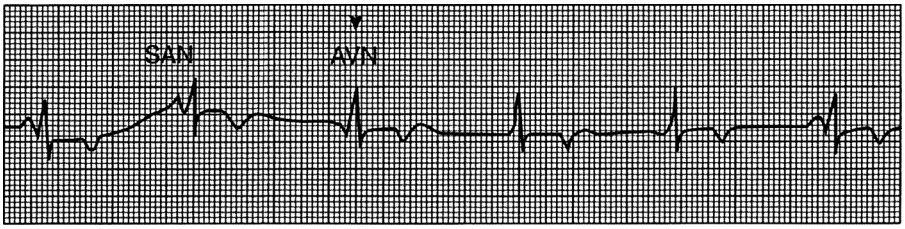

Fig. 27.16 Escape P waves.

Escape beats: If the sinoatrial node fails to generate electrical impulse then another part of the conduction system will discharge instead. This gives rise to an escape beat (Fig. 27.16). As it occurs later than expected, the coupling interval between the normal and escape P wave is longer than the normal (SAN-generated) PP interval.

Key points

Premature beat	**Reduced coupling interval**
Escape beat	**Increased coupling interval**

7. Is there atrial flutter?

In this condition the atria are depolarising at 250–350 beats/min but in most cases the rate is very close to 300 beats/min (i.e. one per large square). This atrial activity gives rise to regular 'F' (flutter) waves which gives the baseline a characteristic 'saw-tooth' appearance (Fig. 27.17).

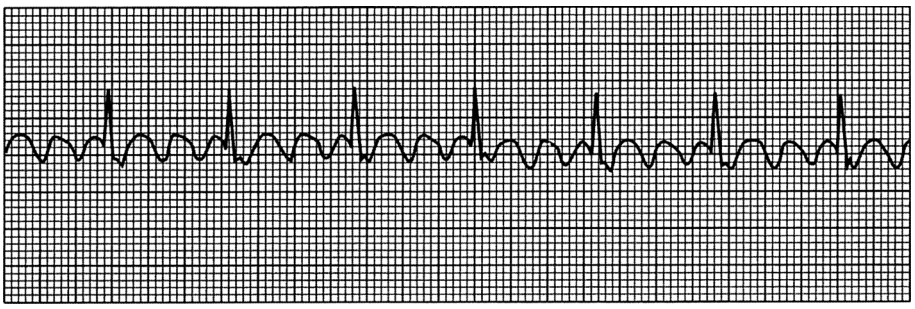

Fig. 27.17 Atrial flutter.

Only rarely does the atrio-ventricular node conduct all the atrial impulses to the ventricles, and atrial flutter is often associated with 2:1 atrio-ventricular block, giving a typical ventricular rate of 150/min, with flutter waves largely hidden in the QRS and T waves making the diagnosis difficult. Any regular tachycardia of 150/min should raise the suspicion of atrial flutter. When there are higher degrees of atrio-ventricular block flutter waves are more evident facilitating the diagnosis. Nevertheless the QRS complexes, which result, have a normal shape if the remaining part of the conduction system has not been altered. In cases where the diagnosis is in doubt, vagal stimulation can be used to increase temporarily the degree of atrioventricular node block so that flutter waves can be seen.

8. Are the number of P and QRS waves the same?

If the number of P and QRS waves are the same, measure the PR interval.

First-degree heart block

There are the same number of P waves as QRS complexes but the PR interval is constant and longer than 1 large square, 0.2 s (200 ms) (Fig. 27.18). This condition is an ECG diagnosis and generally does not progress to more serious forms of heart block. It can, however, result from digoxin and β blockers.

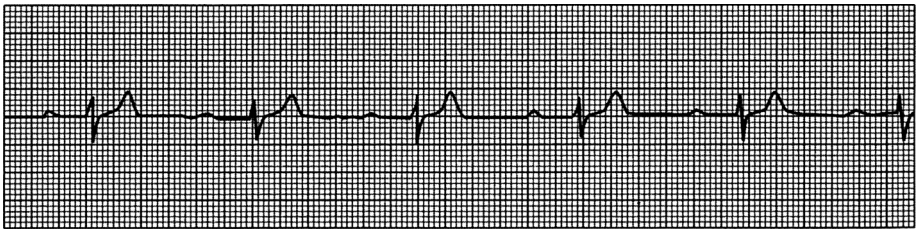

Fig. 27.18 First-degree block.

If the number of P waves is greater than the number of QRS complexes then the patient has either second- or third-degree heart block. To distinguish between them the PR interval must be examined.

Second-degree heart block – Mobitz type I (Wenckebach)

The PR interval progressively lengthens, until a P wave is not followed by a QRS complex. The atrioventricular node then recovers and the next PR interval reverts to the previous shortest conduction time. This rhythm is therefore distinguished by having both varying PR and RR intervals (Fig. 27.19). In some cases this phenomenon is physiological; in others, however, it can be the result of inferior myocardial infarction, digoxin or myocarditis.

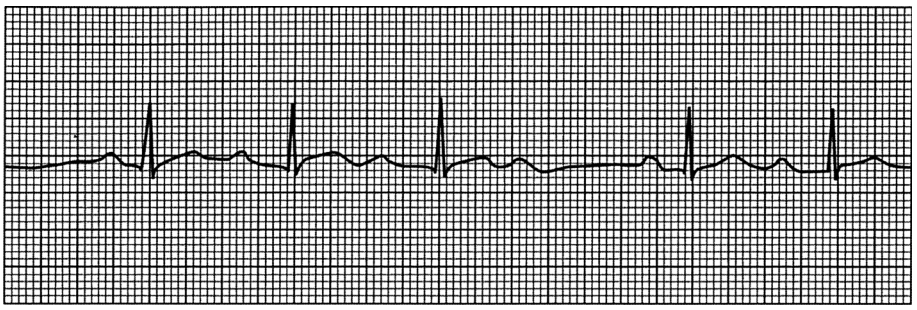

Fig. 27.19 Second-degree block – Mobitz type I.

Second-degree heart block – Mobitz type II

There is an intermittent non-conduction of some P waves but the PR interval remains constant (Fig. 27.20). However, it may be of a normal or prolonged duration. Mobitz type II is much more likely to progress to third-degree heart block

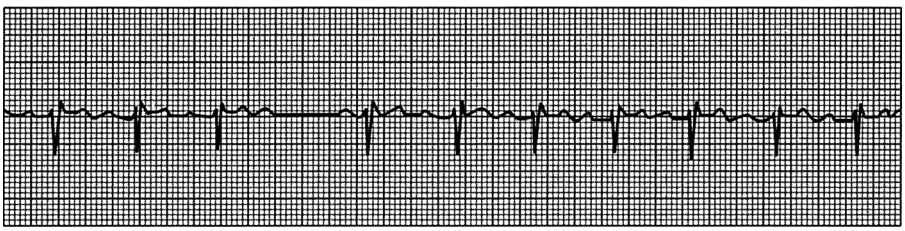

Fig. 27.20 Second-degree block – Mobitz type II.

than type I and there is a higher chance of developing asystole or ventricular dysrhythmias (see later). It is usually associated with a broad QRS complex due to bundle branch block.

Third-degree (complete heart block)

This results in total dissociation between the depolarisation of the atria and the ventricles with each beating independently (Fig. 27.21). As a consequence, there is no consistent relationship between the P waves and the QRS complexes on the ECG trace. The PR interval is, therefore, completely erratic but the PP and RR intervals are constant. Where complete heart block is associated with broad QRS complexes there is a greater risk of ventricular standstill.

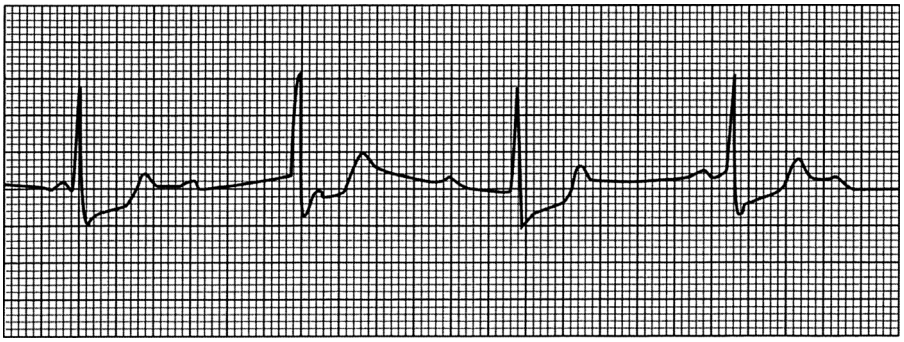

Fig. 27.21 Third-degree (complete) block.

Summary

The different types of heart block are summarised in Table 27.1.

Table 27.1 The different types of heart block

Block	P:QRS	PR interval	RR interval
First degree	Equal	Constant and prolonged	Constant
Second degree – type I	P > QRS	Variable	Variable
Second degree – type II	P > QRS	Constant	Variable
Third degree (complete)	P > QRS	Variable	Constant

9. Is the QRS duration normal?

The normal duration for the QRS is less than 3 small squares, 0.12 s (120 ms) or less. This can only occur if the ventricular depolarisation originates from above the bifurcation of the bundle of His. Broader complexes occur as a result of:

- ventricular premature beats
- ventricular escape beats
- bundle branch blocks
- left ventricular hypertrophy
- aberrant conduction.

Ventricular premature beats or ectopics

Ventricular premature beats present as bizarre, wide complexes with abnormal ST and T waves (Fig. 27.22). Unlike the normal situation, ventricular depolarisation

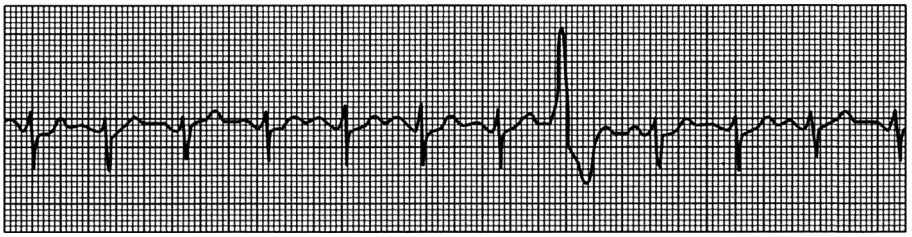

Fig. 27.22 Ventricular ectopic.

is premature thereby reducing the interval between the normal and abnormal beats (RR interval).

In contrast to atrial premature beats, ventricular premature beats neither alter nor reset the sinoatrial node. Consequently, the frequency of the P waves will continue undisturbed by the abnormal ventricular activity. There is, therefore, usually a compensatory pause after the ventricular premature beat. As a result the next P wave occurs at the normal time.

A ventricular premature beat discharging during the repolarisation phase of the ventricle runs the risk of precipitating ventricular fibrillation. The chances of this are thought to be higher if the beat occurs close to the T wave. This is known as the R-on-T phenomenon (Fig. 27.23).

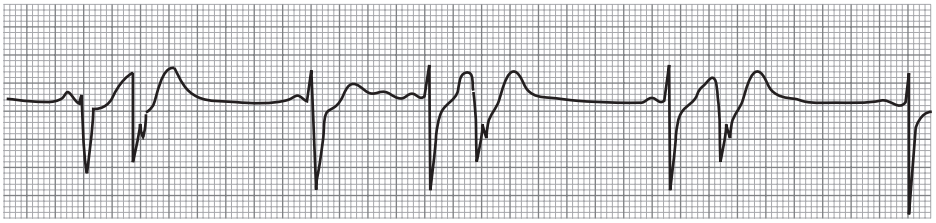

Fig. 27.23 R-on-T ectopic.

When there is more than one ventricular premature beat, specific terms are used if other features exist.

Multifocal ventricular ectopics are seen when the ventricular premature beats vary in shape from beat to beat (Fig. 27.24). This may or may not represent a

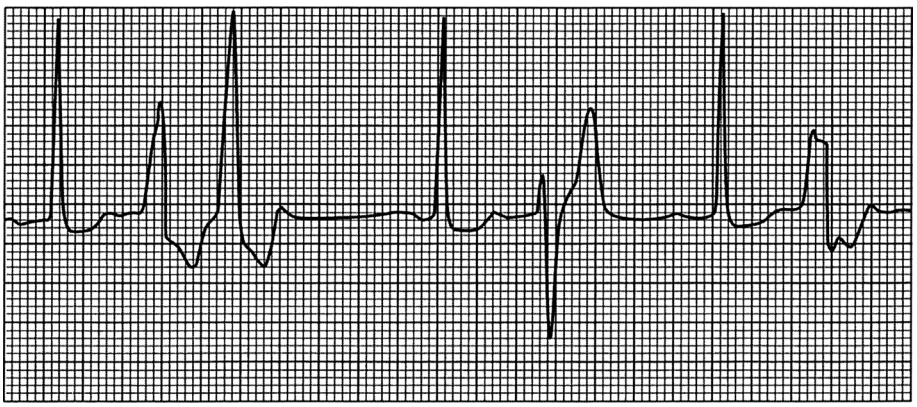

Fig. 27.24 Multifocal ventricular ectopics.

number of separate foci but it does indicate a significant increase in ventricular excitability and a higher chance of deteriorating into ventricular fibrillation.

Bigeminy is seen when a normal QRS complex is followed by a ventricular premature beat (Fig. 27.25).

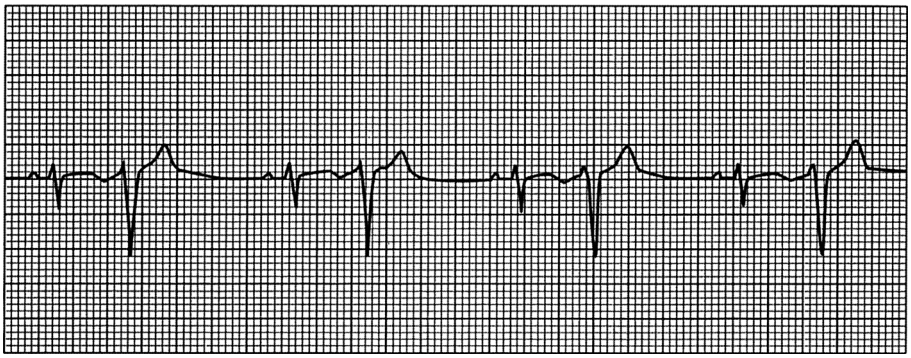

Fig. 27.25 Bigeminy.

Trigeminy occurs when two normal consecutive QRS complexes are followed by a ventricular premature beat (Fig. 27.26).

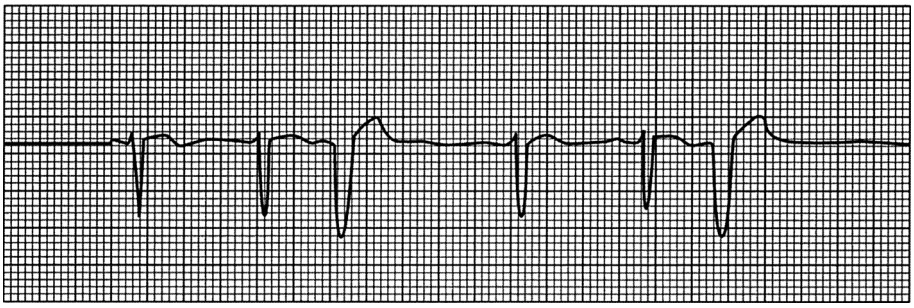

Fig. 27.26 Trigeminy.

A couplet is seen when there are two ventricular ectopic beats in a row (Fig. 27.27).

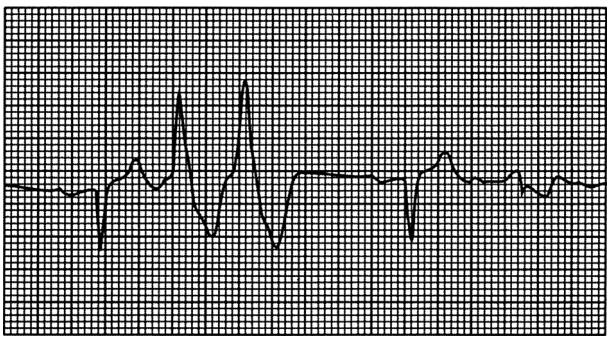

Fig. 27.27 Couplets.

Ventricular escape beats

These occur when the sinoatrial node and atrioventricular node can no longer generate an electrical impulse or stimulate the ventricles. In such circumstances the ventricles have to rely on their own intrinsic pacemaker (see earlier in the chapter). Consequently the heart rate is slow and the RR interval is longer than

normal. If P waves exist they do not have any connection to the QRS (see 'Third-degree heart block').

The QRS complex can be narrow or wide depending on where the source of the ventricular pacemaker is located. An escape focus near the atrioventricular node will result in a rate of around 50/min with narrow complexes as they are conducted via the bundle of His. This can result from congenital abnormalities but is also associated with inferior myocardial infarction.

A ventricular focus from a more distal site in the atrioventricular node will produce an intrinsic rate of around 30/min (see later in this chapter). However, the QRS complexes will be wide (3 small squares, 0.12 s, 120 ms or more) because conduction through the ventricles is not by the normal pathway. This can result from congenital abnormalities as well as from anterior and inferior myocardial infarction. These patients have a worse prognosis than those with a narrow QRS as they have a greater risk of ventricular standstill.

Aberrant conduction with supraventricular premature stimulation

The QRS is abnormal because the premature atrial impulse gets to either the atrioventricular node or the ventricles before they have had a chance to repolarise. Consequently, the conduction through the ventricles is abnormal and the resulting QRS complex is broad and abnormal in shape. Occasionally the shape of the QRS varies from beat to beat because the conduction pathway through the ventricles is not consistent. The PR interval is normal or slightly prolonged in this condition.

10. Is there ventricular tachycardia?

This occurs when there are three or more consecutive ventricular premature beats, with a rate greater than 100/min. It is said to be sustained if it lasts more than 30 s (Fig. 27.28). Ventricular tachycardia produces a regular, or almost regular, rhythm with a constantly abnormally wide QRS complex. The rate is usually between 140 and 280 beats/min. This may be slower when the patient is treated with an anti-arrhythmic drug, particularly amiodarone, which may slow the rate rather than correct the abnormal rhythm.

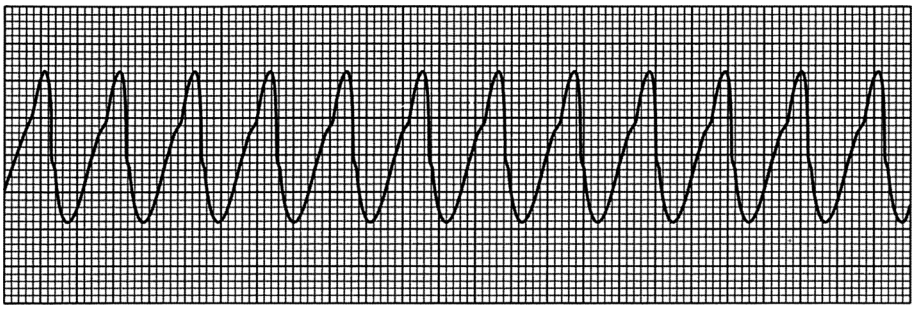

Fig. 27.28 Ventricular tachycardia.

A regular, broad QRS complex tachycardia can also be due to a supraventricular rhythm with an aberrant conduction, but this is rare. A broad complex tachycardia is ventricular tachycardia until proven otherwise. Occasionally the abnormal QRS complexes exist before the supraventricular tachycardia starts and the increase in rate simply reflects the increase in rate of stimulation from the atria. Alternatively, the complexes may only become abnormal once the supraventricular tachycardia starts. In these circumstances the normal conducting system cannot repolarise quickly enough for the new wave of depolarisation from the atria. As a

consequence the ventricles depolarise abnormally, manifested as a bundle branch block pattern and this is reflected in the abnormal QRS shape.

Distinguishing between ventricular tachycardia and supraventricular tachycardia with aberrant conduction can be difficult. A search must therefore be made for specific features.

The following favour VT:

- Evidence of independent P wave activity, including capture beats and fusion beats
- Concordant QRS polarity in chest leads (all QRS deflections in the chest leads in the same direction —- up or down)
- Wide QRS complex: greater than $3^1/_2$ small squares, 0.14 s (140 ms)

The following favour SVT with aberrant conduction:

- QRS morphology shows clear bundle branch block pattern.
- QRS morphology shows no change in pattern from that in sinus rhythm.

The most sensitive discriminator however is the patients past history, particularly of a previous myocardial infarction. It is perverse to assume that a patient who has previously suffered a myocardial infarct and who now presents with a broad complex tachycardia has developed, late in life, an unusual variant of supraventricular tachycardia rather than a typical ventricular dysrhythmia which might be expected given the previous cardiac history.

Fusion beats are produced when the atrial electrical impulse partially depolarises the ventricular muscle, which is also partially depolarised by the ventricular premature beat. The result is a normal P wave followed by an abnormal QRS complex. Capture beats occur in the context of atrioventricular dissociation, when the atrial electrical impulse completely depolarises the ventricle before it is depolarised by the ventricular premature beat. The effect is a normal QRS complex in the midst of the sequence of broad QRS complexes. Both fusion and capture beats are seen most frequently when the rate of the tachycardia is relatively slow.

Further clues as to the origin of the broad complex tachycardia come from studying the patient's 12-lead ECG, previous ECG tracings and medical notes. It is, therefore, essential that attempts be made to obtain these records. However, even after careful ECG evaluation it may still be impossible to distinguish between ventricular tachycardia and a supraventricular tachycardia with an aberrant conduction. In these cases, and especially after myocardial infarction, it is always safer to assume a ventricular origin for a broad complex tachycardia.

11. Is there *torsade de pointes* (polymorphic ventricular tachycardia)?

This is a type of ventricular tachycardia where the cardiac axis is constantly changing in a regular fashion (Fig. 27.29 – see page 429).

Torsade de pointes can occur spontaneously, but it can also result from ischaemic heart disease, hypokalaemia and certain drugs which prolong the QT interval including tricyclic antidepressants and class Ia and III anti-arrhythmic agents. This condition can end spontaneously or degenerate into ventricular fibrillation. Interestingly, ventricular fibrillation may have a similar pattern, particularly soon after its onset, but this is usually short lived.

Furthermore, ventricular fibrillation has a far more random appearance and greater variability in QRS morphology.

Atrial fibrillation in the presence of a Wolff–Parkinson–White type of accessory pathway that bypasses the atrioventricular node, may permit rapid transmission of atrial impulses to the ventricles. The resulting broad complex tachycardia may have a ventricular rate so fast that cardiac output falls dramatically, and the

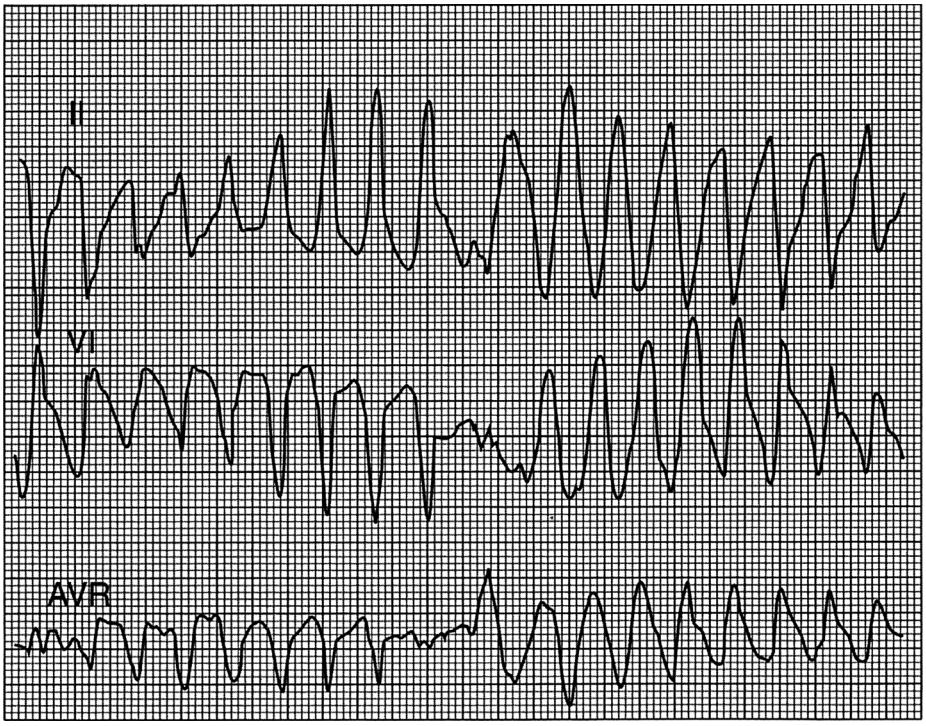

Fig. 27.29 *Torsade de pointes*, showing axis change.

rhythm can decay into ventricular fibrillation. It is one of the causes of sudden cardiac death in young people. The ECG appearances of this pre-excited atrial fibrillation are of a very rapid but irregular, broad complex tachycardia that may show marked variability in the QRS complexes. However, the QRS complexes do not show the twisting axis characteristic of *torsade de pointes*. Furthermore, it is often possible to recognise occasional normally conducted beats.

12. Is there an idioventricular rhythm?

This occurs when the ventricles have taken over as the cardiac pacemaker due to failure of the sinoatrial node, atria or atrioventricular node. In view of the ventricles' slow intrinsic rate of depolarisation the heart rate is usually slow (<40 beats/min). However, it can be accelerated and produce rates up to 100 beats/min.

Acute idioventricular rhythm commonly occurs after a myocardial infarction.

An agonal rhythm is characterised by the presence of slow, irregular, wide ventricular complexes of varying morphology. This rhythm is usually seen during the latter stages of unsuccessful resuscitation attempts. The complexes gradually slow and often become progressively broader before all recognisable electrical activity is lost.

SUMMARY

The electrical activity of the heart is highly organised and monitoring of this activity should be done in a particular way to minimise the chances of artefacts. By using a systematic approach it is possible to interpret ECG rhythm strips effectively. It is important to remember to treat the patient and not the monitor.

Advanced Life Support Group

CHAPTER 28
Chest X-ray interpretation

OBJECTIVES

After reading this chapter you will be able to:
- describe an effective system for non-radiologists to interpret chest X-rays.

INTRODUCTION

To aid understanding, the chapter will begin by reviewing important anatomical features and considering how each particular area can be affected.

It is important that a systematic method is followed when studying any X-ray, so that subtle and multiple pathologies are not missed. Once the patient's details have been checked use the system recommended for chest X-ray interpretation shown in the box.

The 'AABCS' approach to radiographic interpretation

A – Adequacy
A – Alignment
B – Bones
C – Cartilage and joints
S – Soft tissue

Most patients require only one good quality film and the ideal is a posteroanterior view taken in the X-ray department. The clinical condition of acutely ill patients usually prohibits this. Thus an antero-posterior view is taken in the resuscitation room or on the ward. This enables monitoring and treatment to be continued because the film cartridge is placed behind the patient lying on the trolley or bed. However, the apparent dimensions of the heart shadow are altered as a result of this projection.

INTERPRETATION OF FRONTAL CHEST RADIOGRAPHS

Adequacy

Check the patient details are correct and the date and time of the study.

Having made sure it is the correct radiograph, check the side marker and look for any details written or stamped on the film. This will tell you if the film is posteroanterior or anteroposterior, erect, semi-erect or supine. Assess the exposure, by looking at the midthoracic intervertebral discs and noting if they are just visible through the mediastinal density. In overexposed films all the intervertebral discs are seen and the radiograph appears generally blacker. In contrast, underexposure gives rise to poor definition of structures and boundaries.

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The film should show the lung apices and bases including the costophrenic recesses, the lateral borders of the ribs and peripheral soft tissues. The right hemidiaphragm should reach the anterior end of the right sixth/seventh rib or the ninth/tenth rib posteriorly on full inspiration. Poor inspiration (diaphragm higher than anterior fifth rib) affects the lower zone vessels such that they are compressed and appear more prominent. This in turn leads to vague lower zone shadowing. In addition, the heart appears enlarged because the diaphragms are high and the heart lies more horizontally.

Alignment

This is determined by looking at the relationship between the spinous processes of the upper thoracic vertebrae and the medial aspects of the clavicles. The ends of both clavicles should be equidistant from the central spinous process.

As with adequacy, alignment of the patient to the X-ray can significantly alter the size and shape of the chest contents on the radiograph. For example, if the patient is rotated there is distortion of the mediastinal contours as well as inequality in the transradiancy of the hemithoraces.

In addition to postural and rotational artefacts, remember the configuration of the patient's chest wall can give rise to abnormal appearances. For example, pectus excavatum can alter the size, shape and position of the mediastinum, as well as producing inequality in the transradiancy of the lungs.

Bones

The posterior, lateral and anterior aspects of each rib must be examined in detail. This can be done by tracing out the upper and lower borders of the ribs from the posterior costochondral joint to where they join the anterior costal cartilage at the midclavicular line. The internal trabecular pattern can then be assessed. Some people find this easier to do with the X-ray on its side.

Finish assessing the bones by inspecting the visible vertebrae, the clavicles, scapulae and proximal humeri. However, for full assessment specific views must be obtained.

Cartilage and joints

Calcification in the costal cartilage and larynx is common in the elderly. Occasionally the glenohumeral joints are seen on the chest X-ray. They may show either degenerative or inflammatory changes or calcification in the rotator cuff.

Soft tissue

The soft tissue can be considered in three parts:

- Mediastinum
- Lungs and diaphragm
- Extrathoracic soft tissues

Mediastinum

The mediastinum normally occupies the centre of the chest radiograph and has a well-defined margin. You should consider the upper, middle (hilar) and lower (heart) parts of the mediastinum.

Upper mediastinum

Check the position of the trachea. This should be central.

The upper left mediastinal shadow is formed by the left subclavian artery. This normally gives rise to a curved border which fades out where the vessels enter the neck. The left outer wall of the trachea is not visible in this area because

the subclavian vessels separate the trachea from the aerated lung. Inferiorly, the left paratracheal region is interrupted by both the aortic knuckle and the main pulmonary artery with the space between the two being known as the 'aortopulmonary window'. The aortic knuckle should be well defined. Note the presence of any calcification.

Middle mediastinum

The hilar shadows are produced mainly by the pulmonary arteries and veins. The major bronchi can be identified as air-containing structures but the bronchial walls are commonly only visible when seen end on. Though a contribution is made to these shadows by the hilar lymph nodes, they cannot be identified separately from the vascular shadows. The left hilum is usually higher than the right.

Any lobulation of the hilar shadow, local expansion or increase in density compared with the opposite side indicates a central mass lesion. Central enlargement of the pulmonary arteries may mimic mass lesions but the vascular enlargement is usually bilateral, accompanied by cardiomegaly and forms a branching shadow.

Lower mediastinum

The overall position, size and shape of the heart should be noted. Normally the cardiac shadow can have a transverse diameter which is up to 50% of the transverse diameter of the chest on a posteroanterior film. Cardiomyopathy or pericardial effusion can both give rise to a globular heart shadow but further diagnostic clues are usually available from the clinical history and examination.

The heart borders can then be assessed. The heart silhouette should be sharp and single with loss of a clear border indicating neighbouring lung pathology. If the borders are ill-defined then there is likely to be pathology in the adjacent lung. If the right heart border is lost then there may be right middle lobe consolidation. If the left heart border is not clear then there may be left upper lobe consolidation. A double outline suggests pneumomediastinum/pneumopericardium. With a pneumomediastinum, a translucent line can usually be seen to extend up into the neck and be accompanied by subcutaneous emphysema.

Inspection of the heart is completed by checking for calcification (valves and pericardium), prosthetic values, intravascular stents and retrocardiac abnormalities, e.g. hiatus hernia, increased density or the presence of foreign bodies and oesophageal metal stents.

Lungs and diaphragm

Lungs

These are best assessed initially by standing back from the radiograph so that you can compare the overall size and translucency of both hemithoraces. A number of changes may be seen.

Reduced volume: The commonest cause of lung volume loss is lobar collapse. When complete, these give rise to dense white shadows in specific locations and are usually accompanied by hilar displacement, increased radiolucency in the remaining lobes and reduction in the vascular pattern due to compensatory emphysema. When the collapse is incomplete, consolidation in the remaining part of the lobe is evident.

Reduced density: The transradiancy of both lungs should be equal and their outer edges should extend out to the ribs laterally and the diaphragm below. Any separation indicates that there is a pneumothorax. Within the normal lungs the only identifiable structures are blood vessels, end on bronchi and the interlobar fissures. Air trapping gives rise to increased translucency and flattening of the dome of the diaphragm. In extreme cases the mediastinum may be displaced to the contralateral side.

Increased density: There are several causes for an increase in pulmonary density. In consolidation the density is restricted to either part or all of a pulmonary lobe as a result of the air being replaced with fluid. With segmental consolidation the density is rounded and the edges blurred. When the whole of the lobe is involved the interface with neighbouring soft tissues is lost. This can lead to alteration in the outline of the heart and diaphragm depending on the location of the lobe (silhouette sign) (see earlier).

Pleural fluid is seen initially as blunting of the costophrenic angle. As more accumulates, the fluid level is easier to make out. However, if the patient is supine the fluid collects posteriorly and gives rise to a general ground-glass appearance on the affected side. Consequently, an effusion may be missed until it is large or the frontal and erect chest radiograph is carried out.

Pulmonary oedema presents as generalised fluffy air space shadowing which can be accompanied by Kerley B lines due to interstitial lymphatic congestion.

The position, configuration and thickness of the fissures should also be checked – anything more than a hairline thickness should be considered abnormal. To visualise a fissure the X-ray beam needs to be tangential; therefore, only the horizontal fissure is evident on the frontal film, and then only in 50% of the population. It runs from the right hilium to the sixth rib in the axilla. The azygos fissure is seen in approximately 1% of the population. The oblique fissures are only identified on the lateral view.

Diaphragm

The diaphragm must be checked for position, shape and clarity of the cardiophrenic and costophrenic angles. The outline of the diaphragm is normally a smooth curve with the highest point medial to the midline of the hemithorax. Lateral peaking, particularly on the right, suggests a subpulmonary effusion or a haemothorax in the appropriate clinical setting.

In the vast majority of patients the right diaphragm is higher than the left. However, elevation of either side can result from pathology in the chest (anything reducing lung volume), abdomen or damage to the phrenic nerve. In this situation the patient's history will be very helpful in distinguishing between these possible causes.

The upper surface of the diaphragm is normally clearly outlined by air in the lung except where it is in contact with the heart and pericardial fat. Loss of clarity may indicate collapse or consolidation of the lower lobe. It could also indicate diaphragmatic rupture.

Extrathoracic soft tissue

Start at the top with the neck and supraclavicular area, and continue down the lateral wall of the chest on each side. Note any foreign bodies and subcutaneous emphysema. The latter is often seen in the cervical region and appears as linear transradiancies along tissue planes. When gross it may interfere with the assessment of the underlying lung. Finally, check under the diaphragm for abnormal structures or free gas.

Presence and position of any medical equipment

The position and presence of any invasive medical equipment must be assessed while the radiograph is examined so that potential complications can be identified. It is important to check specifically that the following are correctly placed: endo-tracheal tube, CVP line, chest drain, pacing wire and naso-gastric tube. A further chest X-ray should be done after any of these devices have been placed to confirm position and exclude complications relating to insertion.

Reassess commonly missed areas

Once the system described above has been completed, it is important to re-evaluate those areas where pathology is often overlooked. These include:

- the lung apices
- behind the heart shadow
- under the diaphragm
- peripheral soft tissues.

This is particularly important if your eye has been drawn to an obvious abnormality in another area.

SUMMARY

Summary of the system for assessing frontal chest radiographs

Assess the adequacy of the film		
Patient's personal details		
Date and time of study		
Projection of the X-ray beam		
Exposure of the film (Penetration)		
Area of the chest on the film		
Degree of inspiration		
Assess the alignment of the film		
Assess the bones	Spine	
	Scapulae, clavicles	
	Humeri	
	Ribs	
Assess the cartilage and joints		
Assess the soft tissue		
Mediastinum	Upper	
	Middle (hilar)	
	Lower (heart)	
	Foreign bodies	
Lungs and diaphragm	Lungs	Size
		Density
		Fissures
		Nodules
		Opacifications
		Foreign bodies
	Diaphragm	Position
		Shape
		Clarity of the angles
		Foreign bodies
		Air – under the diaphragm
Medical Equipment	Position	
Reassess commonly missed areas of the film		
Apices		
Behind the heart		
Under the diaphragm		
Peripheral soft tissues		

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CHAPTER 29
Haematological investigations

OBJECTIVES

After reading this chapter you will be able to:
- identify which haematological tests are useful in the acute medical patient
- describe the rational use of such tests
- use test results to aid further clinical management.

INTRODUCTION

A full blood count is probably the commonest laboratory investigation that is requested because it is a 'routine test'. There is, however, no such commodity as a routine test and you should be able to justify requesting any investigation. A similar situation, though much less common, exists when requesting assessment of the components of the clotting cascade. It is, therefore, important that you critically appraise your requests and also interpret all the available information provided by a full blood count and not just the haemoglobin as often occurs.

RULES

When interpreting haematological results, always request investigations and interpret results in light of clinical findings.
- Beware of: the isolated abnormality
  bizarre results
  results that do not fit with the clinical picture.
- If in doubt:
  - Repeat the test.
  - Always seek corroborative evidence from: clinical findings
    other test results.
  - Always observe serial results for trends.

REVISION

Many haematological disorders are identified by, or suggested by, an abnormality in the full blood count. The result usually relates to three major cell lines in peripheral blood:
- Erythrocytes
- Leucocytes
- Platelets

In addition, there is a wealth of numerical information describing these cell lines that is often ignored – at the clinician's peril. This information, generated by automatic haematology counters, should be used to the clinician's advantage, hence the need for revision of some of the key cell count components.

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HAEMOGLOBIN LEVEL

The normal levels of haemoglobin are 15 ± 2 g/dl (150 ± 20 g/l) for men and 14 ± 2 g/dl (140 ± 20 g/l) for women. In the acute medical patient a raised haemoglobin often indicates dehydration or polycythaemia. The latter is commonly associated with chronic respiratory disease rather than the rare polycythaemia rubra vera. In contrast, the haemoglobin level may be low, indicating anaemia. However, remember that in patients with acute blood loss the haemoglobin level may be normal initially, until either compensatory measures fail or haemodilution occurs.

Key point

Anaemia is not a diagnosis, but a 'symptom' of an underlying disease

The red cell count is quoted by some laboratories, but this has little diagnostic value in the acute medical patient. However, the combination of haemoglobin and red cell count can be used to derive the mean cell haemoglobin (MCH). This gives a reliable indication of the amount of haemoglobin per red cell and is measured in picograms (normal range 29.5 ± 2.5 pg). The mean cell haemoglobin concentration (MCHC) represents the concentration of haemoglobin in grams per decilitre (100 ml) of erythrocytes (normal range 33 ± 1.5 g/dl). This is obtained by dividing the haemoglobin concentration by the packed cell volume. A low mean cell haemoglobin concentration is due to a low haemoglobin content in the red cell mass and indicates deficient haemoglobin synthesis. Thus the red cells will appear pale (hypochromic).

Remember that high mean cell haemoglobin concentrations do not occur in red cell disorders because the haemoglobin concentration is already near saturation point in normal red cells. The mean cell haemoglobin concentration, unlike the mean cell haemoglobin, assesses the degree of haemoglobinisation of the red cells irrespective of their size and is useful in assessing the extent of underhaemoglobinisation. The packed cell volume (PCV or haematocrit) represents a proportion (by volume) of whole blood occupied by the red cells and is expressed as a percentage (normal range for men 47 ± 7%, women 42 ± 5%). The packed cell volume or haematocrit is always elevated in polycythaemia irrespective of the cause. However, this may only be relative when haemoconcentration occurs as a result of fluid loss producing a decrease in plasma volume. The packed cell volume is therefore reduced in the presence of excess extracellular fluid and raised in fluid depletion. The mean cell volume (MCV) measured in femtolitres (normal range 85 ± 10 fl) indicates erythrocyte size. Thus, it is increased in patients with macrocytic disorders (e.g. vitamin B_{12}/folate deficiency) and reduced in the presence of microcytes (e.g. iron deficiency anaemia).

It is important to realise that red cell indices indicate the average size and degree of haemoglobinisation of red cells. They are, therefore, only of value if combined with a blood film examination that will augment the information about the relative uniformity of changes in either cell size or haemoglobin concentration.

THE BLOOD FILM

The benefits of the blood film have already been described. Some of the common terms used to describe cell morphology are listed in the next box.

Morphological terms on blood cell reports

Red cells	
Pale cells	**Hypochromia indicating defective haemoglobinisation or haemoglobin synthesis**
Macrocytes	**Large cells, abnormal red cell production, premature release, megaloblastic erythropoiesis, haemolysis**
Anisocytes	**Variation in cell size**
Poikilocytes	**Variation in cell shape**
Schistocytes/Burr cells	**Fragmented forms, usually indicate red cell trauma**
Sickle cells	**Sickling disorders**
White cells	
Hypersegmented neutrophils	**Vitamin B_{12} or folate deficiency**
Left shift neutrophils	**Neutrophils are being prematurely released**
Toxic granulation	**Increased neutrophil cytoplasmic granularity usually associated with underlying infection**
Atypical lymphocytes	**Likely viral infection**
Blast cells	**Usually indicate leukaemia**
Platelets	
Clumping	**Often causing an artificially low platelet count**

RED CELL ABNORMALITIES

Red cell abnormalities can be classified as alterations in either number or morphology.

Alteration in number

An increase in red cells is described as polycythaemia (see earlier). In contrast, anaemia is described as diminished oxygen carrying capacity of the blood due to a reduction either in the number of red cells or in the content of haemoglobin or both. This may be due to deficient red cell production and/or excessive loss. Although there is some overlap between these conditions, this classification does provide a convenient way of considering this condition (see the next box).

Another useful way of classifying anaemias is on the basis of the MCV/MCH:

Classifying anaemia

Low MCV/MCH	**Normal MCV/MCH**	**Raised MCV**
Iron deficiency	**Chronic disease**	**Vitamin B_{12} deficiency**
Chronic disease	**Acute haemorrhage**	**Folate deficiency**
Thalassaemia	**Mixed picture, e.g. iron and folate deficiency**	**Alcohol use**
		Haemolysis
	Aplastic anaemia	**Hypothyroidism**

An anaemia with a coexistent reduction in both white cells and platelets is referred to as pancytopenia. Causes of pancytopenia include aplastic anaemia, bone marrow infiltration (e.g. lymphoma, leukaemia, myelofibrosis, myeloma) and hypersplenism.

Alteration in morphology

An anaemia with reduced mean cell volume, mean cell haemoglobin and mean cell haemoglobin concentration, i.e. microcytic hypochromic anaemia, is highly suggestive of iron deficiency. Therefore a serum ferritin should be requested before treatment with iron is started.

However, if there is coexistent thrombocytosis then this type of anaemia could indicate ongoing blood loss or inflammation. If none of these conditions are evident then it is possible that the microcytic hypochromic picture is a manifestation of thalassaemia, which is rare in the UK. In contrast, an anaemia with raised mean cell volume and mean cell haemoglobin is suggestive of a variety of conditions including a deficiency in vitamin B_{12} and/or folic acid, hypothyroidism and alcohol use. An anaemia with normal mean cell volume, mean cell haemoglobin and mean cell haemoglobin concentration, i.e. a normochromic normocytic anaemia, can reflect chronic disease (e.g. inflammation, myeloma), acute blood loss or haemolysis.

Haemolysis is usually associated with a normochromic normocytic anaemia although some of the red cells can be large due to the release of a large number of immature red cells, i.e. reticulocytes. The latter can also occur following haemorrhage or in response to treatment with iron, folic acid and vitamin B_{12}. The comment polychromasia (grey/blue tint to cells) is often recorded on the full blood count indicating a reticulocyte response. A formal count of these cells can also be done.

Haemolytic anaemia is a term that describes a group of anaemias of differing cause, which are all characterised by abnormal destruction of red cells. The questions asked to identify the cause of haemolysis are shown in the box.

These questions can be used to produce a 'user-friendly' classification of haemolytic anaemia as shown in the next box.

Three key questions in the diagnosis of haemolytic anaemia

- **Is it an inherited or acquired disorder?**
- **Is the location of the abnormality within the red cells (intrinsic) or outside (extrinsic)?**
- **Are the red cells prematurely destroyed in the blood stream (intravascular) or outside in the spleen and liver (extravascular)?**

LABORATORY DIAGNOSIS OF HAEMOLYTIC ANAEMIA

The most likely clue is a normochromic normocytic anaemia with prominent reticulocytes. Other laboratory results include:

- unconjugated hyperbilirubinaemia (thus a lack of bilirubin in the urine)
- low haptoglobin (a glycoprotein that binds to free haemoglobin and is thus usually full saturated in haemolysis)

- haemoglobin and haemosiderin can be detected in the urine with intravascular haemolysis, because the haptoglobin which usually 'mops up' free haemoglobin is saturated.

Congenital disorders

More specific tests will be requested after taking a comprehensive history as this is likely to provide clues to underlying inherited disorders. The presence of hereditary spherocytosis or elliptocytosis will be seen on the blood film.

The thalassaemias are a heterogeneous group of disorders affecting haemoglobin synthesis; they will be diagnosed from the medical history, clinical examination, blood film and haemoglobin electrophoresis to identify structural haemoglobin variants. In addition, the presence of sickle cell syndromes will be diagnosed from the clinical history, in particular that of the family, and the presentation with haemolysis, vascular occlusive crises and sequestration crises. Under these circumstances the blood film is likely to show the presence of sickle shaped cells. Haemoglobin electrophoresis may reveal an abnormal haemoglobin such as in sickle cell anaemia with no detectable haemoglobin A.

The two common abnormalities of red cell metabolism resulting in haemolysis are glucose-6-phosphate dehydrogenase deficiency and pyruvate kinase deficiency. As well as the features of intravascular haemolysis described earlier, specific enzyme levels can also be measured to produce a definitive diagnosis.

Acquired disorders

Autoimmune haemolytic anaemia

Autoimmune haemolytic anaemia is a form of acquired haemolysis with a defect outside the red cell. The bone marrow produces structurally normal red cells. These are prematurely destroyed by the production of an aberrant antibody (IgM or IgG) targeted against the red cell membrane. Once the autoantibody has bound with the antigen on the red cell the exact type of haemolysis is determined by the class of antibody as well as the surface antigen. It usually involves sequestration by the spleen leading to extravascular haemolysis or activation of complement leading to intravascular haemolysis. A simple classification of these conditions is either warm or cold depending on whether the antibody reacts better with red cells at 37°C or 4°C respectively.

Warm autoimmune haemolytic anaemia

This is the commonest form of haemolytic anaemia. The erythrocytes are coated with either IgG alone or IgG and complement or complement alone. Premature destruction of red cells usually occurs in the liver and spleen. Half of all warm autoimmune haemolytic anaemias are associated with other autoimmune conditions or lymphoma. The most characteristic laboratory abnormality is a positive direct antiglobulin test (DAT or Coombs test). As a reminder the DAT is where red cells are already sensitised and have immunoglobulin bound to their surface antigens. Addition of extrinsic antihuman globulin results in haemolysis, i.e. a positive direct Coombs test. In contrast, the indirect test is where normal red cells have to be sensitised in vitro by the addition of the test serum containing red cell antibodies. Finally antihuman globulin is added. Agglutination indicates the presence of red cell antibodies, as used in red cell typing.

Cold autoimmune haemolytic anaemia

This is generally associated with an IgM antibody. Isoimmune haemolytic anaemia may occur with rhesus or ABO incompatibility, and in the context of

adult medicine this may follow a blood transfusion reaction. It can also follow infection with mycoplasma or Epstein–Barr virus.

Drug-induced haemolytic anaemia

There are three mechanisms of haemolysis:

- Drug-antibody complex (e.g. quinine) induces complement, leading to erythrocyte destruction.
- Drug-red cell complex (e.g. penicillin) acts as an antigen to which antibodies are formed, leading to erythrocyte destruction.
- A drug (e.g. methyldopa) induces production of an erythrocyte autoantibody, leading to erythrocyte destruction.

Non immune/traumatic haemolytic anaemia

Non-immune and traumatic haemolytic anaemia is most frequently manifested by a microangiopathic picture. This is one of the most frequent causes of haemolysis and describes intravascular destruction of red cells in the presence of an abnormal microcirculation. Causes of microangiopathic haemolytic anaemia are listed in the next box.

Causes of microangiopathic haemolytic anaemia

Disseminated intravascular coagulation
Valve prosthesis
Malignancy
Severe infections
Glomerulonephritis
Vasculitis
Accelerated (malignant) hypertension

WHITE CELLS

Total and differential leucocyte count

The total white count varies markedly as there is a diurnal rhythm with minimal counts occurring in the morning. This may rise during the rest of the day or following stress, eating or during the menstrual cycle.

The total leucocyte count is $(7 \pm 3) \times 10^9$/l. This comprises:

- neutrophils (2–7) $\times 10^9$/l (40–80% of total count)
- lymphocytes (1–3) $\times 10^9$/l (20–40% of total count)
- monocytes (0.2–1) $\times 10^9$/l (2–10% of total count)
- eosinophils (0.04–0.4) $\times 10^9$/l (1–6% of total count)
- basophils (0.02–0.1) $\times 10^9$/l (<2% of total count)

Disorders of leucocytes

There are many conditions that will affect both the total and differential white cell count. Common disorders are listed in Tables 29.1–29.4.

Basophils rarely have any significance in the acute medical setting.

Table 29.1 Disorders of neutrophils

Raised number	Bacterial infection
	Myeloproliferative disease
	Tissue damage
	Malignancy
	Drugs, e.g. prednisolone
Reduced numbers (production failure)	Chemicals
	Severe infection
	Drugs, e.g. carbimazole
	Marrow infiltration by malignant tumour or marrow fibrosis
	Specific deficiencies of vitamin B_{12} and folic acid
	Peripheral sequestration/hypersplenism
	Shock

Table 29.2 Disorders of lymphocytes

Raised numbers (lymphocytosis)	Viral infection, especially glandular fever*
	Chronic lymphatic leukaemia
	Typhoid fever
	Brucellosis
Reduced numbers (lymphopenia)	Corticosteriods
	Viral infections*
	Cytotoxic drugs
	Ionising radiation

*In suspected viral infections a blood film may show changes in lymphocyte numbers and morphology.

Table 29.3 Disorders of monocytes

Raised numbers	Infective endocarditis
	Typhus fever
	Malaria
	Kala-azar
	Systemic lupus erythematosus
	Certain clinical poisonings, e.g. trichloroethylene

Table 29.4 Disorders of eosinophils

Raised numbers	Parasitic infections
	Atopy including asthma and drug sensitivity
	Chronic eczema
	Malignant tumours, e.g. Hodgkin's disease

Haematological malignancies

Chronic myelocytic leukaemia

This is a myeloproliferative disease characterised by increased numbers of neutrophils and their precursors. The clinical features include weight loss, night sweats, pruritus and splenomegaly. It can occur in all age groups but the peak incidence is in middle age and it affects both sexes equally. The disease is associated with Philadelphia chromosomal translocation. Blood results show a leukocytosis, mainly neutrophils; platelet count is variable and anaemia may be a feature. There may also be a low leucocyte alkaline phosphatase score and raised urate.

Treatment consists of hydroxyurea, interferon and allopurinol to prevent gout. Allogeneic stem cell transplantation is an option for patients under 50 years of age.

Chronic lymphocytic leukaemia

This is a B-cell lymphoproliferative disease. Clinical features include lymphadenopathy, night sweats, weight loss and moderate splenomegaly. It is a disease of the elderly and is more common in men. Blood results show a lymhocytosis; anaemia and thrombocytosis may also be a feature.

Treatment consists of chemotherapy with chlorambucil and steroids.

Acute leukaemia

These leukaemias can be classified as lymhpoblastic or myeloblastic. They can occur as primary leukaemias or may follow previous chronic leukaemias that have transformed into an acute (more aggressive) type. Acute lymphoblastic leukaemia (ALL) is common in childhood.

Acute myeloblastic leukaemia is more common in the elderly. Clinical features are those of bone marrow failure and occur over a relatively short period of time. They include bruising, bleeding and infection. Lymphadenopathy and hepatosplenomegaly are common in ALL.

Treatment comprises chemotherapy and stem cell transplantation.

Lymphoma

There are two types: Hodgkin's (HL) and non-Hodgkin's (NHL).

HL is characterised by malignant cells called Reed–Sternberg cells which appear to be derived from B cells. There is an association with Epstein–Barr infection. There is a bimodal distribution of cases in young adulthood and in later life. Features include lymphadenopathy which is usually cervical and weight loss, night sweats and pruritus. Some patients complain of lymph node pain in association with alcohol consumption.

NHL is the term used to describe tumours of lymphoid tissue **without** the characteristic Reed–Sternberg cells. This disease is the most common haematological malignancy. Features include lymphadenopathy, abdominal pain, anaemia, sweating and weight loss.

Blood results usually show an anaemia (normochromic, normocytic), leucocytosis, raised ESR, raised LDH and abnormal LFTs. NHL specifically may cause a pancytopenia and a peripheral blood lymphocytosis.

Treatment for both types of lymphoma involves chemotherapy, radiotherapy and stem cell support/transplantation. There is a very complicated staging system for lymphomas which is beyond the scope of this book and treatment depends on the stage of the disease.

Multiple myeloma

Multiple myeloma is a malignant disease of plasma cells characterised by a monoclonal paraprotein in the urine/serum, boney involvement and an excess of

plasma cells in the bone marrow. It is a disease of the elderly and is slightly more common in men. Features include bone pain, hypercalcaemia, bone marrow failure, immune failure, renal failure and amyloidosis.

Blood results show anaemia and a paraprotein in the serum and/or Bence–Jones proteins in the urine; a blood film shows rouleaux and the bone marrow shows an excess of plasma cells.

Treatment may be conservative or with chemotherapy.

PLATELETS

There is a marked variation in the normal platelet count ranging from 150 to 400 $\times$ 10^9/l. Common platelet disorders are listed in the next box.

Disorders of platelets

Raised number (thrombocytosis)	**Inflammation, e.g. Crohn's disease**
	Haemorrhage
	Essential thrombocytosis (rare)
	Polycythaemia rubra vera (rare)
Reduced number (thrombocytopenia)	**Deficient production, e.g. hypoplasia**
	Replacement with leukaemic cells or fibrosis
	Dyshaemopoiesis secondary to vitamin B_{12} deficiency
	Increased destruction of platelets, e.g. drugs or autoimmune
	Sequestered in the spleen
	Increased consumption (disseminated intravascular coagulation)

Thrombocytopenia is a common finding. The risk of spontaneous haemorrhage is unusual unless the platelet count falls below 20 $\times$ 10^9/l.

COAGULATION

The physiological pathway of blood coagulation is an interlinked cascade of factors which most doctors learn for examinations. The basic principles are three activation pathways (intrinsic, extrinsic and alternative) which have a final common pathway. These pathways are summarised in Fig. 29.1 (see over).

Blood clotting is a vital defence mechanism that is regulated to ensure adequate and appropriate activation.

The major inhibitors of coagulation circulating in the plasma are:

- antithrombin III. This is the most potent inhibitor of the terminal proteins of the cascade, particularly factor X and thrombin. Its activity is greatly increased by interaction with heparin
- protein C is a vitamin K-dependent plasma protein which inactivates cofactors Va and VIIIa as well as stimulating fibrinolysis. Protein C is converted to an active enzyme from interaction with thrombin. Protein S is a cofactor for protein C
- fibrinolytic systems. This system for fibrin digestion is shown in Fig. 29.2. Fibrin clots are broken down by plasmin that is produced from plasminogen by 'activator enzymes'. Plasmin also inhibits thrombin generation, thus acting as an anticoagulant. In addition, fibrin degradation products have a similar effect.

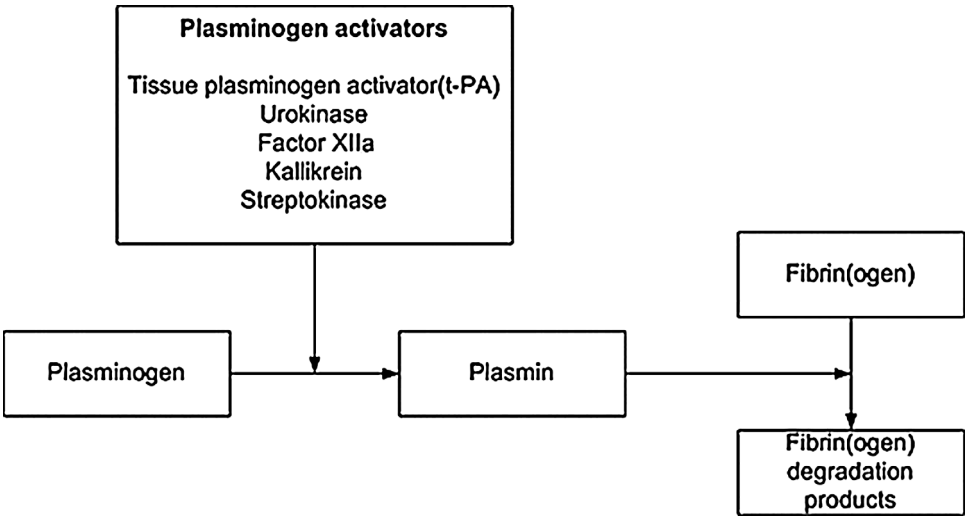

Fig. 29.1 Pathways of blood coagulation.
Streptokinase is an exogenous activator usually from B haemolytic streptococci

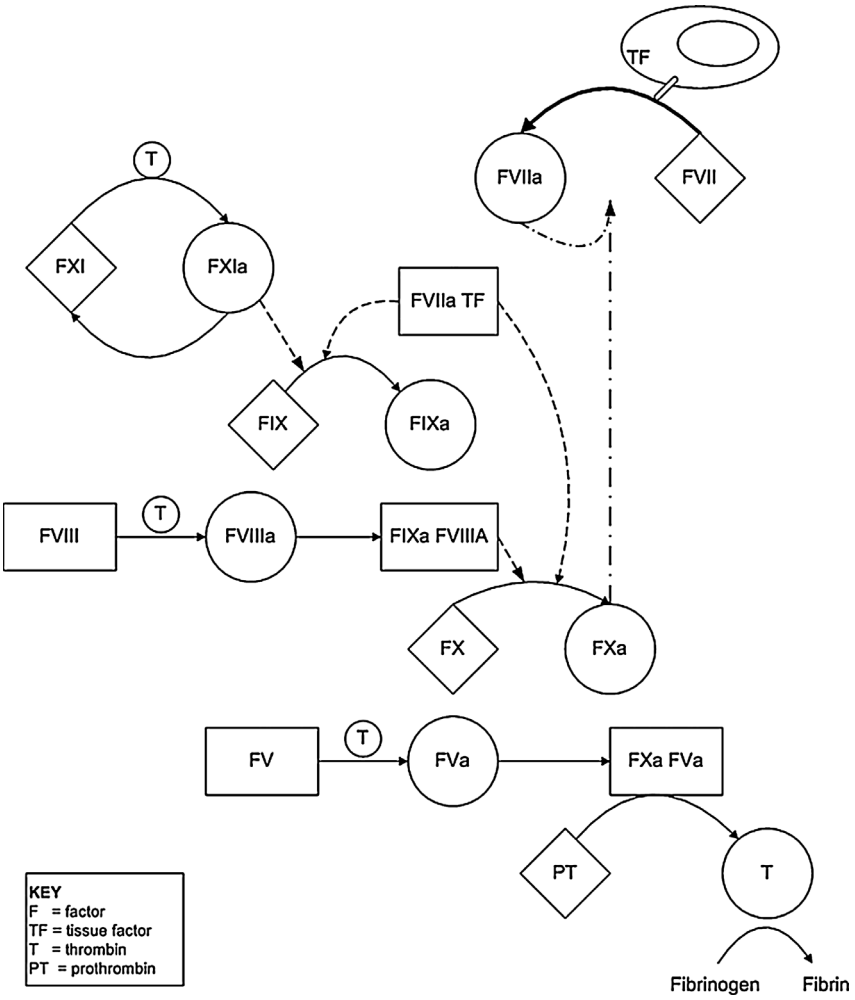

Fig. 29.2 The fibrinolytic system.
Dotted lines = enzyme action on substrates; Solid lines = conversion of protein from one active state to another

Fibrinolysis is also under strict control. Circulating plasmin is inactivated by the protease inhibitor δ2-antiplasmin.

Tests for the assessment of coagulation

In most acute medical situations, an assessment of the coagulation cascade only requires:

- prothrombin time: – a measure of the function of the extrinsic pathway
- activated partial thromboplastin time – assesses the intrinsic pathway
- assessments of fibrinolysis, e.g. fibrinogen level, fibrin degradation product level or D-dimer quantitation – are often used as markers of thromboembolism disseminated intravascular coagulation.

The common causes of prolonged prothrombin and activated partial thromboplastin times are listed in the box.

Common causes of prolonged prothrombin (PTT) and activated partial thromboplastin (APTT) times

PTT	APTT
Warfarin	Heparin
Liver disease	Haemophilia
Vitamin K deficiency	von Willebrand's disease
Disseminated intravascular coagulation	Liver disease
	Lupus anticoagulant syndrome

D-dimer

A D-dimer fragment is generated when cross linked fibrin in thrombi is broken down by plasmin. There are assays available which can detect the precence of D-dimers using monoclonal antibodies. This test is fairly non-specific and can be raised in the presence of a blood clot but also in infection/inflammation. Therefore it tends to be a more useful test if the result is negative. There is good evidence to show that a negative D-dimer test can help rule out a PE/DVT in low risk cases.

SUMMARY

A limited number of haematological investigations are required in the acutely ill medical patient. Much of the information available is often underused; therefore, a thorough understanding of the morphology and normal values of, in particular, red cells is extremely useful. A blood film is an underused investigation that can yield significant relevant information in the acute medical setting. These initial investigations, combined with a 'phrased' history, will influence the selection of subsequent haematological tests.

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CHAPTER 30
Biochemical investigations

OBJECTIVES

After reading this chapter you will be able to:

- understand the importance of interpreting urea, electrolyte and creatinine results in light of clinical findings
- systematically assess urea, electrolyte and creatinine results
- use these results to aid your clinical management.

INTRODUCTION

Urea, electrolytes and creatinine are commonly requested laboratory investigations. All too often there is little thought about why these investigations have been requested and what the abnormalities, in particular of the electrolytes, may indicate. This chapter will provide a systematic approach to the assessment of such investigations, but before this is described there are certain rules which have to be obeyed.

RULES FOR THE INTERPRETATION OF UREA, ELECTROLYTES AND CREATININE

- Always interpret the results in the light of clinical findings.
- Beware: the isolated abnormality
  bizarre results
  results that do not fit the clinical picture.
- If in doubt, repeat the test.
- Always seek corroborative evidence from: clinical findings
  other test results.
- Always observe serial results for trends.

GUIDELINES FOR INTERPRETATION OF UREA, ELECTROLYTES AND CREATININE

A review of essential facts.

Urea

Blood urea provides an assessment of glomerular function. However, it can be influenced by many exogenous factors including food intake, fluid balance, gastrointestinal haemorrhage, drugs and liver function. Normal plasma urea is 4.6–6.0 mmol/l.

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Creatinine

This provides a better indication of renal function. Plasma creatinine levels are proportional to muscle mass. Creatinine gives a reasonable indication of changes in glomerular filtration, provided the body weight remains stable. Normal plasma creatinine is 60–125 μmol/l.

Potassium

This is the most important intracellular cation and only approximately 2% of the total body potassium is found in the extracellular fluid. Normal plasma potassium is 3.5–5.0 mmol/l.

Bicarbonate

Bicarbonate is an important anion. Normal plasma bicarbonate is 24–28 mmol/l. In a venous blood sample the bicarbonate provides a useful, but crude, indication of the acid–base status.

Sodium

Sodium is the major extracellular cation and is intimately related to water balance. The normal plasma sodium is 135–145 mmol/l.

A SYSTEMATIC EXAMINATION OF UREA, ELECTROLYTES AND CREATININE

It is impossible to provide an accurate diagnosis purely by assessing a patient's urea, creatinine and electrolytes. However, this system, especially when viewed with a patient's clinical picture, will help to discriminate between many of the common conditions.

A systematic approach comprises:

- assessment of the patient's urea and creatinine
- the relationship between the urea and creatinine
- assessment of potassium, bicarbonate and sodium.

Examine the urea and creatinine; five common patterns can be seen, as described below.

All results will fall into one of these five broad categories and each will be examined.

Pattern		Diagnoses
↑↑ Urea	↑↑ Creatinine	Renal failure
↓ Urea	↓ Creatinine	Fluid overload
Urea < creatinine		Low protein diet
		Liver failure
		Dialysis
Urea > creatinine		Fluid depletion, e.g. dehydration, fever, trauma
		Drugs, e.g. diuretics
		Elevated protein, e.g. diet, gastrointestinal bleed, catabolism
Urea normal	Creatinine normal	Check for any electrolyte abnormality

Urea raised and creatinine raised

The most common diagnosis would be renal failure. Therefore confirmatory evidence should be sought.

- Check plasma potassium: This will remain normal until the glomerular rate has fallen below 10 ml/min. Hyperkalaemia is common, but can be secondary to a metabolic acidosis, catabolism or haemolysis.
- Check bicarbonate: This is often reduced reflecting the acidosis associated with uraemia or a failure of bicarbonate secretion.
- Check serum sodium: This may be normal but is often low due to overhydration and dilution. To provide further information, plasma osmolality along with urine, sodium, osmolality and urea should be measured to distinguish between acute and established renal failure.

Urea low and creatinine low

This commonly results from fluid overload.

- Check potassium, bicarbonate and sodium. Low values would be expected. The basic problem is that sodium is retained but to a significantly lesser extent than the degree of water retention. This is often referred to as hyponatraemia with clinically normal extracellular fluid volume. This commonly results from two mechanisms.
- Increased water intake, e.g. excess intravenous fluids, excess drinking (both pathological and psychological polydipsia) and water absorption during bladder irrigation.
- Inability to excrete water, e.g. SIADH, adrenocortical insufficiency, hypothyroidism and drugs that reduce renal diluting capacity, e.g. diuretics. Under these circumstances there is water retention but body sodium is normal with possibly only small increase in extracellular fluid volume which will be undetected clinically. In contrast, if both extracellular fluid sodium and water are increased, but more water is retained than sodium, hyponatraemia will result with expansion of the extracellular fluid volume producing oedema. Note that the discriminating factor between these conditions is based on the clinical presence of oedema.

Hyponatraemia with expansion of the extracellular volume occurs with cardiac, renal and liver failure. The urine sodium can provide further clues, in particular, in the patient who is hyponatraemic with an increased extracellular volume where the urine sodium is usually less than 10 mmol/l (except in renal failure).

An interesting variant is beer drinker's hyponatraemia. Beer has a low sodium content. If in excess of 5 litres is consumed daily then hyponatraemia may result, usually with a clinically normal extracellular volume.

Urea less than creatinine

Low urea in relation to the creatinine usually indicates low protein diet or rarely liver failure or post dialysis. Low urea in liver disease is usually attributed to reduced synthesis.

- Check potassium
  - normal with low protein diet and post dialysis
  - normal in liver disease unless diuretics are used
  - low in liver disease with diuretic use.

Urea greater than creatinine

These results suggest fluid depletion, e.g. associated with dehydration, fever, infection or trauma. Drugs, in particular diuretics, can induce a similar problem. An

alternative explanation is increased protein which may be from a dietary source, following a gastrointestinal haemorrhage, or secondary to catabolism.

- Check potassium
  low values would suggest gastrointestinal fluid loss
  high values are likely to indicate incipient renal failure or potassium sparing diuretic
- Check sodium
  low values indicate hyponatraemia with reduced extracellular fluid volume reflecting reduced intake (rare), usually attributed to inappropriate replacement of gastrointestinal fluid loss with 5% dextrose only.

The major cause is excessive sodium loss which is usually:

- from the gastrointestinal tract secondary to vomiting, diarrhoea, fistulae or intestinal obstruction
- from the kidney, e.g. during the diuretic phase of acute tubular necrosis
- due to excess diuretic therapy (including mannitol or the osmotic effect of hyperglycaemia)
- due to postobstructive diuresis
- due to adrenocortical insufficiency or severe alkalosis where increased urinary loss of bicarbonate necessitates an accompanying cation, usually sodium.

In addition, salt may be lost:

- from the skin in severe sweating, burns or erythroderma
- in association with inflammation of the peritoneum or pancreas
- following the removal of serous effusions, e.g. ascites.

The key feature to remember is that salt loss is always associated with loss of water and other ions, in particular, potassium. However, it is often easy to underestimate the loss of salt if another solute such as glucose is present in excess, i.e. hyperglycaemia. This will tend to retain fluid within the extracellular fluid and the severity of the situation will be only unmasked when the hyperglycaemia is treated. The urine sodium again will provide a good indicator in that it will be less than 10 mmol/l in all conditions, unless there is an intrinsic salt losing problem with the kidneys.

Urea normal and creatinine normal

Therefore exclude any electrolyte abnormality.

- Check potassium – high; secondary to haemolysis, increased intake (usually iatrogenic) or redistribution, e.g. with acidosis or muscle injury

Under these situations the serum sodium is normal.

Hyperkalaemia may also be present because of reduced excretion, e.g. with acute renal failure or the use of potassium sparing diuretics. Again the sodium is usually normal although it can be reduced in the former because of dilution.

- An elevated potassium in the presence of reduced sodium is suggestive of adrenal insufficiency.

The commonest cause of a low potassium is a metabolic alkalosis; therefore check the bicarbonate level.

Potassium may be lost from the gastrointestinal tract, e.g. with diarrhoea or malabsorption. Under these circumstances the serum sodium is usually normal. In contrast, renal loss, associated with either diuretic therapy or cardiac or liver failure, is accompanied by hyponatraemia. A normal urea and creatinine with low potassium and high sodium combined means that excess of glucocorticoid and mineral corticoid hormones has to be excluded.

- Check bicarbonate – high, when associated with metabolic alkalosis and hypokalaemia.
- Check sodium – hyponatraemia in the context of normal urea, creatinine and potassium is related to the extracellular fluid volume (see earlier).

Pseudohyponatraemia is a trap for the unwary. Sodium is present only in the aqueous phase of plasma. If there is an associated abnormal amount of lipid the water volume will be reduced and the measured sodium will be low. This result will be spurious because of the high proportion of lipid. In nephrotic syndrome or diabetes mellitus; e.g. 1 litre of plasma may comprise 600 ml of water and 400 ml of lipid with a measured sodium of 120 mmol/l. The true calculated value of sodium, however, when expressed according to the volume of water, is 120 mmol/l of sodium in 600 ml of water; and this equates to 200 mmol/l. Although this is an extreme example, it indicates that if such problems are not identified, inappropriate treatment may occur. A way to clarify this situation is to measure urine sodium and chloride which are low in true hyponatraemia.

SUMMARY

Abnormalities in urea, electrolytes and creatinine are common in acutely ill patients. These guidelines must be interpreted in the light of clinical findings. They will, however, facilitate the diagnosis of common conditions.

PART VII
Practical Procedures

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CHAPTER 31

Practical procedures: airway and breathing

PROCEDURES

- Oropharyngeal airway insertion
- Nasopharyngeal airway insertion
- Ventilation via a Laerdal pocket mask
- Orotracheal intubation
- Insertion of a laryngeal mask airway
- Needle cricothyroidotomy
- Needle thoracocentesis
- Aspiration of pneumothorax
- Aspiration of pleural fluid
- Chest drain insertion

OROPHARYNGEAL AIRWAY

Equipment

- A series of oropharyngeal (Guedel) airways (sizes 1, 2, 3, 4)
- Tongue depressor
- Laryngoscope

Procedure

The correct size of airway is selected by comparing it with the vertical distance from the angle of the mandible to the centre of the incisors. The airway is inserted in adults and older children as follows:

1 Open the patient's mouth and check for debris. This may be inadvertently pushed into the larynx as the airway is inserted

2 Insert the airway into the mouth either (a) 'upside down' (concave uppermost) as far as the junction between the hard and soft palates and rotate through 180° or (b) use a tongue depressor or the tip of a laryngoscope blade to aid insertion of the airway 'the right way up' under direct vision.

3 Insert so that the flange lies in front of the upper and lower incisors or gums in the edentulous patient (Fig. 31.1).

4 Check the patency of the airway and ventilation by 'looking, listening and feeling'.

Complications

- Trauma resulting in bleeding
- Vomiting or laryngospasm: if the patient is not deeply unconscious and has not lost gag reflex.

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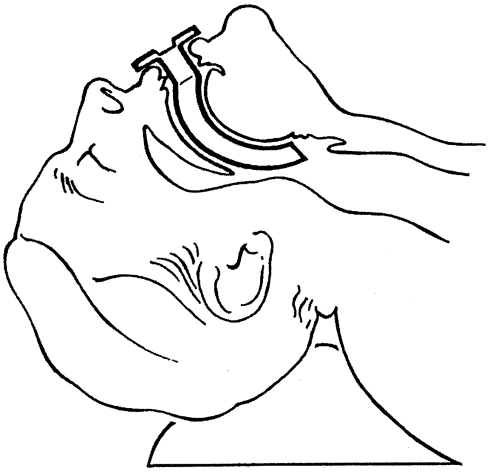

Fig. 31.1 Oropharyngeal airway *in situ*.

NASOPHARYNGEAL AIRWAY

Equipment

- A series of nasopharyngeal airways (sizes 6, 7, 8)
- Lubricant
- Safety pin

Procedure

Choose an airway approximately the same size as the patient's little finger or similar in diameter to the nares. Nasopharyngeal airways are designed to be inserted with the bevel facing medially. Consequently, the right nostril is usually tried first, using the following technique:

1. Lubricate the airway thoroughly.
2. Check the patency of right nostril.
3. Insert the airway bevel end first, along the floor of the nose (i.e. vertically in a supine patient) with a gentle twisting action.
4. When fully inserted, the flange should lie at the nares (Fig. 31.2).
5. Once in place insert a safety pin through the flange to prevent the airway being inhaled.
6. If the right nostril is occluded or insertion is difficult, use the left nostril.
7. Check the patency of the airway and ventilation by 'looking, listening and feeling'.

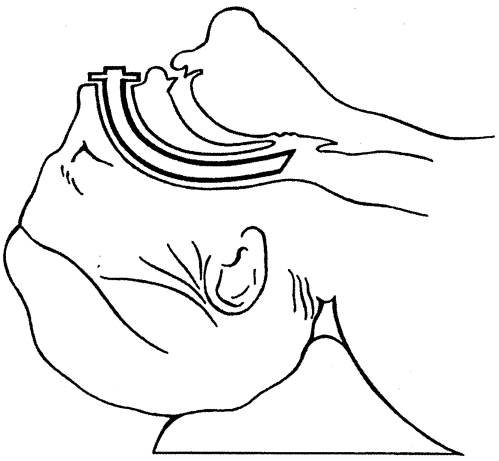

Fig. 31.2 Nasopharyngeal airway *in situ*.

Complications

- Trauma resulting in bleeding
- Vomiting

> **Key point**
>
> **Use the nasopharyngeal airway with caution in patients with a suspected base of skull fracture**

LAERDAL POCKET MASK

Equipment

- Laerdal pocket mask
- Airway manikin

Procedure

The technique for using the mask is as follows.

1. With the patient supine, apply the mask to the patient's face using the thumbs and index fingers of both hands.
2. The remaining fingers are used to exert pressure behind the angles of the jaw (as for the jaw thrust) at the same time as the mask is pressed on to the face to make a tight seal (Fig. 31.3).
3. Blow through the inspiratory valve for 1–2 s, at the same time looking to ensure that the chest rises and then falls.
4. If oxygen is available, add via the nipple at 12–15 l/min.

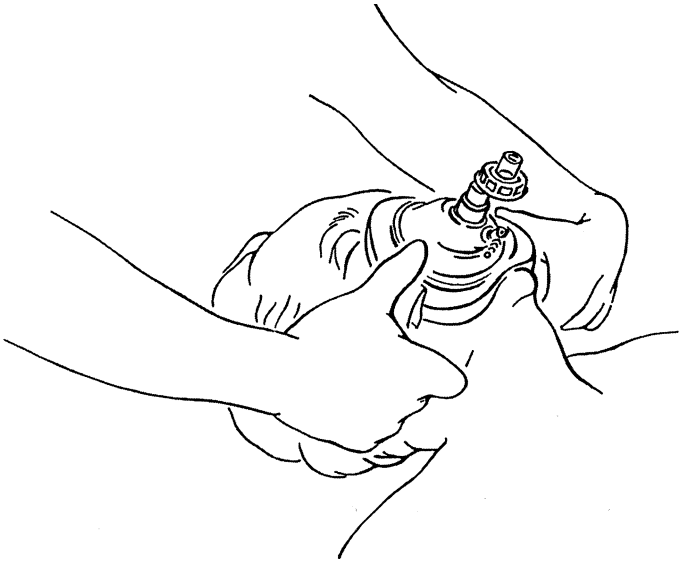

Fig. 31.3 Laerdal pocket mask.

OROTRACHEAL INTUBATION

Equipment

- Laryngoscope: most commonly with a curved (Macintosh) blade
- Tracheal tubes: females 7.5–8.0 mm internal diameter, 21 cm long
  males 8.0–9.0 mm internal diameter, 23 cm long

- Syringe, to inflate the cuff
- Catheter mount, to attach to ventilating device
- Lubricant for tube, water soluble, preferably sterile
- Magill forceps
- Introducers, malleable and gum elastic, for difficult cases
- Adhesive tapes or bandages for securing tube
- Ventilator
- Suction
- Stethoscope

Procedure

1. Whenever possible, ventilate the patient with 100% oxygen, using a bag–valve–mask device before intubation. During this time, check the equipment and ensure that all components are complete and functioning, particularly the laryngoscope, suction and ventilating device.
2. Choose a tracheal tube of the appropriate length and diameter, and check the integrity of the cuff.
3. Position the patient's head to facilitate intubation; flex the neck and extend the head at the atlantooccipital joint ('sniffing the morning air' position), provided there are no contraindications. This is often made easier by having a small pillow under the patient's head.
4. Hold the laryngoscope in your left hand, open the patient's mouth and introduce the blade into the right-hand side of the mouth, displacing the tongue to the left.
5. Pass the blade along the edge of the tongue. The tip of the epiglottis should be seen emerging at the base of the tongue.
6. Advance the tip of the blade between the base of the tongue and the anterior surface of the epiglottis (vallecula).
7. The tongue and epiglottis are then **lifted** to reveal the vocal cords; **note that the laryngoscope must be lifted in the direction that the handle is pointing and not levered** by movement of the wrist, as this might damage the teeth and will not provide as good a view (Fig. 31.4).

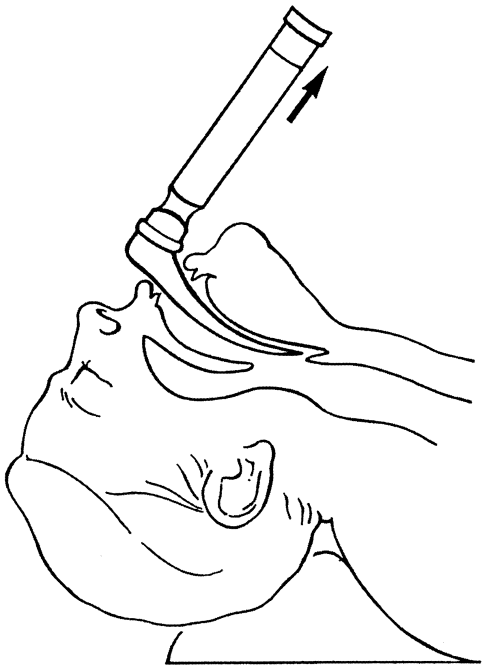

Fig. 31.4 Laryngoscopy.

8 Introduce the tracheal tube from the right-hand side of the mouth and insert it between the vocal cords into the larynx under direct vision, until the cuff just passes the cords.
9 Once the tube is in place, inflate the cuff sufficiently to provide an airtight seal between the tube and the trachea. (As an initial approximation, the same number of millilitres of air can be used as the diameter of the tube in millimetres, and adjusted later.)
10 Attach a catheter mount to the tube and ventilate. Ensure that the tube is in the correct position and confirm ventilation of both lungs, by:
   - looking for bilateral chest movement with ventilation
   - listening for breath sounds bilaterally in the midaxillary line
   - listening for gurgling sounds over the epigastrium, which may indicate inadvertent oesophageal intubation
   - measuring the carbon dioxide in the expired gas. This will be greater than 0.2% in gas leaving the lungs providing there is a spontaneous circulation or good quality cardiopulmonary resuscitation in progress. Less than 0.2% indicates oesophageal placement of the tube.

Manoeuvres to assist with intubation

Occasionally, when the larynx is very anterior, direct pressure on the thyroid cartilage by an assistant may aid visualisation of the cords (not to be confused with cricoid pressure). However, despite this manoeuvre, in a small percentage of patients only the very posterior part of the cords (or none) can be seen and passage of the tracheal tube becomes difficult. In these cases, a gum elastic introducer can often be inserted into the larynx initially and then the tracheal tube slid over the introducer into the larynx. However, remember that the patient must be oxygenated between attempts at intubation.

Complications

- All the structures encountered from the lips to the trachea may be traumatised.
- When the degree of unconsciousness has been misjudged, vomiting may be stimulated.
- A tube that is too long may pass into a main bronchus (usually the right), causing the opposite lung to collapse, thereby severely impairing the efficiency of ventilation. This is usually identified by the absence of breath sounds and reduced movement on the unventilated side.
- The most dangerous complication associated with tracheal intubation is unrecognised oesophageal intubation. Ventilation may appear adequate, but in fact the patient is not receiving oxygen and is rapidly becoming hypoxaemic. If in doubt, take it out and ventilate the patient using a bag–valve–mask.

INSERTION OF THE LARYNGEAL MASK AIRWAY (LMA)

Equipment

- Laryngeal mask airway:

Size	Cuff volume
• 5 – large adult	40 ml
• 4 – adult male	30 ml
• 3 – adult female	20 ml

- Lubricant
- Syringe to inflate cuff
- Adhesive tape to secure laryngeal mask airway
- Suction
- Ventilating device

Procedure

- Whenever possible, ventilate the patient with 100% oxygen using a bag–valve–mask device before inserting the laryngeal mask airway. During this time, check that all the equipment is present and working, particularly the integrity of the cuff.
- Deflate the cuff and lightly lubricate the back and sides of the mask.
- Tilt the patient's head (if safe to do so), open the mouth fully, and insert the tip of the mask along the hard palate with the open side facing, but not touching the tongue (Fig. 31.5a).
- Insert the mask further, along the posterior pharyngeal wall, with your index finger initially providing support for the tube (Fig. 31.5b). Eventually resistance is felt as the tip of the laryngeal mask airway lies at the upper end of the oesophagus (Fig. 31.5c).
- Fully inflate the cuff using the air-filled syringe attached to the valve at the end of the pilot tube using the volume of air shown in the earlier box (Fig. 31.5d).

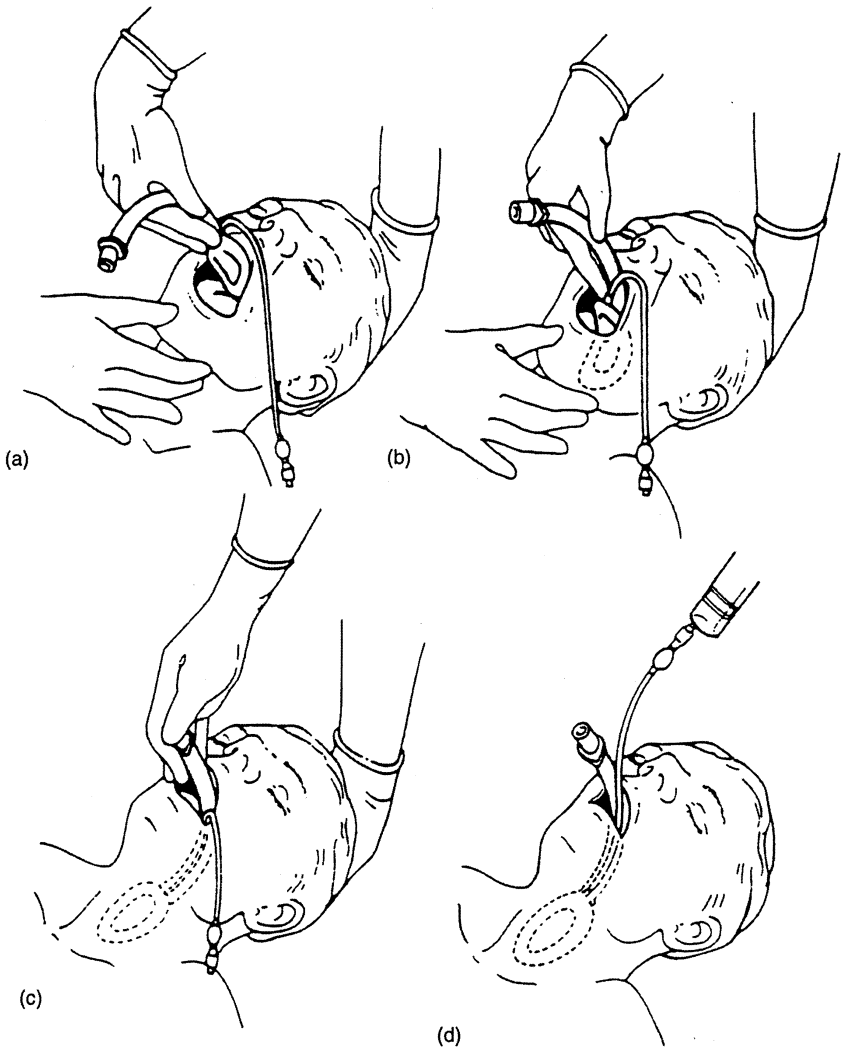

Fig. 31.5 Insertion of the laryngeal mask airway.

- Secure the laryngeal mask airway with adhesive tape and check its position during ventilation as for a tracheal tube.
- If insertion is not accomplished in less than 30 s, reestablish ventilation using a bag–valve–mask.

Complications

- Incorrect placement is usually due to the tip of the cuff folding over during insertion. The laryngeal mask airway should be withdrawn and reinserted.
- Inability to ventilate the patient, because the epiglottis has been displaced over the larynx. Withdraw the laryngeal mask airway and reinsert ensuring that it closely follows the hard palate. This may be facilitated by the operator or an assistant lifting the jaw upwards. Occasionally, rotation of the laryngeal mask airway may prevent its insertion. Check that the line along the tube is aligned with the patient's nasal septum; if not, reinsert.
- Coughing or laryngeal spasm is usually due to attempts to insert the laryngeal mask airway into a patient whose laryngeal reflexes are still present.

Intubation via the laryngeal mask airway

Insert an introducer through the laryngeal mask airway into the trachea, remove the laryngeal mask airway and then pass the tracheal tube over the introducer into the trachea. Alternatively, a small diameter cuffed tracheal tube (6.0 mm) may be passed directly through a size 4 laryngeal mask airway into the trachea. An intubating LMA (ILMA) is now available and this can accommodate a size 7 or 8 tracheal tube.

NEEDLE CRICOTHYROIDOTOMY

It is important to realise that this technique is a temporising measure, while preparing for a definitive airway.

Equipment

- Venflons 12–14 gauge
- Jet insufflation equipment
- Oxygen tubing with either a three-way tap or a hole cut in the side
- 20-ml syringe
- Gloves (sterile)

Procedure

1 Place the patient supine with the head slightly extended.
2 Identify the cricothyroid membrane as the recess between the thyroid cartilage (Adam's apple) and cricoid cartilage (approximately 2 cm below the 'V'-shaped notch of the thyroid cartilage) (Fig. 31.6).
3 Puncture this membrane vertically using a large bore (12–14 gauge) intravenous cannula attached to a syringe.
4 Aspiration of air confirms that the tip of the cannula lies within the tracheal lumen.
5 Angle the cannula at 45° caudally and advance over the needle into the trachea (Fig. 31.7).

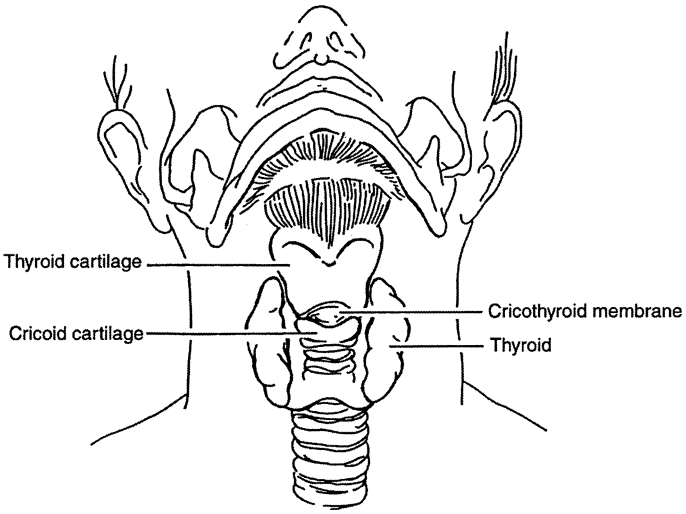

Fig. 31.6 Cricothyroidotomy: relevant anatomy.

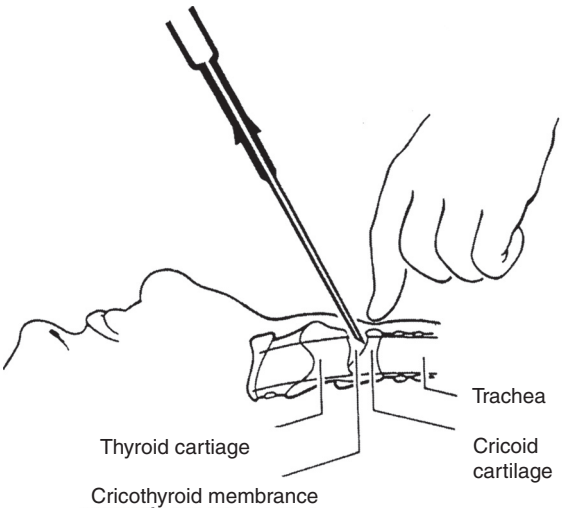

Fig. 31.7 Needle cricothyroidotomy.

6 Attach the cannula to an oxygen supply at 12–15 l/min either via a 'Y' connector or a hole cut in the side of the oxygen tubing. Oxygen is delivered to the patient by occluding the open limb of the connector or side hole for 1 s and then releasing for 4 s.

7 Expiration occurs passively through the larynx. Watch the chest for movement and auscultate for breath sounds, although the latter are difficult to hear.

8 If satisfactory, secure the cannula in place to prevent it being dislodged.

An alternative method of delivering oxygen is to use jet ventilation. This involves connecting the cannula to a high pressure oxygen source (4 bar, 400 kPa, 60 psi) via luerlock connectors or by using a Sanders injector. The same ventilatory cycle is used.

Complications

- Asphyxia
- Pulmonary barotrauma
- Bleeding
- Oesophageal perforation

- Kinking of the cannula
- Subcutaneous and mediastinal emphysema
- Aspiration

Occasionally, this method of oxygenation will disimpact a foreign body from the larynx, allowing more acceptable methods of ventilation to be used.

There are two important facts to remember about transtracheal insufflation of oxygen:

- Firstly, it is not possible to deliver oxygen via a needle cricothyroidotomy using a self-inflating bag and valve. This is because these devices do not generate sufficient pressure to drive adequate volumes of gas through a narrow cannula. In comparison, the wall oxygen supply will provide a pressure of 400 kPa (4000 cm H_2O), which overcomes the resistance of the cannula.
- Secondly, expiration cannot occur through the cannula or through a separate cannula inserted through the cricothyroid membrane. The pressure generated during expiration is generally less than 3 kPa (30 cm H_2O), which is clearly much less than the pressure required to drive gas in initially. Expiration must occur through the upper airway, even when partially obstructed. If the obstruction is complete, then the oxygen flow must be reduced to 2–4 l/min to avoid the risk of barotrauma, in particular the creation of a tension pneumothorax

NEEDLE THORACOCENTESIS

Equipment

- Alcohol swab
- Intravenous cannula (minimum 16 gauge)
- 20-ml syringe
- Gloves (sterile)

Procedure

1. Identify the second intercostal space in the midclavicular line on the side of the pneumothorax (the opposite side to the direction of tracheal deviation).
2. Swab the chest wall with surgical preparation or an alcohol swab.
3. Attach the syringe to the cannula.
4. Insert the cannula into the chest wall, just over the rib, aspirating all the time.
5. If air is aspirated, remove the needle, leaving the plastic cannula in place.
6. Tape the cannula in place and proceed to chest drain insertion (see later) as soon as possible.

> **Key point**
>
> **If needle thoracocentesis is attempted, and the patient does not have a tension pneumothorax, the chance of causing a pneumothorax is 10–20%. Patients must have a chest X-ray, and will require chest drainage if ventilated**

Complications

- Local haematoma
- Lung laceration

ASPIRATION OF PNEUMOTHORAX

Equipment

- Alcohol swab
- Intravenous cannula (minimum 16 gauge)
- 20-ml syringe
- Three-way tap
- Gloves (sterile)

Procedure

The equipment is the same as for needle thoracocentesis, plus a three-way tap.

1. Explain to the patient the nature of the procedure.
2. Use appropriate aseptic techniques.
3. Identify the second intercostal space in the midclavicular line.
4. After appropriate skin preparation, infiltrate the area with 1% lignocaine.
5. Insert a large (14 or 16 gauge) cannula, remove the central trochar and attach a three-way tap and 50-ml syringe.
6. Continue to aspirate until resistance is encountered or the patient experiences discomfort or coughing.

> **Key point**
>
> **Aspiration of 2 litres of air may suggest a persistent air leak; the procedure should be abandoned and a formal chest drain insertion considered**

Complications

- As for needle thoracocentesis

ASPIRATION OF PLEURAL FLUID

Equipment

- Skin preparation
- Local anaesthetic
- 5-ml syringe with orange, blue and green hubbed needles
- 50-ml syringe with three-way tap
- 16-gauge cannula

Procedure

It is recommended by the National Patient Safety Agency (NPSA) that pleural fluid aspiration should be done under ultrasound control.

1. Explain to the patient the nature of the procedure and obtain written consent.
2. Identify the appropriate side for aspiration of pleural fluid using the ultrasound probe.
3. Clean the skin.
4. After raising the skin bleb, the local anaesthetic is injected via the orange hubbed needle. Introduce the larger blue hubbed needle over the superior aspect of the rib through the intercostal tissues down to the pleura.
5. Always aspirate before injecting to ensure that a blood vessel has not been traumatised.

6 For a diagnostic aspiration a green hubbed 21-gauge needle can be inserted through this anaesthetised area into the pleural space, and fluid aspirated into a 30-ml syringe.
7 In contrast, fluid can be aspirated after insertion of a large cannula through this area and attaching the syringe to the cannula via a three-way tap.

Failure of aspiration

Attempted aspiration either too high or too low
Thickened pleura
Pleural tumour
Viscid empyema fibrinous exudate
Dry tap for reasons described above
Haematoma
Bleeding
Pneumothorax

Complications

- As for needle thoracocentesis

Chest drain insertion

(a) Seldinger technique

Equipment

- Skin preparation and surgical drapes
- Local anaesthetic
- Scalpel
- Scissors
- Seldinger chest drain kit comprising guide wire, dilator, over the wire drain
- Suture
- Underwater seal
- 10-ml syringe with orange, blue and green needles

Procedure

1 Confirm correct side for insertion.
2 Identify relevant landmarks (usually the fifth intercostal space anterior to the midaxillary line) on the side of the pneumothorax.
3 Identify relevant landmarks.
4 Swab the chest with skin preparation.
5 Use local anaesthetic – as described above.
6 Make a 'stab' incision (approximately 0.5 cm).
7 Insert the introducer cannula with an attached syringe; aspirate gently as you advance the cannula.
8 Remove syringe.
9 Insert wire through the cannula.
10 Remove cannula, maintaining wire position.
11 Advance dilator over wire, ensuring you hold the free end of the wire before advancing the dilator into the chest. Ensure the dilator moves freely (if not the drain will be difficult to insert).

12 Remove the dilator.
13 Insert the drain over the wire as per Step 9.
14 Remove the wire.
15 Connect the drain to an underwater seal.
16 Ensure either the tube is fogging (in patient with pneumothorax) or fluid is draining (pleural effusion).
17 Secure the drain.
18 X-ray the patient's chest.

(b) Dissection technique

Equipment

- Skin preparation and surgical drapes
- Local anaesthetic
- Scalpel
- Scissors
- Large clamps ×2
- Chest drain tube without trochar
- Suture
- Underwater seal
- 10-ml syringe with orange, blue and green needles

Procedure

1 Confirm correct side for insertion.
2 Identify relevant landmarks (usually the fifth intercostal space anterior to the midaxillary line) on the side with the pneumothorax.
3 Swab the chest wall with surgical preparation or an alcohol swab.
4 Use local anaesthetic if necessary – as described earlier.
5 Make a 2–3 cm transverse skin incision along the line of the intercostal space, towards the superior edge of the sixth rib (thereby avoiding the neurovascular bundle).
6 Bluntly dissect through the subcutaneous tissues just over the top of the rib, and puncture the parietal pleura with the tip of the clamp.
7 Put a gloved finger into the incision and clear the path into the pleura.
8 Advance the chest drain tube into the pleural space **without** the trochar.
9 Ensure that the tube is in the pleural space by listening for air movement, and by looking for fogging of the tube during expiration.
10 Connect the chest drain tube to an underwater seal.
11 Suture the drain in place, and secure with tape.
12 Obtain a chest X-ray.

Complications

- Damage to intercostal nerve, artery or vein
- Introduction of infection
- Tube kinking, dislodging or blocking
- Subcutaneous emphysema
- Persistent pneumothorax due to faulty tube insertion, leaking around chest drain, leaking underwater seal, bronchopleural fistula
- Failure of lung to expand due to blocked bronchus
- Anaphylactic or allergic reaction to skin preparation

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CHAPTER 32

Practical procedures: circulation

PROCEDURES

- Peripheral venous cannulation
- Central venous cannulation:
  - Internal jugular vein
  - Subclavian vein
  - Femoral vein
  - Seldinger technique

VENOUS ACCESS

Venous access is an essential part of managing any acutely ill patient. It is an invasive procedure that must not be treated with complacency.

Vascular access can be achieved via several routes:

- percutaneous cannulation of a peripheral vein
- following surgical exposure of a vein in the 'cutdown' technique
- percutaneous cannulation of a central vein
- intraosseous route.

Success is optimised and complications minimised when the operator understands the:

- local anatomy
- equipment
- technique
- complications.

PERIPHERAL VENOUS CANNULATION

The antecubital fossa is the commonest site for peripheral venous cannulation.

The cephalic vein passes through the antecubital fossa on the lateral side and the basilic vein enters very medially just in front of the medial epicondyle of the elbow. These two large veins are joined by the **median cubital or antecubital vein**. The median vein of the forearm also drains into the basilic vein (Fig. 32.1).

Although the veins in this area are prominent and easily cannulated, there are many other adjacent vital structures which can be easily damaged.

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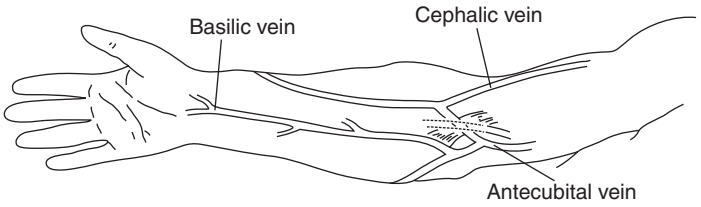

Fig. 32.1 Veins of the forearm and antecubital fossa.

The most popular device for peripheral intravenous access is the cannula over needle, available in a wide variety of sizes, 12–27 gauge. It consists of a plastic (PTFE or similar material) cannula which is mounted on a smaller diameter metal needle, the bevel of which protrudes from the cannula. The other end of the needle is attached to a transparent 'flashback chamber', which fills with blood, indicating that the **needle** bevel lies in the vein. Some devices have flanges or 'wings' to facilitate attachment to the skin. All cannulae have a standard Luer-Lock fitting to attach a giving set and some have a valved injection port attached through which drugs can be given.

Equipment

- Alcohol swab
- Intravenous cannulae
- Tourniquet
- Tape
- Commercial fixing system
- Gloves (need not be sterile)

Procedure

1. Choose a vein capable of accommodating a large cannula, preferably one that is both visible and palpable. The junction of two veins (see Figure 32.2) is often a good site as the 'target' is relatively larger and more stable.
2. Encourage the vein to dilate as this increases the success rate of cannulation. In the limb veins use a tourniquet that stops venous return but permits arterial flow. Further dilatation can be encouraged by gently tapping the skin over the vein. If the patient is cold and vasoconstricted, if time permits, topical application of heat from a towel soaked in warm water can cause vasodilatation.
3. If time permits, the skin over the vein should be cleaned. Ensure there is no risk of allergy if iodine-based agents are used. If alcohol-based agents are used, they must be given time to work (2–3 min), ensuring that the skin is dry before proceeding further.
4. In the conscious patient, consider infiltrating a small amount of local anaesthetic into the skin at the point chosen using a 22–25-gauge needle,

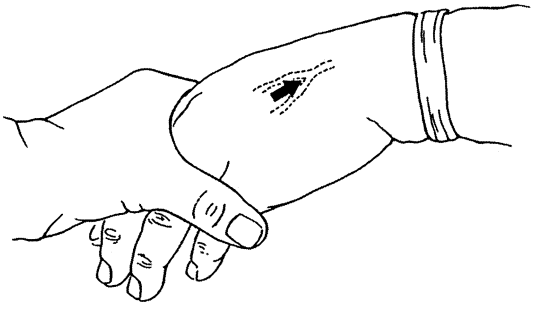

Fig. 32.2 Vein immobilised.

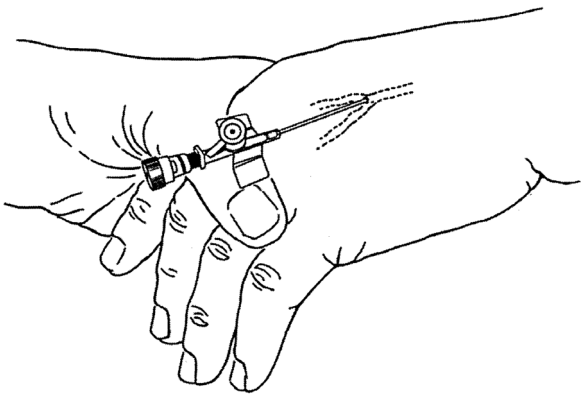

Fig. 32.3 Cannula inserted (note the flashback of blood).

particularly if a large (>1.2-mm, 18-gauge) cannula is to be used. This reduces the pain of cannulation, therefore making the patient less likely to move and less resistant to further attempts if the first is unsuccessful!

5 If a large cannula is used, insertion through the skin may be facilitated by first making a small incision with either a 19-gauge needle or a scalpel blade, taking care not to puncture the vein.

6 Immobilise the vein to prevent displacement by the advancing cannula. Pull the skin over the vein tight, with your spare hand (Fig. 32.2).

7 Hold the cannula firmly, at an angle of 10–15° to the skin and advance through the skin and then into the vein. Often a slight loss of resistance is felt as the vein is entered. This should be accompanied by the appearance of blood in the flashback chamber of the cannula (Fig. 32.3). However, the appearance of blood only indicates that the tip of the needle is within the vein, not necessarily any of the cannula.

8 Whilst keeping the skin taut, the next step is to reduce the angle of the cannula slightly and advance it a further 2–3 mm into the vein. This is to ensure that the first part of the plastic cannula lies within the vein. Care must be taken at this point not to push the needle out of the back of the vein.

9 Withdraw the needle 5–10 mm into the cannula so that the point no longer protrudes from the end. Often as this is done, blood will flow between the needle body and the cannula, confirming that the tip of the cannula is within the vein (Fig. 32.4).

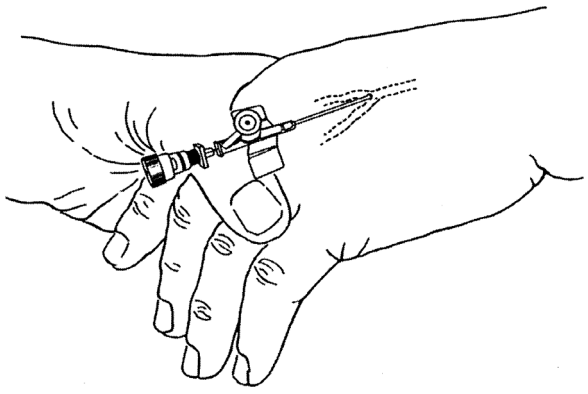

Fig. 32.4 Cannula with needle slightly withdrawn.

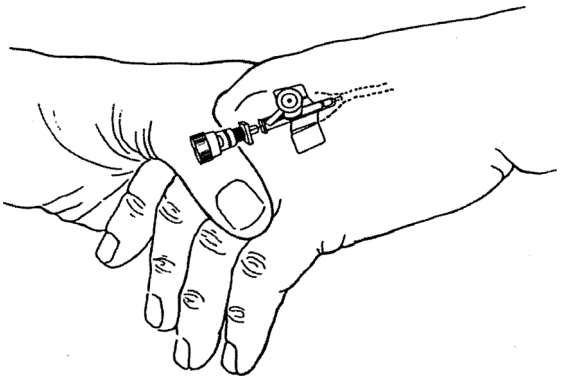

Fig. 32.5 Cannula fully inserted.

10 Advance the combined cannula and needle along the vein. The needle is retained within the cannula to provide support and prevent kinking at the point of skin puncture (Fig. 32.5).

11 Insert the cannula as far as the hub, release the tourniquet and remove the needle and place in a sharps bin.

12 Confirm that the cannula lies within the vein either by attaching an intravenous infusion, ensuring that it runs freely, or by injecting saline. Watch the tissues around the site for any signs of swelling that may indicate that the cannula is incorrectly positioned. Finally, secure the cannula in an appropriate manner.

Complications

- Failed cannulation is the most common, usually as a result of pushing the needle completely through the vein. It is inversely related to experience.
- Haematomata are usually secondary to the above with inadequate pressure applied to prevent blood leaking from the vein after the cannula is removed. They are made worse by forgetting to remove the tourniquet!
- Extravasation of fluid or drugs is commonly a result of failing to recognise that the cannula is not in the vein before use. Placing a cannula over a joint or prolonged use to infuse fluids under pressure also predisposes to leakage. The faulty cannula must be removed. Damage to the surrounding tissues will depend primarily on the nature of the extravasated fluid.
- Damage to other local structures is secondary to poor technique and lack of knowledge of the local anatomy.
- The plastic cannula can shear, allowing fragments to enter the circulation. This is usually a result of trying to reintroduce the needle after it has been withdrawn. The safest action is to withdraw the whole cannula and attempt cannulation at another site with a new cannula.
- The needle may fracture as a result of careless excessive manipulation with the finer cannulae. The fragment will have to be removed by either an interventional vascular radiologist or a surgeon.
- Inflammation of the vein (thrombophlebitis) is related to the length of time the vein is cannulated and the irritation caused by the substances flowing through it. High concentrations of drugs and fluids with extremes of pH or high osmolality are the main causes. Once a vein shows signs of thrombophlebitis, i.e. tender, red and the flow rate is deteriorating, the cannula must be removed to prevent subsequent infection or thrombosis which may spread proximally.
- To reduce the risk of thromobophlebitis/infection, many hospitals have a policy for peripheral venous access which states that cannulae should be changed

every 3 days and that the date of insertion should be written on the securing tape and in the patient's records.

CENTRAL VENOUS CANNULATION

Catheterisation of a central vein is relatively easy. As with all procedures, it is best learned under supervision (NICE guidelines advise ultrasound guidance). However, in an acutely ill patient, it may be necessary for someone to catheterise a central vein safely and quickly. Therefore an easy technique is required that has a high success rate with few complications.

Central venous cannulation is a common technique in acutely ill patients for:

- drug delivery
- central pressure monitoring
- pacing
- inserting a pulmonary artery flotation catheter
- parenteral nutrition.

Equipment

- Skin preparation
- Local anaesthetic
- 10-ml syringe with blue and green needles
- Appropriate catheter for central venous cannulation (see 'Seldinger technique')
- Suture
- Tape
- No 11 blade
- Ultrasound probe
- Gloves (sterile)

Procedure

Many approaches and different types of equipment have been described to secure central venous access. This chapter describes three approaches – internal jugular, subclavian and femoral – using a single standard technique. Whenever possible for neck vein access, place the patient in a head-down position to dilate the vein and reduce the risk of air embolus. This has been found to be successful in both experienced and inexperienced hands. No further justification of the choice is offered. For those already skilled at central venous cannulation using a different technique (**with an acceptable rate of complications**), carry on!

The internal jugular vein – paracarotid approach

At the level of the thyroid cartilage, the internal jugular vein (Fig. 32.6 – see over) runs parallel to the carotid artery in the carotid sheath and therefore rotation of the head, obesity and individual variations in anatomy have less effect on the location of the vein.

1. Place the patient in the supine position, arms at their side and the head in a neutral position.
2. Standing at the head of the patient identify the thyroid cartilage and use the fingers of the left hand to palpate the carotid pulse. The right internal jugular vein is the one most commonly used initially.
3. Identify the apex of a triangle formed by the two heads of sternoclavicular muscle (the base is the clavicle).
4. Under aseptic conditions, infiltrate with 1% lignocaine.

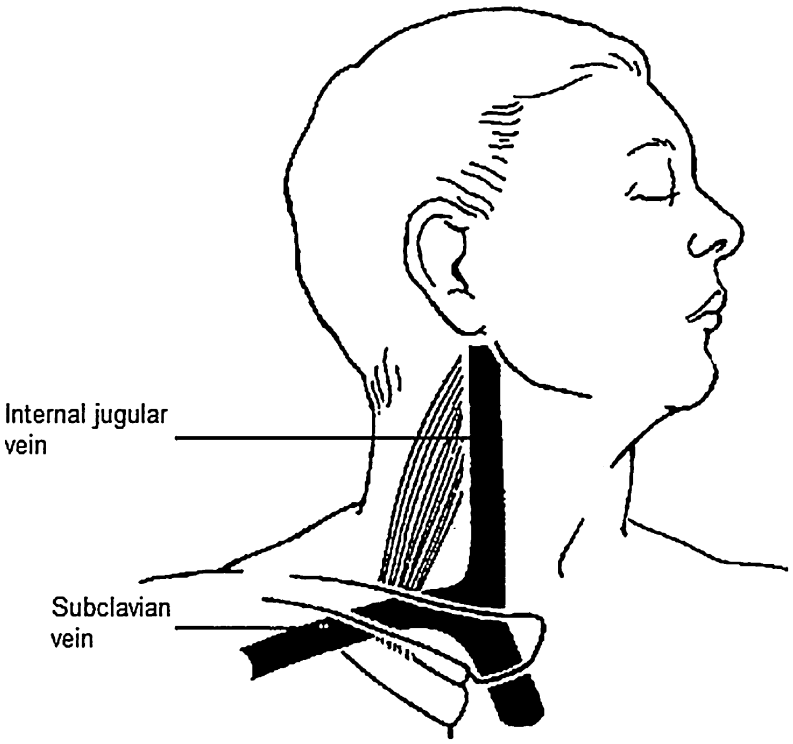

Fig. 32.6 The course of the central veins of the neck.

5 With the fingers of the left hand 'guarding' the carotid artery, insert a needle 0.5 cm lateral to the artery. Inject 0.5–1 ml of air to expel any skin plug in the needle tip.
6 Advance the needle slowly caudally, parallel to the sagittal plane at an angle of 45° to the skin, aspirating at all times.
7 Confirm entry into the vein by blood entering the syringe. Introduce the catheter via a guide wire as described later.
8 If the vein is not entered at the first attempt, then subsequent punctures should be directed slightly more laterally (never medially towards the artery).

Although a chest X-ray should be taken it is less urgent than when using the subclavian vein, as the catheter is more likely to be correctly positioned and the incidence of pneumothorax is much lower with this approach.

The subclavian vein – infraclavicular approach

1 Place the patient in supine position, with arms at their side and head turned away from the side of the puncture. Occasionally it may be advantageous to place a small support (a 500 ml bag of fluid!) under the scapula of the side of approach, to raise the clavicle above the shoulder.
2 Standing on the same side as that to be punctured (usually the right), identify the midclavicular point and the suprasternal notch.
3 Under aseptic conditions infiltrate with 1% lignocaine.
4 Insert the needle 1 cm below the midclavicular point and inject 0.5–1 ml of air to expel any skin plug in the needle tip. Advance the needle posterior to the clavicle towards a finger in the suprasternal notch. Keep the syringe and needle horizontal during advancement. Aspirate at all times.
5 Blood entering the syringe will confirm entry into the vein. Introduce the catheter via a guide wire as already described.

6 A chest X-ray should be taken as soon as possible to exclude a pneumothorax and confirm the correct position of the catheter.

The femoral vein

1 Place the patient in a supine position.
2 Under aseptic conditions, palpate the femoral artery (midinguinal point). The femoral vein lies directly medial to the femoral artery (remember lateral to medial structures are femoral nerve, artery, vein, space).
3 Infiltrate the puncture site with local anaesthetic.
4 While palpating the femoral artery insert the needle over the femoral vein parallel to the sagittal plane at an angle of 45° to the skin, aspirating at all times. A free flow of blood entering the syringe will confirm entry into the vein.
5 Advance the guide wire through the needle as described below.

Complications

- Venous thrombosis
- Injury to artery or nerve
- Infection
- Arteriovenous fistula
- Air embolism

In addition, attempts at internal jugular or subclavian vein access may cause pneumothorax, haemothorax and chylothorax.

SELDINGER TECHNIQUE

Equipment

- Skin cleaning swabs
- Lignocaine 1% for local anaesthetic with 2-ml syringe and 23-gauge needle
- Syringe and heparinised 0.9% saline
- Seldinger cannulation set: syringe
  needles
  Seldinger guide wire
  cannula
- Suture material
- Prepared infusion set
- Tape
- No 11 blade
- Gloves (sterile)

Procedure

Although initially described for use with arterial cannulation, this technique is very suitable for central venous cannulation and is associated with an increased success rate. It relies on the insertion of a guide wire into the vein over which a suitable catheter is passed. As a relatively small needle is used to introduce the wire, damage to adjacent structures is reduced.

Having decided which approach to use (see earlier), the skin must be prepared and towelled. Full aseptic precautions are necessary as a 'no-touch' technique is impossible.

1 Check and prepare your equipment; in particular, identify the floppy end of the guide wire and ensure free passage of the guide wire through the needle.
2 Attach the needle to a syringe and puncture the vein.

3 After aspirating blood, remove the syringe taking care to avoid the entry of air (usually by placing a thumb over the end of the needle).
4 Insert the floppy end of the guide wire into the needle and advance 4–5 cm into the vein.
5 Remove the needle over the wire, taking care not to remove the wire with the needle.
6 Load the catheter on to the wire, ensuring that the proximal end of the wire protrudes from the catheter. Holding the proximal end of the wire, insert the catheter and wire together into the vein. It is important never to let go of the wire!
7 Remove the wire holding the catheter in position.
8 Reattach the syringe and aspirate blood to confirm placement of the catheter in the vein.

If it is difficult to insert the wire, the needle and wire must be removed together. Failure to do this may damage the tip of the wire as it is withdrawn past the needle point. After 3 min gentle pressure to reduce bleeding, the needle can be reintroduced.

It is a good idea to make a small incision in the skin to facilitate the passage of the catheter.

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CHAPTER 33
Practical procedures: medical

PROCEDURES

- Joint aspiration
- Balloon tamponade of oesophageal varices
- Lumbar puncture
- Blood cultures
- Insertion of pulmonary arterial flotation (Swan–Ganz) catheter
- Pulmonary capillary wedge pressure

JOINT ASPIRATION

Diagnostic indications

- Suspected septic arthritis
- Crystal induced synovitis
- Haemarthrosis

Therapeutic indications

- Tense effusions
- Septic effusions
  - Recurrent aspiration
  - Lavage (rare)
- Haemarthrosis
- Steroid injection

Contraindications

- Overlying skin infection/cellulitis

Equipment

- Antiseptic solution, e.g. ethanol, povidone iodine
- Swabs
- Sterile gloves
- Syringes: 5, 10, 20 ml
- Needles: large joint (21 gauge) green
- Small joint (23 gauge) blue

Procedure

1 Explain to the patient what you are going to do, and obtain written consent.
2 Identify the bony margins of the joint space.
3 Ensure you have all the appropriate materials required.
4 Using a sterile technique prepare the skin.

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5 Inject a small amount of local anaesthetic (1% lignocaine) into the skin over the joint to be aspirated.
6 Gently insert the needle into the joint space. Normally a green needle (21 gauge) will suffice for most joints, but for finger and toe joints a blue (23-gauge) needle is advised.
7 Aspirate fluid and send for microbiological assessment, crystals, cytology, protein, lactate dehydrogenase (LDH), and glucose estimation.

Specific procedures

Knee joint aspiration

1 Ensure the patient is as comfortable as possible.
2 Slightly flex the knee to ensure relaxation of the quadriceps muscles.
3 Palpate the posterior edge of the patella medially or laterally. Using the general technique, described earlier, insert the needle horizontally or slightly downwards into the joint between the patella and femur (often a slight resistance is felt when the needle penetrates the synovial membrane).

Shoulder joint aspiration

This joint is easier to access through an anterior approach although a lateral and posterior approach's are also possible. The anterior approach will be described.

1 Ensure that the patient is seated with their arm relaxed against the side of the chest.
2 Palpate the space between the head of the humerus and the glenoid cap, about 1 cm below the coracoid process.
3 Using the procedure described earlier, insert the needle into the space with a slight medial angle (it should enter the joint easily and to almost the length of the green needle).

Complications

- Reaction to topical skin preparation.
- Inappropriate puncture of blood vessels or nerves.
- Introduction of infection into joint space.

BALLOON TAMPONADE OF OESOPHAGEAL VARICES

Equipment

- Sengstaken–Blakemore tube
- Two spigots
- 60-ml bladder syringe
- Saline/Contrast media
- Tongue depressors
- Tape
- Pressure gauge
- Suction
- Drainage bags

Procedure

Variceal bleeding can be controlled by balloon compression either at the cardia or within the oesophageal lumen. A large number of devices are available for this purpose. The commonest is a Sengstaken–Blakemore tube that has been modified to allow aspiration of gastric and oesophageal contents as well as inflation of gastric and oesophageal balloons.

Insertion of the tube usually occurs in conscious patients and, therefore, the nasal route is advocated. Unfortunately this can make insertion difficult but is subsequently better tolerated by the patient. If the airway is in jeopardy, ensure that it is cleared and secured before attempting to insert the tamponade tube. If the patient has an endotracheal tube *in situ*, the oral route is advocated. Although you may be faced with torrential bleeding from oesophageal varices, ensure that you have all the equipment available before you attempt insertion of this tube and, more importantly, that the associated oesophageal and gastric balloons will inflate and remain inflated.

It is important to realise that tamponade tubes are difficult to introduce and they require meticulous supervision whilst inflated.

1. Lubricate the tube with water-soluble jelly.
2. Providing that there are no contraindications, insert the tube into the right nostril using a technique similar to that described for nasopharyngeal airway insertion in Chapter 30. Ensure you direct the tube backwards (not superior or inferior).
3. Advance the tube gently. It will follow the contour of the oropharynx into the oesophagus.
4. Advance the tube until you reach the 50-cm mark (note that the tube has 5-cm graduations). Advancing the tube to at least 50 cm will, in most patients, ensure that it is in the stomach. Aspiration of blood does not, however, verify this.
5. Inflate the gastric balloon with 200 ml of air or alternatively 200 ml of water-soluble contrast material. Gentle traction of the nasal end of the tube will ensure that the inflated gastric balloon is adjacent to the cardia and gastro-oesophageal junction.
6. Tape the balloon to the side of the patient's face. Often inflation of the gastric balloon, with gentle traction, is all that is required to stem variceal bleeding as the feeding vessel to the varices, the left gastric vein, is tamponaded by this manoeuvre. If this fails to control the bleeding then inflate the oesophageal balloon with air to 4.5–5.4 kPa (30–40 mm Hg) using a pressure gauge. If a specific pressure gauge is not available then it is possible to adapt a sphygmomanometer for this purpose.
7. Ensure that both gastric and oesophageal aspiration ports are draining freely. Both the gastric and oesophageal balloons seal automatically once inflated by one-way valves. Continuous oesophageal suction reduces the risk of aspiration.
8. Deflate the balloon after 24 h. This will reduce the risk of oesophageal mucosal ulceration and perforation.

It is important to realise that balloon tamponade is only a temporising procedure and once the bleeding has stopped the patient should have a repeat endoscopy to assess and treat varices.

Complications

- Aspiration, especially without continuous aspiration of the oesophageal port
- Hypoxaemia, if the balloon is inadvertently inserted into the trachea
- Tracheal rupture, as above
- Oesophageal rupture. The procedure is performed blindly and with the presence of a hiatus hernia or an oesophageal stricture it is possible for the Sengstaken–Blakemore tube to coil in the oesophagus. Inflation produces catastrophic results
- Mucosal ulceration in the oesophagus and stomach
- Failure to stop variceal haemorrhage

LUMBAR PUNCTURE

Indications

- Suspected meningitis
- Subarachnoid haemorrhage
- Encephalitis
- Benign intracranial hypertension

Contraindications

A lumbar puncture should not be done in the unconscious patient until a CT scan has excluded a mass lesion. Failure to do this may precipitate central/uncal herniation as cerebrospinal fluid is drained via the lumbar puncture needle. Furthermore, a diagnosis of subarachnoid haemorrhage on CT will negate the need for lumbar puncture. However, CT scans can miss a small subarachnoid bleed and as this often heralds subsequent catastrophic haemorrhage, a lumbar puncture must be done in any patient who has a clinical history suggestive of subarachnoid haemorrhage and a negative CT scan.

Equipment

- Antiseptic solution
- Gauze swabs
- Sterile drapes and gloves
- 1% lignocaine (max 5 ml)
- 5 ml syringe
- Needles: 25 gauge (orange)
  21 gauge (green)
- Lumbar puncture needles
- Manometer
- Collection bottles
- Tape

Procedure

1. Explain to the patient what you are going to do, and obtain written consent.
2. Place the patient in the left lateral position, ensuring that their back, in particular the lumbar spine, is parallel to the edge of the bed. The hips and knees should be flexed to greater than 90° and the knees separated by one pillow. Ensure that the head is supported on one pillow and that the patient's cervical and thoracic spine are gently flexed.
3. Check that you have all the necessary equipment.
4. Identify the fourth lumbar vertebra, i.e. a line drawn between the top of the iliac crests.
5. Thoroughly cleanse the skin using an aseptic technique.
6. Identify the interspace between the second and third or third and fourth lumbar vertebrae (hence the spinal cord will not be damaged). In the midline, inject a small amount of 1% lignocaine to raise a skin bleb.
7. Through the skin bleb, advance a green needle and ensuring that the blood vessel has not been punctured. Inject 1 ml local anaesthetic into the interspinous ligament in the respective interspace. Too much local anaesthetic will cause damage to these tissues and produce profound discomfort.
8. Using a sterile spinal needle advance through the anaesthetised tissues, directing the needle slightly cephalad and maintaining a midline position.

9 As you enter the subarachnoid space, a sudden change in resistance on advancing the needle is felt. Then gently remove the inner trochar and watch for a drop of cerebrospinal fluid appearing at the end of the needle. If this does not occur, replace the central trochar and advance the needle again, until a change in resistance is felt. Repeat the procedure until cerebrospinal fluid is seen.
10 Attach the manometer and measure the pressure of the cerebrospinal fluid.
11 Place five drops of cerebrospinal fluid sequentially in three tubes for red cell count, then five drops in a further two for microscopy culture and sensitivity. Similar samples should be taken for protein estimation, spectroscopy, virology, and glucose (the latter should be placed in a fluoride tube).
12 Note the colour of the cerebrospinal fluid, i.e. whether it is clear, opalescent or yellow (Remember xanthochromia is a spectroscopic diagnosis).
13 Remove the needle. Occasionally, postlumbar puncture headache may result which necessitates simple analgesia with paracetamol.

Complications

- Failure to obtain cerebrospinal fluid may be due to incorrect anatomical positioning, 'a dry tap', degenerative or inflammatory changes in the lumbar spine
- Nerve root pain when inserting the needle – usually transient
- Introduction of sepsis
- Bleeding
- Headache
- Coning

BLOOD CULTURES

Indications

- Pyrexia of unknown origin
- Sepsis
- Suspected infective endocarditis

Procedure

- Thoroughly cleanse the skin, ideally with an alcohol-based solution.
- Whilst this is evaporating to dryness wash your hands thoroughly; under aseptic conditions don surgical gloves.
- At the previously prepared site perform a venepuncture and aspirate 40 ml of blood.
- Thoroughly cleanse the top of the blood culture bottle.
- Insert 10 ml of blood into each blood culture bottle.

INSERTION OF PULMONARY ARTERIAL FLOATATION (SWAN-GANZ) CATHETER

Indications

1 Measurement of pulmonary capillary wedge pressure (PCWP)
2 Pulmonary artery end diastolic pressure (PAEDP)
3 Cardiac output

Equipment

See Central venous cannulation

The Catheter

This is a balloon tipped device which has a single distal hole. It can be inserted at the bedside without X-ray control or under fluoroscopy.

The balloon serves two purposes. Firstly as soon as the catheter is inserted into a central vein, inflation of the balloon with air will ensure that it acts as a sail navigating the catheter through the tricuspid and pulmonary valves. Changes in the pressure tracing, as described later, will enable these structures to be identified. Secondly, once the catheter is inserted into a small pulmonary artery, the balloon may then be inflated, occluding the artery proximally. This will leave the catheter tip exposed to the PCWP.

Catheter Insertion

Using central venous access as described earlier advance the catheter into a large vein. If an insertion sheath is used, ensure it is one size larger than the catheter, to ensure passage of the deflated balloon through the insertion sheath.

1 Advance the catheter into the vein and connect to transducer.
2 Inflate the balloon.
3 Slowly advance the catheter tip, guided by the blood flow.
4 Advance the catheter through into the pulmonary artery bed, trying to find a position which gives a good pulmonary artery tracing with the balloon deflated and a good wedge pressure with the balloon inflated.
5 X-ray the chest.

Alternatively, the catheter can be inserted under fluoroscopic control. It is, however, still important to ensure that a good pulmonary artery tracing is obtained with the balloon deflated, and a good wedge pressure is obtained with the balloon inflated.

Measurement

Specific details of pulmonary capillary wedge pressure measurement will vary according to the equipment available. There are, however, certain common features, in particular:

1 Most equipment is designed for continuous monitoring, as such it is precalibrated. Therefore the only major adjustment is to zero the transducer to atmospheric pressure before recording. To do this, ensure that the catheter is connected via a three-way tap to the manometer line, the other portholes of the three-way tap should be connected to a flushing system and to the air. Ensure that in setting up the equipment all air bubbles are removed from the system.
2 Move the three-way tap to ensure that blood cannot flow back from the catheter to the transducer but that the final port of the three-way tap is open to the air.
3 Adjust the tracing on the monitor to zero.
4 Close the transducer sidearm and open the transducer to the catheter. Ideally allow approximately 30 min for the transducer to warm up.

Measurements are made with the patient flat and the transducer at the angle of Louis. You will note that during measurements the pressure swings related to respiration will induce a biphasic nature to the pulmonary wedge pressure. It is therefore important the mean wedge pressure is utilised.

It is always important to:

- check the transducer level
- check the system is set at zero
- check that wedging does not occur.

Problems

1 Failure to wedge – reposition the catheter
2 Flat/damp trace – unblock catheter. Ensure that there is no air in the system and that the transducer is not open to both the patient and air. Flush the system – usually a hand flush of 1 ml of saline is required, but ensure that no air is introduced
3 Over wedging – occasionally the catheter is lodged in a pulmonary artery, unfortunately the diameter of this vessel is less than that of the balloon and does not allow accurate pressure recording. This usually manifests by a fluctuating, steadily increasing pressure trace. Ideally deflate the balloon and reposition the catheter.

Pulmonary capillary wedge pressure

This can be recorded as described above, along with pressures in the pulmonary artery, right ventricle and right atrium (Table 33.1).

The PCWP is an indirect reflection of left arterial pressure (LAP). This in turn is similar to left ventricular end diastolic pressure (LVEDP).

In certain acute medical conditions the PCWP does not accurately reflect LVEDP. With pulmonary venous obstruction, e.g. pulmonary emboli or raised intrathoracic pressure (for instance intermittent positive pressure ventilation) the PCWP is less than the LVEDP – thus PCWP is a particularly useful measurement in patients with poor left ventricular function and it may be used to optimise fluid therapy.

Table 33.1 Normal pressure ranges

Site	Pressure (mm Hg)
Right atrium (mean)	−1 to +6
Right ventricle	0 to 25
Pulmonary artery (mean)	10 to 20
Pulmonary capillary wedge pressure (mean)	8 to 15

CARDIAC OUTPUT

Cardiac output may be assessed using the Fick equation which relates cardiac output (CO) to oxygen uptake (VO_2). In this manner cardiac output equals oxygen uptake divided by the difference in arteriovenous oxygen content.

$$\text{Cardiac output} = \frac{\text{Oxygen uptake}}{\text{Arteriovenous oxygen content difference}}$$

$$\text{Therefore CO (1/min)} = \frac{VO_2 \text{ (ml/min)}}{CaO_2 - CvO_2 \text{ (ml/l)}}$$

To obtain these values a true mixed venous sample of blood must be taken from the tip of the pulmonary artery. This will allow the difference in the arteriovenous oxygen content to be assessed.

Complications

As with central venous access
Pulmonary parenchymal damage

PART VIII
Appendix

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Appendix: drugs commonly used in the management of medical emergencies

Drug	Indications	Dose and route	Notes
		IV infusion	
N-Acetyl cysteine	Paracetamol poisoning	150 mg/kg over 15 min then 50 mg/kg over 4 h, then 100 mg/kg over 16 h	Most effective if given less than 8 h post overdose. Requirement for treatment based on blood paracetamol levels at least 4 h post ingestion. Nomogram available on data sheet or British National Formulary. Treat at lower levels for at risk patients – alcoholics, anorexics and patients on liver enzyme inducing drugs
	Renal dysfunction in a patient with decompensated liver disease		Same dose for hepatorenal failure – continue 100 mg/kg every 16 h until improvement
Aciclovir	Herpes simplex encephalitis, varicella zoster virus in immunocompromised	100 mg/kg IV 8 hourly	Most effective if started at onset of infection. Can be used orally, topically or intravenously at lower dose for immunocompetent adults with herpes infections or prophylaxis in immunocompromised patients
Adenosine	Cardioversion of paroxysmal supraventricular arrhythmias	3–12 mg by rapid IV injection (see notes)	Do not use in Wolff–Parkinson–White syndrome with atrial fibrillation as increased conduction via accessory pathways may result in circulatory collapse or ventricular fibrillation. Use lower initial dose (0.5–1 mg) if heart transplant patient or patient taking dipyridamole (avoid use unless essential). Antagonised by theophyllines

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Drug	Indications	Dose and route	Notes
Adrenaline	Cardiac arrest	1 mg IV	Improves circulation achieved by chest compressions. Central line is the preferred route
		2 mg ETT	
	Anaphylaxis	0.5 ml 1 in 1000 IM	ECG monitoring necessary. Will frequently require adrenaline infusion after (see inotropic support next)
	Inotropic support	0.1–0.5 μg/kg/min IV	Give by continuous infusion (through dedicated line to avoid boluses). Predominantly increases cardiac output at lower doses. Also causes vasoconstriction at higher doses
Aminophylline	Acute severe asthma	5 mg/kg IV over 20 min	Do not give this initial loading dose to patients on oral theophyllines
		0.5 mg/kg/h IV	Vary infusion rate according to plasma theophylline levels (aim for 10–20 mg/l)
Amiodarone	Ventricular tachycardia, atrial fibrillation and flutter, supraventricular tachycardia	300 mg IV over 20–60 min, followed by 1200 mg/24 h IV	Effective antiarrhythmic with many complications. When given intravenously must be via a central venous catheter. Hypotension or cardiovascular collapse possible with rapid administration
Atenolol	Myocardial infarction	5–10 mg IV, followed by 50 mg orally at 15 min and 12 h, then 100 mg each day	Early use of atenolol post myocardial infarction reduces mortality. Should not be given to patients with a high degree of heart block, hypotension or overt left ventricular failure
Atropine	Asystole	3 mg IV	Used once only in the management of asystole
		6 mg ETT	
	Bradycardia	0.5 mg IV	Use incremental doses of 0·5 mg up to a maximum of 3 mg
Benzylpenicillin	Meningococcal septicaemia	2.4 g IV 4 hourly	Give immediately if meningococcal septicaemia suspected. If possible do blood cultures first. Do not delay for lumbar puncture
	Community acquired pneumonia	1–2 g qds	

Drug	Indications	Dose and route	Notes
Clarithromycin	Atypical pneumonia, other infections in penicillin allergic patients	500 mg IV 12 hourly	Similar spectrum of activity to erythromycin but slightly greater activity and higher tissue levels Fewer gastrointestinal side effects than erythromycin
	Community acquired pneumonia		Can be given orally
Diazepam	Fitting	5–10 mg IV, repeated if necessary 10–20 mg rectally	May cause respiratory depression and hypotension. Use of flumazenil to reverse this may precipitate further seizures. Use rectal route if IV access not easily attainable
Digoxin	Atrial fibrillation	500 μg IV over 30 min	Loading dose. Do not give if patient taking digoxin
		62.5–500 μg/day orally or IV	Used to control ventricular response rate. Does not cause chemical cardioversion. Maintenance dose. Be wary that arrhythmias may be caused by digoxin toxicity
Dobutamine	Cardiogenic shock	2.5–20 μg/kg/min	Inodilator. Give by continuous infusion. May cause paradoxical hypotension with increasing doses as a result of tachycardia and vasodilatation
Dopamine	Shock with inadequate urine output	1–3 μg/kg/min	Used as an adjunct to inotropic support. Increases renal blood flow and urine output
Frusemide	Pulmonary oedema secondary to left ventricular dysfunction	50–100 mg IV	Works initially by vasodilator effect, and later as diuretic. Use with extreme caution in hypotensive patients as severe hypotension may develop. Consider use in conjunction with inotropic support In the acutely anuric patient, bumetanide may be a better alternative as excretion of the drug into the tubule is not required. Higher doses required in patients on large oral doses of a loop diuretic or with known renal impairment

Drug	Indications	Dose and route	Notes
Glucagon	Hypoglycaemia	1 mg SC/IV/IM	Mobilises glycogen from the liver. If not recovered within 10 min give IV glucose
	β-Blocker overdose	50–150 μg/kg IV	Useful in shock refractory to atropine therapy in patients with β-blocker overdose. Is only available as 1 mg vials. Total dose required is up to approximately 10 mg
Glyceryl trinitrate	Pulmonary oedema secondary to left ventricular dysfunction	1–10 mg/h IV	In hypotensive patients, use only in conjunction with inotropic support
	Ischaemic chest pain	500 μg sublingual	If chest pain not rapidly relieved by nitrates, myocardial infarction should be excluded and alternative diagnoses considered
		1–5 mg buccal	
		1–10 mg/h IV	
Hydrocortisone	Anaphylaxis and angiooedema Bronchospasm	100–300 mg IV	Of secondary benefit as onset of action delayed for several hours. Use in more severely affected patients
	Acute adrenocortical insufficiency	100 mg IV 6–8 hourly	
Ipratropium bromide	Acute asthma	500 μg nebulised 4 hourly	Indicate in life-threatening asthma in conjunction with a β_2-agonist. In severe acute asthma use as a second line treatment. Beneficial in a small group of patients with chronic obstructive pulmonary disease
Lignocaine	Ventricular tachycardia	100 mg IV 1–4 mg/min	Commonly used to treat ventricular tachycardia
			Myocardial depressant. Use cautiously if impaired left ventricular function. Treat underlying cause of arrhythmia – usually myocardial ischaemia
	Local anaesthetic	3 mg/kg maximum	Infiltrate locally or perineurally. Facilitates procedures – large IV line, intercostal tube insertion, lumbar puncture. Increased dose (7 mg/kg) may be used if infiltrated with adrenaline (not fingers, toes, nose, ears or penis)

Drug	Indications	Dose and route	Notes
Lorazepam	Status epilepticus	4 mg IV	May cause respiratory depression or apnoea. Longer duration of action compared to diazepam
Metronidazole	Anaerobic infections including clostridium difficile	IV 500 mg 3 times daily	Avoid alcohol
Morphine	Myocardial infarction	2.5–20 mg IV (titrate against response)	Anxiolysis and analgesia reduce catecholamine levels, decreasing heart rate, afterload and hence myocardial oxygen consumption
	Pulmonary oedema	2.5–10 mg IV	Acts as above. Also effects on pulmonary vasculature reduce left ventricular preload
	Pain	2.5–20 mg IV (titrate against response)	Diamorphine is an alternative as may cause less hypotension and nausea. Powerful analgesic
			May cause respiratory depression
Naloxone	Opiate poisoning	0.4–2 mg IV 4 μg/min IV (increase dose as required to maintain required response)	Deliberate self-harm, iatrogenic or recreational use of opiates may result in respiratory arrest. Beware opiates with long half lives, especially methadone. IV infusion should be used if long acting opiate involved or recurrent coma or respiratory depression
Phenytoin	Status epilepticus	15 mg/kg loading dose IV at <50 mg/min	Second line drug in status epilepticus.
		Maintenance 100 mg IV 6–8 hourly	Phenytoin offers theoretical advantages as it can be infused rapidly. May cause central nervous system or cardiovascular depression, more marked with rapid infusion rates
Salbutamol	Acute asthma	2.5–5 mg nebulised as required	β_2-agonist. Nebulise with high concentrations of inspired oxygen
		250 μg IV	In severe or life-threatening acute asthma not responding to nebulised β_2-agonist, IV therapy is indicated. Consider need for anaesthetic help. Infusions of salbutamol are used following IV bolus
		3–20 μg/min IV	

Drug	Indications	Dose and route	Notes
Streptokinase	Myocardial infarction	1.5 million units IV over 1 h	Reduces mortality post myocardial infarction Indicated when potential benefits outweigh risks Risks mainly relate to haemorrhage (see Chapter 10)
Tazocin	Community acquired pneumonia, chest infection or urinary sepsis in the acutely ill	4.5 g Tazocin EF every 8 h	
tPA	Myocardial infarction	15 mg bolus followed by 50 mg over 30 min, then 35 mg over 60 min IV	See streptokinase. May have mortality benefits in some subgroups compared to streptokinase. Use dictated by local protocols, commonly including patients with anterior myocardial infarction or hypotension related to myocardial infarction
	Pulmonary embolism	10 mg bolus followed by 90 mg over 2 h	Use in haemodynamically significant pulmonary embolus or pulmonary embolus causing severe hypoxaemia despite high FiO_2

IM, intramuscular; IV, intravenous; ETT, endotracheal tube; SC, subcutaneous; tPa, tissue plasminogen activator.

Answers to time out questions

CHAPTER 2: RECOGNITION OF THE MEDICAL EMERGENCY

Time Out 2.1 (see page 7)

This time out has allowed to reflect on your own practice and prioritise the importance of the components of your assessment.

Time Out 2.2 (see page 11)

Ensure that your list of clinical features is in a logical order.

CHAPTER 3: A STRUCTURED APPROACH TO MEDICAL EMERGENCIES

Time Out 3.1 (see page 24)

The primary assessment would comprise:

- Airway – assess patency. As the patient is talking no intervention at this stage is required except for high concentration of inspired oxygen (FiO_2 0.85).
- Breathing – assess rate, effort and symmetry of respiration. Look for an elevated JVP whilst palpating the trachea for tug or deviation. Percuss the anterior chest wall in upper, middle and lower zones, and in the axillae. Listen to establish whether breath sounds are absent, present or masked by added sounds. As no abnormality has been detected arterial oxygen saturation can be measured using the pulse oximeter.
- Circulation – assess pulse – rate, rhythm and character; blood pressure and capillary refill time. If there is no evidence of shock, a single cannula is inserted and blood taken for baseline haematological and biochemical values including a serum glucose. A bedside measurement of glucose is also important. Continuous monitoring of pulse, blood pressure and ECG will provide valuable baseline information as will a 12-lead ECG. The BM stix shows the glucose to be 1.2 mmol/l. The patient is therefore immediately treated with 250 ml 10% dextrose while the assessment continues.
- Disability – assessment of pupils – mildly dilated, symmetrical and slowly reacting to light. GCS 13/15: E4, V4, M5, no obvious lateralising signs.
- Exposure – no evidence of acute skin rash. Core temperature 36.8°C.

This assessment would be repeated and the patient would be monitored until the blood glucose had returned to normal. If the patient's conscious level did not change, however, treatment would continue to prevent secondary brain injury while reassessment and further investigations were requested. In contrast, if the patient's condition did improve then it would be appropriate to start the secondary assessment.

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Time Out 3.2 (see page 28)

Primary assessment:

a Reassess the patient

b Shock – likely hypovolaemic

c Continue with high concentrations of inspired oxygen and give a fluid challenge, then reassess the patient. Correction of hypovolaemia restores sinus rhythm.

CHAPTER 4: AIRWAY ASSESSMENT

Time Out 4.1 (see page 40)

- Look – paradoxical (see-saw movement of the chest and abdomen in complete obstruction due to increased respiratory effort), accessory muscle use.
- Listen – stridor indicates upper airway obstruction, wheezes usually signify obstruction of the lower airways. Crowing accompanies laryngeal spasm while snoring indicates that the pharynx is partially occluded by the tongue. Gurgling usually signifies presence of semi-solid material.
- Feel – for expired air against the side of your cheek, chest movement, the position of trachea, any tracheal tug and the presence of subcutaneous emphysema.

CHAPTER 5: BREATHING ASSESSMENT

Time Out 5.1 (see page 53)

a

i This is the amount of air inspired per breath and is equivalent to 7–8 ml/kg bodyweight or 500 ml for the 70-kg patient.

ii This is the amount of air inspired each minute and is calculated by multiplying the respiratory rate by the tidal volume.

$$15 \text{ breaths/min} \times 500 \text{ ml} = 7.5 \text{ l/min}$$

b Alveolar ventilation can be calculated from the respiratory rate × (tidal volume – anatomical dead space). The anatomical dead space is constant. However as the respiratory rate increases, the amount of inspired air per breath or tidal volume is reduced. Therefore, as the respiratory rate increases in particular over 20 breaths/min, the tidal volume is reduced dramatically as is the alveolar ventilation. For further details the reader is referred to Chapter 5.

c This is shown in Chapter 5. The important feature however is that the relationship between the PaO_2 and O_2 saturation of haemoglobin is not linear. This means that haemoglobin O_2 saturation is initially well maintained over a very wide arterial oxygen concentration from 50 to 100 mm Hg.

d

- Airway obstruction
- Breathing
  – bronchospasm, pulmonary oedema, tension pneumothorax
  – critical oxygen desaturation.

CHAPTER 6: CIRCULATION ASSESSMENT

Time Out 6.1 (see page 63)

Please see Fig. 6.2.

Time Out 6.2 (see page 64)

- History of asystole

- When there is any pause ≥3 s in the presence of Mobitz Type II or complete heart block with wide QRS complexes. Clinical features that indicate treatment with atropine include cardiac failure, systolic blood pressure ≤90 mm Hg, heart rate <40 beats/min, presence of ventricular arrhythmias compromising blood pressure.

Time Out 6.3 (see page 65)

There are many ways to remember the causes of shock and one system in use is the preload, pump, afterload, (peripheral classification often referred to as the three Ps). Preload causes of shock are due to hypovolaemia that may be real, e.g. following haemorrhage, profuse diarrhoea or vomiting; and apparent, due to venodilation following treatment with intravenous nitrates. In addition, venous return can be obstructed by a gravid uterus, severe asthma or tension pneumothorax. Pump problems include severe left or right ventricular failure, cardiac tamponade. Peripheral or afterload causes are associated with widespread vasodilation (reduced systemic vascular resistance) seen with anaphylaxis, systemic inflammatory response syndrome including septicaemia and toxaemia and neurogenic shock.

CHAPTER 7: DISABILITY ASSESSMENT

Time Out 7.1 (see page 84)

See Fig. 7.5 showing dermatomes.

CHAPTER 8: THE PATIENT WITH BREATHING DIFFICULTIES

Time Out 8.1 (see page 89)

Key components of the assessment so far:

Airway:

- Look
- Listen
- Feel

Breathing:

- Look – colour, sweating, posture, respiratory effort, rate and symmetry
- Feel – tracheal position, tracheal tug, chest expansion
- Percuss
- Listen.

Time Out 8.2 (see page 90)

a Rapid primary assessment and treatment with:

A High concentrations of inspired oxygen

B Assessment indicates pulmonary oedema

C Supports the diagnosis of left ventricular failure with hypotension therefore cardiogenic shock, so the patient requires intravenous access and, after appropriate bloods have been taken including markers of myocardial damage, inotropes should be started.

b Investigations should include a full blood count to ensure that there is no anaemia, baseline renal function and blood glucose, chest X-ray and 12-lead ECG. The patient will also require appropriate monitoring including pulse oximetry and continuous ECG.

Time Out 8.3 (see page 96)

a This is a chronic inflammatory condition resulting in reversible narrowing of the airways.

b A susceptible airway in which bronchospasm may occur precipitated by IgE mast cell degranulation or exposure to environmental factors which will induce chronic inflammation. Bronchial contraction, mucosal oedema, increased mucous production and epithelial cell damage will drive the inflammatory response and exacerbate the airway narrowing. Persisting inflammation will induce collagen deposition under the basement membrane.

c Airway narrowing.

d It reduces the forced expiratory volume and peak expiratory flow rate. There is also increased functional residual capacity due to air trapping but no change in total lung capacity. Thus because of increased airways resistance, the work of breathing is increased and hence the patient feels breathless. In addition, in an acute attack some of the airways may be blocked by mucous plugs resulting in hypoxemia due to ventilation perfusion mismatch. This will also increase the work of breathing.

e By giving high concentrations of inspired oxygen.
Nebulised – (a) salbutamol 5 mg or terbutaline 10 mg; (b) ipratroprium bromide 0.5 mg or given via an oxygen driven nebuliser.
Intravenous – (a) hydrocortisone 200 mg; (b) salbutamol 250 μg over 10 min. Alternatively terbutaline or aminophylline can be used.
Chest X-ray to exclude a pneumothorax.

f The conditions are:
- **i** Hypoxaemia (PaO_2 <8 kPa despite FiO_2 >0.6)
- **ii** Hypercapnia ($PaCO_2$ >6 kPa)
- **iii** Exhaustion
- **iv** Altered conscious level
- **v** Respiratory arrest

Time Out 8.4 (see page 101)

a *Streptococcus pneumoniae*

b The patient may experience prodromal features of malaise, anorexia, myalgia, arthralgia and headache; there may also be a history of pyrexia and sweating. In addition, the patient will have had a cough productive of sputum and experience breathlessness, possible pleuritic pain and even haemoptysis. One third of patients may develop herpes simplex labialis. It is important to remember however that elderly patients may remain afebrile.

c High concentration of inspired oxygen, titrated to the arterial blood gas results, intravenous fluids and antibiotics according to local policy such as benzylpenicillin 1.2 g qds and clarithromycin 1 g daily.

d High risk factors in patients with pneumonia are summarised as the CURB 65 score (see table on page 105).

Time Out 8.5 (see page 106)

a This is pneumonia developing more than 48 h after admission to hospital.

b Those patients who are ill, bed-bound and who have impaired consciousness. This may be exacerbated by an inability to clear bronchial secretions, e.g. after a general anaesthetic or thoracic and abdominal surgery where coughing is impaired. The risk of a post operative pneumonia is also exacerbated in the

elderly and those patients who have a history of smoking, obesity and underlying chronic illness.

c Make sure that they are on supplemental oxygen and intravenous fluids along with an appropriate antibiotic regime. As there is a wide range of potential organisms, an early liaison with a microbiologist is advocated. Appropriate antibiotic regimes include tazocin, metronidazole plus gentamicin. If pseudomonas is suspected then either ceftazidime or ticarcillin may be required.

Time Out 8.6 (see page 107)

See Fig. 8.6 on page 103.

Time Out 8.7 (see page 108)

Larger emboli that block larger branches of the pulmonary artery provoke a rise in pulmonary artery pressure and rapid shallow respiration. Tachypnoea is also a reflex response to activation of vagal innovated luminal stretch receptors and interstitial J-receptors within the alveolar and capillary network.

CHAPTER 9: THE PATIENT WITH SHOCK

Time Out 9.1 (see page 125)

a The five factors are:

- **i** Concentration of oxygen reaching the alveoli
- **ii** Pulmonary perfusion
- **iii** Adequacy of pulmonary gas exchange
- **iv** Capacity of blood to carry oxygen
- **v** Blood flow to the tissues

b The sympathetic nervous system can help in several ways – increasing venous return by reducing the diameter of the veins and hence the capacity of the venous system; positively inotropic and the positively chronotropic effect.

Time Out 9.2 (see page 145)

a Clear, and if necessary, secure airway
Give high concentrations of inspired oxygen

b Measure SpO_2
Check for signs of aspiration
Monitor respiratory rate
Book chest X-ray

c Intravenous access × 2, start fluid replacement in one and take bloods from the other for FBC, U&E, glucose, clotting profile, cross match 4 units.
Monitor pulse, blood pressure, 12-lead ECG and urine output
Liaise with gastroenterologist/upper GI surgeon
Subsequent management will depend on the response to fluid resuscitation, ongoing haemorrhage, change in physical/monitored signs.

CHAPTER 11: THE PATIENT WITH ALTERED CONSCIOUS LEVEL

Time Out 11.1 (see page 168)

a Consciousness is a function of the integrated action of the brain. The two interlinked key areas are the reticular formation and the cerebral cortex.

b This assessment comprises:
- Pupillary response
- Eye movement
- Corneal response
- Respiratory pattern.

Time Out 11.2 (see page 185)

a These develop on medium sized arteries at the base of the brain and the commonest sites are the distal internal carotid/posterior communicating artery and the anterior communicating artery complex.

b Intracranial saccular aneurysm and arterio venous malformation

c Greater than 12 h

d 25%

e *Streptococcus pneumoniae* and *Neisseria meningitidis*

f The immunocompromised patients including those with the human immuno deficiency virus; immigrants from Pakistan, India, Africa and the West Indies; alcoholics and intravenous drug users; patients with previous pulmonary tuberculosis.

g Gustatory and olfactory hallucinations, amnesia, expressive dysphasia, temporal lobe seizures, anosmia and behavioural abnormalities. This specific symptom complex occurs because herpes simplex encephalitis involves primarily the temporal and frontal cortex.

h Cerebral malaria should be considered a differential diagnosis of any acute febrile illness until it can be excluded by – definite lack of exposure, repeat examination of blood smears, following a therapeutic trial of antimalarial chemotherapy.

i Non-neurological features of *Plasmodium falciparum* infection include anaemia, spontaneous bleeding from the GI tract, jaundice, hypoglycaemia, shock, oliguria, acute renal failure and pulmonary oedema.

j Although intracranial abscesses can be caused by infection from sinuses or penetrating trauma, intracerebral abscesses can also follow septicaemia due to infective endocarditis, pulmonary abscess and bronchiectasis. Extradural abscesses are difficult to diagnose and may present with a localised headache in association with mastoiditis and sinusitis. In contrast, subdural and intracerebral abscesses present with headache, vomiting, impaired consciousness and neurological signs.

k An extradural haematoma follows a tear to the middle meningeal artery. It is a classic sequence of events after the head injury when the patient becomes unconscious, develops a lucid interval and then becomes comatosed. In contrast, subdural haematoma usually occurs in patients who are over anticoagulated or following falls in the elderly or alcoholic patients. Intracerebral haematoma occurs spontaneously and the clinical features and signs will be dictated by the area of the brain that has been affected.

CHAPTER 12: THE 'COLLAPSED' PATIENT

Time Out 12.1 (see page 201)

a Stroke is a syndrome characterised by an acute onset of focal, but occasionally global, loss of function lasting more than 24 h. This can be brought about by a number of causes including reduction in cerebral blood flow along a known vascular pathway affecting neurological tissue, generalised cerebral hypoperfusion from whatever cause and localised vascular disease.

b The patient should have a rapid primary assessment with reference to a serum glucose estimation.

CHAPTER 14: THE PATIENT WITH A HEADACHE

Time Out 14.1 (see page 221)

a These can be classified as intracranial – e.g. meningitis, encephalitis and subarachnoid haemorrhage, or extracranial – e.g. acute sinusitis, acute viral illness, malaria or typhoid.

b Weakness usually affects the proximal and distal limb muscles equally, wasting occurs but is not prominent, reflexes are diminished or absent. Sensation may be unaffected although there may be variable loss.

Time Out 14.2 (see page 226)

a Initial diagnosis includes acute sinusitis, cervical spondylosis, giant cell arteritis and acute glaucoma.

b Acute sinusitis – orbital cellulitis/abscess, meningitis, cerebral abscess, osteomyelitis and cavernous sinus thrombosis.

Cervical spondylosis – spinal cord compression.

Giant cell arteritis – visual problems including blindness ischaemia/infarction of the heart, intestine and brain; acute glaucoma. Ischaemia of the optic nerve and retina.

Time Out 14.3 (see page 231)

a Tension headache – diffuse, commonly at the vertex frequently bilateral and described as a pressure tight band or squeezing. Usually starts in the morning and increases throughout the day. There is no vomiting and no visual disturbance.

Migrainous headaches can be paroxysmal, unilateral, bilateral and are often described as throbbing. In the minority of patients 20% will develop visual aura or some sensory disturbance.

Cluster headaches – unilateral with ipsilateral corneal injection, nasal congestion, and possibly a transient Horner's syndrome. The headache is usually centred around the orbit and lasts for between 30 min and 2 h each day for between 4 and 16 weeks.

b A lumbar puncture must be considered in any patient who has an acute onset of a headache that is new, progressive and awakes them from a sleep, especially those people who have a history suggestive of coital migraine.

CHAPTER 15: THE PATIENT WITH ABDOMINAL PAIN

Time Out 15.1 (see page 238)

a The differential diagnosis is:

i Consider:

- Leaking abdominal aortic aneurysm
- Gastrointestinal bleeding (e.g. bleeding peptic ulcer)
- Acute pancreatitis
- Severe gastroenteritis
- Cardiogenic shock (acute myocardial infarction)
- Small bowel infarction (mesenteric artery occlusion)
- Sepsis (e.g. colonic perforation, pneumonia)

ii Consider:
- Ectopic pregnancy
- Gastrointestinal bleeding (e.g. bleeding peptic ulcer)
- Acute pancreatitis
- Severe gastroenteritis/ulcerative colitis
- Diabetic ketoacidosis
- Ruptured spleen (spontaneous rupture occurs rarely in infectious mononucleosis)
- Sepsis (e.g. lower lobe pneumonia, meningococcal septicaemia)
- Acute adrenal insufficiency.

b The management priorities are:
- Is the airway unprotected and is there a risk of aspiration (particularly in the patient with vomiting and/or a depressed level of consciousness)? If so, secure the airway. Start high concentrations of inspired oxygen by facemask with a non-rebreathing reservoir bag.
- Examine the chest. Is ventilation and gas exchange adequate? If not, consider the need for intubation and ventilation (e.g. in patients with severe acute pancreatitis or septic shock).
- Assess degree of circulatory failure. Obtain vascular access with two large bore (peripheral) cannulae; take samples for blood cross match, baseline haematology and biochemistry (including amylase and glucose stick test), blood gas analysis, and – when appropriate – coagulation screen, β-hCG pregnancy test and sickle cell screen. For hypovolaemia initiate fluid resuscitation with 0.9% saline, followed by blood for haemorrhagic shock.
- 12-lead ECG if myocardial infarction/arrhythmia/pulmonary embolism suspected; urgent chest X-ray for pneumonia or other chest pathology; establish monitoring of SaO_2, ECG and BP.
- Perform abdominal, rectal and – if indicated – vaginal examination.
- Urgent surgical or gynaecological referral and/or other emergency treatment (analgesia, antibiotics), as appropriate.
- Consider the need for nasogastric tube and/or urinary catheter.
- Perform/arrange a portable ultrasound scan where this may confirm the diagnosis (e.g. suspected abdominal aortic aneurysm).

Reassess and go on to complete the secondary assessment.

Time Out 15.2 (see page 254)

Adverse prognostic factors in acute pancreatitis (within 48 h):
- Age >55 years
- White blood cell count $>15 \times 10^9$/l
- Blood glucose >10 mmol/l (no diabetic history)
- Serum urea >16 mmol/l (no response to IV fluids)
- PaO_2 <8 kPa
- Serum calcium <2.0 mmol/l
- Serum albumin <32 g/l
- Lactate dehydrogenase >600 U/l.

CHAPTER 16: THE PATIENT WITH HOT RED LEGS OR COLD WHITE LEGS

Time Out 16.1 (see page 263)

Post-operative, immobile, pregnant patients, women on the oral contraceptive pill, family history of coagulopathy.

Time Out 16.2 (see page 268)

- Arterial emboli tend to occur at the bifurcation of arteries. These will depend on the extent of the occlusion of the circulation and the degree of colateral circulation.
- Medical features include pain, pallor, pulselessness, parathesia, paralysis and perishing cold.
- In contrast, a closed compartment syndrome is caused by a swollen or a contused muscle or bleeding into the muscle from inside a rigid fascial envelope. Pain and parasthesia are early symptoms but the affected limb may also be pale and cool with slow capillary refill. However the presence of a distal pulse does not help diagnosis.

CHAPTER 17: THE PATIENT WITH HOT AND/OR SWOLLEN JOINTS

Time Out 17.1 (see page 280)

There are many algorithms but the important step is the first step which is to exclude a septic arthritis.

CHAPTER 18: THE PATIENT WITH A RASH

Time Out 18.1 (see page 292)

a The four categories are:
- **i** Urticaria
- **ii** Erythema
- **iii** Purpura and vasculitis
- **iv** Blistering disorders

b The reader is referred to Chapter 18 for the algorithms for each of these conditions.

CHAPTER 20: ORGAN FAILURE

Time Out 20.1 (see page 318)

The immediate management comprises a rapid primary assessment to ensure airway patency. Her FiO_2 should be increased to 0.85. Breathing must be reassessed to exclude life-threatening bronchospasm, tension pneumothorax and pulmonary oedema. She should be treated with nebulised bronchodilators including salbutamol and ipratropium, along with intravenous hydrocortisone and a bronchodilator. An urgent chest X-ray is required. The result is a right-sided pneumothorax. Whilst there are no clinical features to indicate underlying tension, even a small pneumothorax in a person with pre-existing chest disease can cause rapid decompensation. Therefore a chest drain is also required.

Time Out 20.2 (see page 341)

a The causes are:
- **i** Acute asthma; pulmonary embolus; cardiac, e.g. dysrhythmia; neurological, e.g. status epilepticus; neuromuscular, e.g. myasthenia gravis.
- **ii** The commonest cause is ischaemic heart disease. Others include valvular pathology, acute hypertension, cardiomyopathy.
- **iii** Any chronic neurological disorder can have the final common pathway of brain failure. Dementia is another cause. These are not acute medical emergencies, however, the important point is to be able to differentiate these

conditions from potentially treatable problems such as a patient with an acute confusional state or underlying depression.

iv These may be classified as prerenal, intrinsic renal or post renal conditions. The commonest group is the prerenal, which usually arises as secondary to hypovolaemia. The second most common cause is post-renal or obstructive uropathy, e.g. in association with prostatic pathology. Intrinsic renal disease in comparison is rare.

v Acute liver failure is usually caused by drugs such as an overdose of paracetamol and with increasing frequency of Ecstacy. It can also occur in pregnancy. Acute on chronic liver failure is commonly caused by alcoholic liver disease.

vi The causes of endocrine failure will depend on the particular gland that is affected and also the hormone or hormones that are not being produced. Irrespective of these conditions considered, there are common features that influence all aspects of the primary assessment.

CHAPTER 21: THE ELDERLY PATIENT

Time Out 21.1 (see page 354)

10%

CHAPTER 22: TRANSPORTATION OF THE SERIOUSLY ILL PATIENT

Time Out 22.1 (see page 366)

A 27-year-old mechanic with a subarachnoid haemorrhage is stable with a respiration rate of 14/min, sinus tachycardia 110/min, BP 120/70, blood glucose by BM stick test 7 mmol/l, GCS 15/15 PERLA. The decision has been made to transfer this patient to the local neurological centre for assessment before surgery.

Assessment

Male, 27, clinical details as described above.

- What is the problem?
  Diagnosis – subarachnoid haemorrhage.
- What has been done?
  Prevention of secondary brain injury, CT scan and lumbar puncture.
- What was the effect?
  Maintaining the status quo and preventing secondary brain injury.
- What is needed now?
  Transfer for further assessment.
  There are potential problems that may arise during transfer:

Airway – obstruction, hypoxaemia
Breathing – hypocarbia respiratory arrest
Circulation – cardiac arrest, dysrhythmia, hypoglycaemia, hyponatremia
Disability – deterioration in Glasgow Coma Score, fit, extension of subarachnoid haemorrhage, development of raised intracranial pressure.

Control

A comprehensive assessment by the clinician in charge and the decision has been made to transfer the patient to hospital for further investigation.

Communication

With:
The consultant
Intensive care consultant
Patient's relatives
Accepting consultant
Ambulance control

Communication also includes determining the lines of responsibility and using the assessment questions to provide a structure to tell the receiving team the salient points before transfer. In addition, the reason for transfer and what is needed for the receiving centre should be explained.

Evaluation

The need for specialist care has already been determined as part of the initial assessment.

Package and preparation

The patient has been stabilised before transfer and all baseline blood tests including arterial gases have been requested, reviewed and appropriate action taken. All relevant equipment monitoring and treatment has been pre-packed and checked. Furthermore, this also includes contingency equipment that should be required in case the patient deteriorates and incurs one of the problems that were listed earlier. The neurosurgical centre is 30 miles away by motorway with no predicted problems and a stable patient, so the decision has been made to transport by ambulance.

Personnel

The ambulance crew
One doctor
One nurse

Part of the regular transfer team has been briefed and have the appropriate personnel. Ensure all personal equipment is available and working. The ambulance should have appropriate monitoring equipment and back up systems including oxygen should any problems arise. Shortly before transfer, all the patient's documentations have been photocopied and appropriate forms are available to record the patient's condition during transfer.

Index

Note: Page numbers in **bold** type refer to figures; those in *italic* refer to tables or boxed material.

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