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The Complications of Musculoskeletal Conditions and Trauma: Preventing Harm

Julie Santy-Tomlinson[1], Sonya Clarke[2], and Peter Davis[3]

[1] *International Journal of Orthopaedic and Trauma Nursing*
[2] *Queen's University Belfast, Belfast, UK*
[3] *Newark, Nottinghamshire, UK*

Introduction

Patient harm during healthcare is a leading cause of morbidity and mortality, and early detection and prevention of patient harm is a priority (Panagioti et al. 2019). This includes preventable complications following orthopaedic and trauma care. The aim of this chapter is to provide evidence-based guidance for the identification of risk, detection, prevention and management of those complications which are significant risks for the orthopaedic and trauma patient. Preventable complications are major causes of both morbidity (complications) and mortality (death), and are of considerable significance in providing evidence-based care. Harm to a patient following musculoskeletal care and procedures is almost always the result of one or more complications which can also lead to poorer outcomes, patient distress and discomfort, significant delays in recovery and increased costs. Much care provided in both the acute, rehabilitation and community setting is aimed at minimising the potentially harmful effects of four factors that lead to complications:

- *tissue injury* to bone and/or soft tissue due to trauma or surgery
- *surgery*, i.e. the effects of anaesthesia and surgical procedures
- *reduced mobility* because of musculoskeletal conditions, injury or surgery and associated care
- *stasis* of major body systems because of reduced mobility.

It is worth noting poor life choices may predispose patients to an increased risk of complications, delayed recovery and death when musculoskeletal conditions and trauma occur. For example, lack of activity/exercise, stress, reduced sleep, poor nutrition and hydration, excessive alcohol and smoking. This chapter describes appropriate nursing interventions based on Roper et al.'s (2000) activities of daily living. This model is still used to identify and direct nursing care today even though it was first described in the 1970s. Those activities relevant to this chapter are:

1) breathing
2) eating food and drinking fluids
3) eliminating body waste
4) mobilising
5) sleeping.

Finally, while there are many potential complications for the orthopaedic and trauma patient, this chapter will focus on those which are either the most common or likely to result in the most significant harm: infection, shock, venous thromboembolism (VTE), fat embolism syndrome (FES), acute compartment syndrome (ACS), urinary retention, urinary tract infection (UTI), respiratory tract infection and constipation.

Infection

The human body is constantly exposed to microorganisms from the environment. The immune system provides a range of defence mechanisms against infection, which include physical, chemical, innate and adaptive responses to attack by organisms. A large and diverse community of commensal organisms (which inhabit human mucosa and skin without causing harm) play an important role in defence against pathogenic (causing diseases) organisms.

Orthopaedic and Trauma Nursing: An Evidence-based Approach to Musculoskeletal Care, Second Edition. Edited by Sonya Clarke and Mary Drozd.

Infections can occur when damaged or vulnerable tissue is exposed to harmful pathogens (such as bacteria, viruses and fungi), leading to a complex tissue response brought about by the multiplication of and attack by such microorganisms. This depends on the susceptibility of the patient and the virulence of the organism. Potentially harmful organisms such as bacteria, viruses and fungi may *contaminate* tissue. Multiplication of the organisms may then lead to *colonisation*. Infection is not, however, considered to be present until attack from a pathogenic organism results in an acute or chronic tissue reaction. Bacteria may contaminate or colonise tissue without causing infection. When the patient's immune system is compromised due to factors such as age, ill-health or depleted nutrition, colonisation is more likely to progress to *infection* and host defences are less effective in fighting infection.

Both tissue injury and infection result in an inflammatory reaction, which is part of the immune response; a distinct reaction brought about by both chemical and physical phenomena which results in the 'cardinal' signs of inflammation/infection: redness, pain, swelling and heat. There may also be increased exudate. If the organism causing infection is 'pyogenic' (pus producing) collections of pus may also form as abscesses.

Infection is best diagnosed by observing the tissue response to microbial invasion and the subsequent symptoms of infection as a manifestation of the inflammatory response. This response will vary depending on the infecting organisms and the tissue or system affected. They can include:

- pain, swelling, redness and heat at the site of infection, in the surrounding area or deep within the tissues
- loss of function of the area affected, particularly if pain and/or swelling affect joints and other musculoskeletal structures
- tissue exudate, which may or may not contain pus
- pyrexia and/or
- generally feeling unwell, with malaise or lethargy.

A diagnosis of infection should be made based on the symptoms reported by the patient. This can be augmented, but not replaced by, microbiological culture and analysis of wound samples in the laboratory.

The orthopaedic and trauma patient is particularly vulnerable to the following types of infection:

- wound infection (Chapter 12): categorised as either surgical site infection (SSI) or traumatic wound infection
- bone infection (osteomyelitis) and joint infection (infective arthritis) (Chapter 13)
- UTI
- respiratory tract infection.

Healthcare-associated infection is the main cause of infection in orthopaedic and trauma patients, acquired by transfer from one person or surface to another. The way by which an infection can spread involves five links in the chain of infection (Public Health England 2017). Understanding how the links are made is important in understanding the ways in which the chain can be broken and infection prevented:

1) *A causative organism*: a pathogenic organism is present which can cause infection
2) *A reservoir of infection*: a place (human or environmental) which provides conditions for the organism to multiply
3) *A portal of exit*: allows the organism to leave the reservoir, e.g. in body fluids, on the skin (particularly the hands) and in various body fluids such as respiratory droplets
4) *A mode of transmission*: a method through which the organism is spread to another person, e.g. through body fluids, the hands of patients and healthcare workers, ingestion and airborne transmission
5) *A susceptible patient*: vulnerable to infection because their immune response is compromised, e.g. hospitalised patients, the injured, surgery, the very young, the very old (and frail) and those with concurrent medical conditions and malnutrition.

A significant concern is also the ability of remote infections such as UTIs and SSIs to transfer to sites of orthopaedic implants because of 'seeding' of bacteria to implant sites. The prevention of infections in orthopaedic patients is particularly important because of the potentially devastating consequences of transfer of infection to bone and subsequent osteomyelitis, which is difficult to eradicate and results in long-term pain and distress; the avoidance of this is a central aim of infection control in orthopaedic settings. Infection may also lead to life-threatening sepsis (see Sepsis and septic shock section).

Infection Prevention

Prevention of infection measures has been standardised because of a large body of amassed evidence that demonstrates the most effective approaches (see Box 9.1 for an example of evidence-based guidelines), which include the following interventions (Loveday et al. 2014):

- *Hospital environmental hygiene* through rigorous cleaning processes and assessment of cleanliness, equipment decontamination.
- *Hand hygiene/hand decontamination*: many healthcare-associated infections are transferred from one person to the other on the hands of healthcare staff.

Box 9.1 Evidence Digest

Evidence-based guidelines for preventing healthcare-associated infections

Many counties have developed national guidelines for the prevention of healthcare-associated infection. The epic 3 guidelines (Loveday et al. 2014) provide an example from England. These were commissioned by the Department of Health (England) and revised, reviewed and updated for a second time in 2014. They were created by a nurse-led multiprofessional team who undertook extensive consultation using multiple systematic reviews of the evidence and other guidelines as well as expert opinion, demonstrating use of all levels of evidence available at that time. The guidelines are freely available as open access at https://doi.org/10.1016/S0195-6701(13)60012-2.

The guidelines describe in detail the interventions and precautions practitioners should take to prevent healthcare-associated infection, which include the recommendations outlined in Loveday et al. (2014). These precautions are designed to break the chain of infection. The authors point out that effective infection prevention and control are essential in ensuring patient safety and preventing harm. The orthopaedic and trauma team must incorporate such national, regional and local guidelines into everyday practice to reduce infection risk during care and intervention.

- The use of *personal protective equipment* to provide a barrier between the healthcare provider and a source of infection.
- The *safe use and disposal of sharps*: there is a high risk of blood-borne infection from accidental inoculation with contaminated sharps.
- Preventing infections associated with the use of *short-term indwelling urethral catheters*, which provide a major portal for infection.
- Preventing infections associated with *central venous catheters*, which are linked with blood-borne infection.

Evidence has shown that effective hand hygiene is the most effective method of preventing the transfer of infection. National and international guidelines emphasise the importance of adherence to hand hygiene guidance and provide an overview of the barriers and factors that influence hand hygiene compliance (Loveday et al. 2014). Compliance is much lower than the target of 100% (Box 9.2) and measures are needed to ensure that compliance is as high as possible (Tromp et al. 2012).

Box 9.2 Evidence Digest

Hand hygiene compliance: what the evidence says

Hand decontamination is a universally accepted elementary method for infection prevention. It is easy, cheap and quick to perform. Remarkably, compliance with the five moments of hand hygiene (HH) (WHO 2009) is consistently poor among all health professionals in every setting. A systematic review conducted by Erasmus et al. (2010) found that 60% of HH opportunities are missed. Some studies have already been conducted which show that even during the COVID-19 pandemic healthcare workers returned to poor compliance levels surprisingly quickly (Stangerup et al. 2021). The reasons for poor HH compliance among practitioners are complex and every practitioner will be subtly aware of their own reasons for such failures in safe practice.

Many studies have also been conducted which consider the interventions most likely to improve HH compliance. Researchers in the USA (Sands and Aunger 2020) conducted a survey that considered the factors, barriers and levers that influence HH among nurses. They found the following situations were associated with higher compliance:

- leadership
- conducting tasks perceived as dirty
- access to HH facilities and materials
- availability of alcohol rub/gel
- good practice examples of other team members
- feedback on performance.

However, a systematic review by Doronina et al. (2017) found that education regarding HH is forgotten as little as a month later. While all staff should undergo regular education to support compliance and to ensure that skills are up to date and embedded in their practice, there is a need to consider how this can be maintained during daily practice.

There are three common healthcare-associated infections of particular concern to the orthopaedic and trauma patient: SSI, respiratory tract infection and UTI. Several evidence-based guidelines exist globally which provide the practitioner with best practice advice for the prevention and management of each of these types of infections. Some of these are listed in Box 9.3.

Box 9.3 Evidence-based Guidance for the Prevention and Management of Surgical Site Infection and Respiratory Tract Infection

Surgical site infection

Centers for Disease Control and Prevention (CDC) (2017) Guideline for the Prevention of Surgical Site Infection. https://jamanetwork.com/journals/jamasurgery/fullarticle/2623725.

Ling, M.L., Apisarnthanarak, A., Abbas, A. et al. APSIC guidelines for the prevention of surgical site infections. Antimicrob Resist Infect Control 8, 174 (2019). https://doi.org/10.1186/s13756-019-0638-8.

National Institute for Health and Care Excellence (NICE) (2019) Surgical site infections: prevention and treatment. NICE Guideline NG12G. www.nice.org.uk/guidance/ng125.

World Health Organisation (2018). Global guidelines for the prevention of surgical site infection, 2nd edn. World Health Organisation. https://apps.who.int/iris/handle/10665/277399.

Respiratory tract infection

NICE (2019) Pneumonia (hospital-acquired): antimicrobial prescribing. NICE guideline [NG139]. www.nice.org.uk/guidance/NG139. (Note: NICE withdrew their guideline Pneumonia in adults: diagnosis and management. Clinical guideline [CG191] in 2019 during the COVID pandemic and stated that they are reviewing the recommendations.)

Pássaro, L., Harbarth, S. & Landelle, C. Prevention of hospital-acquired pneumonia in non-ventilated adult patients: a narrative review. Antimicrob Resist Infect Control 5, 43 (2016). https://doi.org/10.1186/s13756-016-0150-3.

Urinary tract infection

Centers for Disease Control and Prevention (CDC) (2009) Guideline for Prevention of Catheter-Associated Urinary Tract Infections. https://www.cdc.gov/infectioncontrol/guidelines/cauti/index.html.

Surgical Site Infection

SSI is a major risk of orthopaedic surgery, particularly following implantation of, for example, internal fixation devices or arthroplasty. After internal fixation of fractures SSI rates have been reported to be as high as 3.6% (Bai et al. 2019) and 10% after hip fracture surgery (de Jong et al. 2017). Patients with SSI have longer hospitals stays, more readmissions and higher mortality. SSI following orthopaedic surgery can lead to poorer outcomes from surgery, functional loss, implant failure and amputation (Copanitsanou 2020).

Prevention of SSI is an interdisciplinary team priority. Prevention should be focused on adherence to infection prevention guidelines (see previous section) and up-to-date evidence-based guidance as outlined in Box 9.3. Prophylactic prevention of infection using antibiotics in the orthopaedic and trauma setting is standard practice and has been shown to reduce rates of infection where risk is high, such as in traumatic wounds and surgery which involves implantation (Gillespie and Walenkamp 2010). However, resistance is an increasing problem across all healthcare settings and the careful and prudent use of antibiotic therapy is increasingly important. This reinforces the need for measures that minimise all infections (Bryson et al. 2016).

Urinary Tract Infection

The urinary tract is the most common source of healthcare-associated infection. Because of stasis in the urinary system during anaesthesia, surgery and post-operative recovery, the risk is high in all surgical patients and in those with restricted mobility. The risk of infection is significantly increased by bladder catheterisation, so this should be avoided in orthopaedic patients because of the link between bacteriuria and implant site infection (Meddings et al. 2019).

Guidance for the prevention of healthcare-associated UTI is provided in Box 9.4. Engaging all clinical staff in measures to prevent UTI, particularly urinary catheter-associated UTI, is essential (Thakker et al. 2018).

Respiratory Tract Infection (Pneumonia)

Pneumonia (lower respiratory tract infection) is a potentially fatal hospital-acquired infection. It is a significant cause of death for orthopaedic patients, particularly following fragility hip fracture. Mucosal surfaces in the respiratory tract contain large numbers of resident flora, which combat pathogenic attack. Hypostatic pneumonia is a risk when mechanisms such as the cough reflex are suppressed due to anaesthesia and sedation, and patient mobility problems mean that respiratory ventilation is decreased, particularly in older and frail patients and those with concurrent medical conditions.

Box 9.4 Guidance for the Prevention of Healthcare-associated UTI

Avoid unnecessary placement of indwelling urinary catheters
Removal of urinary catheters as early as possible, preferably within 24 hours
Use of closed drainage systems and aseptic management of catheters and systems
Avoidance of dehydration
Early mobilisation to prevent urinary stasis and return to normal toileting habits as soon as possible
Evidence-based continence management without the use of indwelling urinary catheters where required
High standards of perineal/penile hygiene
Early identification and treatment of UTI and good hydration

Measures for prevention of respiratory tract infection in the orthopaedic patient include:

- avoidance of elective surgery in patients with pre-existing respiratory infections (usually viral) through preoperative screening
- post-operative pain relief to facilitate coughing and deep breathing
- early post-operative mobilisation
- since chest infection is often hospital-acquired, universal infection control precautions are an essential aspect of prevention.

Observation of the at-risk patient for symptoms of pneumonia is essential in enabling early management. Symptoms may be insidious or develop quite suddenly and include:

- shortness of breath/rapid and/or shallow breathing
- a cough which may or may not be productive initially
- sputum which may be yellow, green, brown or blood stained; a specimen should be obtained for culture
- chest pain
- pyrexia
- tachycardia
- acute confusion
- general malaise and fatigue
- loss of appetite.

A diagnosis of pneumonia is made based on the above symptoms along with chest auscultation (abnormal lung sounds can be heard) and chest X-ray (demonstrating lung consolidation). There will also be a raised white cell count. A positive sputum culture will help to identify the causative organism and direct treatment.

Management of pneumonia should be commenced immediately due to the potentially life-threatening nature of the infection. This should include the following considerations:

- Antibiotic therapy according to the nature and sensitivity of the pathogen causing the pneumonia. The timeliness and appropriateness of this is central to the recovery of the patient along with other supporting measures but may be complex if a resistant strain of bacteria is the causative organism.
- Constant monitoring of the vital signs of the patient with a view to detecting and acting on any further deterioration.
- Maintenance of hydration using intravenous infusion of fluids if necessary.
- Optimum nutrition using nutritional supplementation, nasogastric or enteral feeding as necessary.
- The patient should be cared for sitting up or in the semi-recumbent position, providing it is aligned with their orthopaedic condition.
- Deep-breathing exercises and chest physiotherapy.

Sepsis and Septic Shock

Sepsis is a leading cause of avoidable death. It is a clinical syndrome in which immune and coagulation responses are triggered by an infection. Sepsis can lead to septic shock, a life-threatening condition that occurs when sepsis leads to low blood pressure and low blood flow that can result in organ failure (NICE 2016) (as described in the section considering shock). Sepsis can be caused by any source of infection, including cellulitis and wound infections, UTIs, respiratory infections, septicaemia, septic arthritis and osteomyelitis, meaning that the orthopaedic patients is at significant risk. The mortality rate is estimated to be as much as 56% (Bauer et al. 2020).

Early recognition may be difficult and the practitioner's role is central in recognising changes in patient condition and seeking medical attention. The person may present with similar signs to hypovolemic shock (see below) but without a potential source of bleeding. There may also be hypotension, altered coagulation, inflammation, impaired circulation, anaerobic metabolism and changes in mental status. NICE (2016) provide a series of tools/algorithms for the identification of those who could be presenting with sepsis (see Box 9.5).

Shock

Shock is a complex life-threatening physiological syndrome resulting from a significant reduction in systemic tissue perfusion, and subsequent hypotension and reduced

Box 9.5 Could this Be Sepsis? Sepsis: Risk Stratification Tools (NICE 2016)

Could this be sepsis?

For a person of any age with a possible infection:

- Think – could this be sepsis? Does the person have signs or symptoms that indicate infection, even if they do not have a high temperature?
- Be aware that people with sepsis may have non-specific, non-localised presentations (e.g. feeling very unwell).
- Pay particular attention to concerns expressed by the person and their family or carer.
- Take particular care in the assessment of people who might have sepsis if they, or their parents or carers, are unable to give a good history (e.g. people with English as a second language, people with communication problems).

Assessment

- Assess people with suspected infection to identify:
  - Possible source of infection
  - Risk factors for sepsis (see below)
  - Indicators of clinical concern such as new onset abnormalities of behaviour, circulation or respiration.

Risk factors for sepsis

- The very young (under 1 year) and older people (over 75 years) or very frail people.
- Recent trauma or surgery or invasive procedure (within the last 6 weeks).
- Impaired immunity due to illness (e.g. diabetes) or drugs (e.g. people receiving long-term steroids, chemotherapy or immunosuppressants).
- Indwelling lines, catheters, intravenous drug misusers, any breach of skin integrity (e.g. any cuts, burns, blisters or skin infections).

Management of sepsis and associated septic shock includes (NICE 2016):

- urgent medical review
- structured face-to-face assessment and observation (essential in both recognising sepsis and in monitoring the patient with sepsis) at least every 30 minutes, and using an early warning score and recording system
- early administration of broad-spectrum antibiotics until the source and microbiology of infection is identified
- identification and medical treatment of the source of infection
- intravenous fluid resuscitation according to recent guidance
- referral to critical care services for ongoing treatment and monitoring.

The UK Sepsis Trust (www.sepsistrust.org) provides resources to help clinicians learn about recognising and managing sepsis, including education resources and guidelines.

oxygen (O_2) delivery to the tissues. Early recognition and management are vital in increasing the odds of survival. Poor perfusion of cells leads to an imbalance between O_2 delivery and O_2 consumption. Oxygen deprivation (hypoxia) causes cellular hypoxia and disruption of critical cellular processes, including failure to meet the demands of cell metabolism and removal of waste. Without intervention, the result is sequential cell death, multisystem organ failure and death (Archbold and Naish 2015). There are several types of shock with different causes and presentations. The focus here will be on hypovolaemic shock (having considered septic shock above) since this is most relevant to the orthopaedic/trauma practitioner.

There are four stages of shock, which are initially reversible but then rapidly become irreversible:

Stage 1 Initial stage of shock: This is reversible, but easily missed as there are very few signs. Cardiac output is reduced and metabolism switches from aerobic to anaerobic, potentially leading to lactic acidosis (due to the inadequate clearance of lactic acid from the blood).

Stage 2 Compensatory stage of shock: The sympathetic nervous system produces catecholamine in an attempt to regain homeostasis and improve the perfusion of tissues by dilating the bronchi and constricting peripheral blood vessels. Water conservation is initiated by the release of aldosterone by the adrenal/renal system. Changes to vital signs will occur.

Stage 3 Progressive stage of shock: The compensatory mechanisms that sustain the perfusion of tissues are lost, resulting in metabolic and repository acidosis along with electrolyte imbalance. A visible deterioration in the patient's condition is seen.

Stage 4 Refractory stage of shock: There is irreversible cellular and organ damage. The condition becomes unresponsive to treatment and death is imminent (Tait et al. 2015).

Hypovolaemic Shock

Hypovolaemic shock (HS) is a life-threatening condition in which a reduction in blood volume leads to insufficient oxygen and nutrient supply to the cells. Uncontrolled bleeding is the leading cause of HS (Mutschler et al. 2014) and the most common cause in the orthopaedic patient is blood loss following trauma or surgery.

Trauma to both soft tissue and bone can lead to significant bleeding. If injuries are superficial, bleeding may be obvious, alerting the practitioner to potential hypovolaemia early. However, trauma to major skeletal structures and associated soft-tissue damage can lead to bleeding that is not immediately obvious. Fractures of the femoral shaft, for example, are usually the result of high-energy trauma and bleeding at the fracture site can be a significant cause of hypovolaemia without obvious external signs. Pelvic fractures have a particularly high risk of haemorrhage because the mechanism of injury is usually crushing of the pelvis with subsequent potential damage to the structures contained in the pelvic cavity, including major blood vessels.

In the patient undergoing elective orthopaedic or emergency trauma surgery, there is always a risk of postoperative haemorrhage, not only from the surgical site itself, but also 'hidden' losses from the surrounding tissues. Early warning score systems and modified versions are likely to be helpful in enabling collection of observation data and recognising the deteriorating patient so that management of shock can be initiated (Qin et al. 2017).

Early detection of uncontrolled bleeding and shock involves close observation of the person who has recently sustained trauma or undergone orthopaedic surgery. Understanding the nature and progression of the signs and symptoms of HS assists the practitioner in recognising the need for intervention. The ATLS® (American College of Surgeons 2008; Mutschler et al. 2014) (see Table 9.1) classification of HS provides an overview of the observation/vital sign parameters that are seen as shock progresses and represent the physiological mechanisms which try to maintain homeostasis. These parameters reflect decreasing circulatory volume and blood flow, hypoxia, altered cognition due to decreased cerebral perfusion, sympathetic nervous system outflow and acidosis. Changes in skin appearance such as mottling, coolness or clamminess may also be evident due to reduced peripheral perfusion. Progression of symptoms indicates increasing blood and fluid loss, worsening the potential outcome, so identifying early subtle signs is essential.

Management of hypovolaemia in both the trauma and orthopaedic surgery patient follows similar principles:

- senior medical assessment for sources of bleeding and prompt action to control bleeding, including surgery/further surgery
- close monitoring and recording of patient vital signs and condition using an evidence-based early warning score tool
- supplementary oxygen therapy as prescribed
- assessment of blood levels of arterial blood gases, electrolytes, haemoglobin, lactate and haematocrit to identify deficits
- optimal fluid resuscitation according to most recent evidence-based guidelines, including use of blood and plasma replacement products, usually a combination of plasma, platelets and red blood cells (Holcomb et al. 2015).

Venous Thromboembolism

VTE is a condition in which a blood clot (thrombus) forms in a vein. Blood flow through the affected vein can be restricted by the clot, leading to swelling and pain.

Table 9.1 ATLS® classification of hypovolaemic shock (American College of Surgeons 2008; Mutschler et al. 2014).

	Class I	Class II	Class III	Class IV
Blood loss in % of total volume	<15	15–30	30–40	>40
Pulse rate	Normal	Normal	Decreased	Greatly decreased
Blood pressure	Normal	Normal	Decreased	Greatly decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14–20	20–30	30–40	>35
Mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Urine output (ml/h)	>30	20–30	5–15	Minimal

Venous thrombosis usually occurs in the deep veins of the leg or pelvis, known as a deep vein thrombosis (DVT). An embolism can occur if all or part of the clot breaks off from the site where it has formed and then travels through the venous system. If the clot lodges in the lung, a serious and sometimes fatal condition, pulmonary embolism (PE), occurs.

Venous thrombosis can occur in any part of the venous system, but DVT and PE are the most common manifestations of this. The term VTE embraces both the acute conditions of DVT and PE as well as the chronic conditions that can occur after acute VTE, such as post-thrombotic syndrome and pulmonary hypertension, problems associated with significant ill-health and disability.

Risk Factors for VTE

The three main underlying factors that lead to VTE were first described by Rudolph Virchow in 1853 and are commonly referred to as Virchow's triad:

venous stasis
vein injury
changes in blood chemistry.

It is accepted that it is usually a combination of these factors that causes a thrombus to form, rather than one factor in isolation. The inherent impaired physical mobility and activity intolerance that affects orthopaedic patients gives rise to circulatory stasis. If they also have existing conditions of, or have experienced trauma to, the circulatory system as well as alterations in blood coagulation, then they are in danger of developing VTE (Davis 2004a).

Orthopaedic patients are at significant risk of developing VTE due to the nature of their condition and its management. The most significant general risk factors for VTE are listed in Box 9.6. Many of these apply specifically to orthopaedic patients.

Box 9.6 General Risk Factors for VTE

- Immobility
- Major surgery, including orthopaedic surgery
- Major fractures of the pelvis, lower limb and long bones
- Multiple trauma
- Cancer
- Cancer treatment, including chemotherapy and surgery
- Age over 60 years
- Known thrombophilias
- Dehydration
- Obesity
- Close family history of VTE
- Varicose veins with associated phlebitis
- Oestrogen contraceptive therapy
- Hormone replacement therapy
- One or more signify ant comorbidities (e.g. heart disease, metabolic, endocrine or respiratory conditions, acute infections, inflammatory conditions)

Risk Assessment

Most hospitalised orthopaedic patients should be considered at risk of developing a VTE so that they can receive appropriate prophylactic interventions. Those in the community or following discharge are also at risk. Assessment is based on some, but not all, of the predisposing factors referred to previously and listed in Box 9.3. NICE guidelines (2018) recommend that all hospitalised patients should receive a risk assessment for VTE at pre-admission, on admission and at every point that their condition changes, especially for trauma and surgical patients. Various risk assessment tools are available to support practitioners in undertaking and recording risk assessment, and local guidance should be followed in this respect. NICE (2010) have provided an example of a risk assessment proforma that begins with an assessment of mobility (www.nice.org.uk/guidance/ng89/resources/department-of-health-vte-risk-assessment-tool-pdf-4787149213).

Methods of Prevention

VTE is a leading cause of adverse events, disability and death directly due to hospital admission (International Society on Thrombosis Haemostasis 2018) although the true incidence of DVT, PE and related deaths is difficult to calculate. Importantly, however, VTE is a preventable condition. There have been numerous research studies that provide evidence of the effectiveness of methods to prevent VTE; this evidence-base is constantly progressing. Regularly updated guidance exists that identifies those preventative methods most likely to be successful. Some of the latest guidelines are listed in Box 9.7.

Prevention interventions for VTE fall into three main categories: (i) pharmacological, (ii) mechanical and (iii) care.

Pharmacological Interventions

Pharmacological measures for VTE prevention focus on the inhibition of thrombus formation by acting on clotting mechanisms. These measures are recommended for all orthopaedic patients assessed as at risk of VTE. NICE (2018) currently recommends prophylaxis with low molecular weight heparin (LMWH) or fondaparinux sodium for

Box 9.7 Evidence Digest

Examples of recent evidence-based guidelines for the prevention of VTE in hospitalised patients

Australian Commission on safety and quality and healthcare (2020) Venous Thromboembolism Prevention Clinical Care Standard. www.safetyandquality.gov.au/publications-and-resources/resource-library/venous-thromboembolism-prevention-clinical-care-standard.

Afshari, A., Ageno, W., Ahmed, A., Duranteau, J., Faraoni, D., Kozek-Langenecker, S., Llau, J., Nizard, J., Solca, M., Stensballe, J., Thienpont, E., Tsiridis, E., Venclauskas, L., Samama, C.M, for the ESA VTE Guidelines Task Force European Guidelines on perioperative venous thromboembolism prophylaxis, European Journal of Anaesthesiology. February 2018, Volume 35, Issue 2, p. 77–83. Doi: 10.1097/EJA.0000000000000729.

NICE (2018).www.nice.org.uk/guidance/ng89/resources/venous-thromboembolism-in-over-16s-reducing-the-risk-of-hospitalacquired-deep-vein-thrombosis-or-pulmonary-embolism-pdf-1837703092165.

people with lower limb immobilisation or having lower limb or spinal surgery and those with pelvic, lower limb or spinal fractures or spinal injury. A medical decision needs to be made about whether the risk of VTE outweighs the additional risk of bleeding resulting from the administration of anticoagulation therapy.

Mechanical Interventions

There are three main evidence-based mechanical methods for VTE prophylaxis. The main advantage of such interventions is that they do not increase the risk of bleeding as pharmacological methods do.

1) Graduated compression stockings/hosiery (also known as anti-embolic stockings, AES) are designed and manufactured to exert pressure on the limb with the aim of improving the efficiency of the circulation. The stockings exert the greatest degree of compression at the ankle and the level of compression gradually decreases up the garment. Sachdeva et al. (2018) found high-quality evidence that graduated compression stockings are effective in reducing the risk of DVT in hospitalised patients who have undergone general and orthopaedic surgery. Wellington et al. (2015) provide guidance for the assessment, fitting, application and ongoing safe care of the patient with compression/AES stockings.
2) Intermittent pneumatic compression (IPC) devices involve inflatable garments (boots or stockings, thigh or knee-length) which are wrapped around both legs. A pneumatic air pump inflates and deflates the garments in cycles, which intermittently apply and release pressure on the limb, helping to improve the venous circulation in the legs with the aim of preventing thrombosis formation. This inflation–deflation cycle 'simulates the thigh, calf and foot's normal ambulatory pump action, thus increasing both the volume and rate of blood flow, eliminating venous stasis and replicating the effects of the natural muscle pump. Intermittent pneumatic compression devices can be thigh- or knee-length sleeves that are wrapped around the leg, or a garment that can be wrapped around or worn on the foot that is designed to mimic the actions of walking' (NICE 2018, p. 33). This has been shown to decrease the incidence of VTE both as a single measure and combined with other measures (Pavon et al. 2016). NICE (2018) recommend the use of IPC in patients undergoing major orthopaedic surgery that places them at risk of VTE when other methods of VTE prophylaxis are not feasible or contraindicated, particularly in patients with fragility fractures and those undergoing total knee arthroplasty. The device should continue to be used until the patient is fully mobile, so they may need to continue at home. However, compliance with this method of VTE prophylaxis has been found to be low due to issues such as: patient discomfort, patient knowledge, healthcare professional knowledge and behaviours, use of guidelines, mobilisation issues, equipment availability and, prescribing issues (Greenall and Davis 2020). Practitioners can therefore help to improve compliance by ensuring that patients are well educated about the need for and use of the devices.

Care Interventions

Two important factors in prevention include the fact that modern orthopaedic care means that patients now have less invasive surgery and great emphasis is placed on early mobilisation and early discharge from hospital. Some clinicians believe that DVT and PE are less prevalent than they used to be in surgical patients, but this may be hidden due to enhanced recovery pathways, and many DVTs occur after discharge.

The two most important nursing interventions for VTE prevention are:

1) Early mobilisation and leg exercises
2) Hydration (but through the oral route, not intravenous).

Current guidelines recommend the use of these interventions in combination. They also increasingly highlight the patient's view such as the difficulty and discomfort associated with graduated compression stockings (NICE 2010). All prophylactic interventions carry risk as

well as benefit and this must be balanced in any care decisions for individual patients. The risk of bleeding is an example with pharmacological interventions for VTE.

The linking of evidence through an evidence-based practice approach to orthopaedic nursing practice is well illustrated by the issue of VTE. Research, such as in areas of early mobilisation and hydration, is often lacking or so poor quality that it cannot be relied on to direct practice decisions. Even when evidence is strong, it has to be applied consistently and with knowledge and understanding. Davis (2004b) discusses ways in which the problems of translation and utilisation can be overcome with respect to VTE.

NICE (2020) have developed a three-page visual summary of the recommendations on diagnosis and anticoagulation treatments for venous thromboembolism (www.nice.org.uk/guidance/ng158/resources/visual-summary-pdf-870909145).

Fat Embolism Syndrome

FES is a life-threatening condition that can lead to respiratory failure in orthopaedic patients. It is a medical emergency that requires immediate recognition and action.

The term 'fat embolism' (FE) refers to the presence of fat globules in the peripheral circulation and lung parenchyma most commonly following fracture of long bones, the pelvis or other major trauma, although it can also occur following major implant surgery that involves reaming of bone. The condition is usually asymptomatic and may not become evident unless it develops further. Fat embolism syndrome is a severe and life-threatening manifestation of FE that usually occurs within 24–72 hours of injury or surgery to a long bone. The patient presents with a collection of clinical symptoms, including dyspnoea, hypoxaemia, mental confusion (due to hypoxia) and petechial rash. Some patients will develop signs and symptoms of multiorgan dysfunction, particularly involving the lungs, brain and skin.

The clinical signs/symptoms and course of FES are a result of a series of pathological processes (Jain et al. 2008; Kosova et al. 2015; Rothberg and Makarewich 2019). Fat emboli are released from the marrow of long bones following fracture or reaming of bone.

The trauma responses at the injury (or surgery) site release chemical mediators, which bring about various coagulation events, including coagulation of circulating lipids, which result in the development of fat emboli large enough to occlude pulmonary vessels. Emboli obstruct the capillaries of major organs (most commonly the lungs, but also the brain and kidneys). The ensuing inflammatory response results in acute respiratory distress syndrome (ARDS) and signs of dysfunction in other organs.

Box 9.8 Signs of Fat Embolism Syndrome (Gurd and Wilson 1974.

Major signs:
- petechial rash
- respiratory insufficiency
- cerebral involvement.

Minor signs:
- tachycardia >120 bpm
- fever >39.4 °C
- retinal signs – fat or petechiae
- jaundice
- renal signs – anuria/oliguria.

There is evidence that the incidence of FES has reduced over the last few decades due to improvements in fracture management (Lempert et al. 2021), suggesting that prevention should be focused on fracture care.

The clinical team caring for patients in the first few days following injury or surgery to long bones needs a high level of suspicion in recognising the early signs of FES so that respiratory support can be instigated early. As part of the general observation of the patient following trauma/surgery, nurses should observe for any signs listed in Gurd and Wilson's (1974) diagnostic criteria (shown in Box 9.8), which are still considered relevant today (Kosova et al. 2015).

Any of these clinical signs should be reported to a senior clinician as an emergency. Because of the risk of respiratory and multiple organ failure, patients with clinical signs of FES require emergency management, respiratory support and fluid resuscitation in a critical/intensive care facility or high-dependency unit. Supplemental oxygenation should be prescribed and administered early on, and mechanical ventilation may be necessary along with constant monitoring of vital signs. If early and effective emergency and intensive care is provided, outcomes are good (Kosova et al. 2015).

Acute Compartment Syndrome

ACS is a clinical condition that occurs when there is an increase in pressure and/or a decrease in the size of a muscle compartment resulting in reduced capillary blood flow and leading to cell death. It is an orthopaedic emergency because if the pressure is not relieved within hours, irreversible damage to the tissues and nerves may result in contractures, paralysis, loss of sensation and amputation (Ali et al. 2014). This can lead to muscle ischaemia, infarction, and necrosis and rhabdomyolysis,

a life-threatening condition in which toxins released from ischaemic/necrotic muscle into the circulation can lead to organ failure.

The most common site of ACS is the anterior compartment of the lower leg. Any compartment, however, can be affected, although most cases present following a tibial fracture. The condition can be difficult to diagnose in all patients, including children, with delays in diagnosis leading to disastrous outcomes (Bae et al. 2001). Although ACS is an uncommon condition, it is dangerous and life threatening, so the clinician needs to be able to recognise it immediately so that it can be treated.

The orthopaedic patient (including children) can present with ACS following:

- *trauma*: fracture, haematoma, vascular damage, electrical injuries, particularly to the lower leg and forearm
- *surgery* to the lower or upper limb
- *external compression* due to casts or restricting bandages.

In acute limb compartment syndrome (ALCS), limb compression leads to local pressure with local tamponade (blockage). This results in capillary necrosis and oedema with increased compartment pressure and muscle ischaemia. This then leads to compartment tamponade, nerve injury and muscle infarction.

The British Orthopaedic Association (BOA)/British Association of Plastic Reconstructive and Aesthetic Surgeons provide standards for the diagnosis and management of compartment syndrome of the limbs (BOA/BAPRAS 2014). They state that the key clinical indicators of ACS in the conscious patient are:

- pain out of proportion to the associated injury
- pain on passive movement of the muscles of the involved compartments.

It is pain that is the earliest sign of ACS and it is this on which the practitioner should focus. While neurological symptoms such as tingling (paraesthesia) and numbness, and circulatory symptoms such as changes in colour and warmth should be recorded, these are late signs and do not contribute to early diagnosis of the condition (BOA/BAPRAS 2014). These late signs demonstrate that neurovascular and muscle damage have already occurred and the priority for the practitioner is to recognise the condition from the early signs so that permanent damage can be prevented. Monitoring of compartment pressure is considered helpful in the unconscious patient but is not available in many units (Ali et al. 2014).

Bandages and dressings should be removed if ACS is suspected, and the limb should not be elevated even though this is a common principle of care for the orthopaedic/trauma patient with swelling and pain.

Neurovascular Assessment

All patients who have a musculoskeletal injury, undergone orthopaedic surgery or have cast immobilisation of a limb are at risk of developing neurovascular compromise, which can lead to compartment syndrome. Such neurovascular compromise can include ALCS as well as other damage to neurovascular structures in the limbs due to injury, surgery and other procedures, and swelling (Box 9.9).

Peripheral neurovascular assessment involves the systematic assessment of the neurological and vascular integrity of a limb, with the aim of recognising any neurovascular deficit promptly (Judge 2007). Tissue damage deteriorates with time, so prompt identification and intervention are necessary. The optimum frequency of assessment is unclear but should reflect the acute nature of the patients' recent injury/procedure and the potential for compromise to occur with the rationale recorded. The main parameters of neurovascular assessment are listed, with their rationale, in Box 9.10. These parameters should be recorded in a neurovascular assessment chart – sometimes included in early warning score charts – so that comparisons can be made and deterioration identified quickly. Shields and Clarke (2011) recommended the use of a dedicated chart to record pain intensity and type, alongside warmth, sensation, colour, capillary refill time and the movement of the affected

Box 9.9 Evidence Digest

Royal College of Nursing guidelines for peripheral neurovascular observations for acute limb compartment syndrome

Peripheral neurovascular observations for acute limb compartment syndrome. RCN consensus guidance

Royal College of Nursing (2014) Peripheral neurovascular observations for acute limb compartment syndrome RCN consensus guidance. Royal College of Nursing. London. Available at: www.rcn.org.uk/professional-development/publications/pub-004685.

The Royal College of Nursing's Society of Orthopaedic and Trauma Nursing published these guidelines following a 3-year project to develop consensus regarding best practice in the early identification of ALCS, reducing its risk, the role of clinical observation and compartment pressure measurement in early detection.

Following a literature review (Ali et al. 2014) which found that the evidence was insufficient to guide best practice, the guideline development group held a consensus conference to develop guidance for practice by seeking the consensus of expert clinicians.

Box 9.10 Neurovascular Assessment Parameters for Patients with Limb Injuries, Surgery or Casts

Parameter	Rationale
Pain (priority sign)	Both vascular (depleted blood supply leading to ischaemia) and neurological (damaged nerve conduction) damage result in a pain response. This is always (except in the unconscious patient) the earliest sign of damage. Practitioners should take any reports of pain in the area around and/or peripheral to the injury seriously and report this to a medical practitioner immediately. Pain is unresolved by analgesia.
Pulses	Pulses peripheral to the site of injury/surgery provide an assessment of the circulation (vascular) at the site. Absence of pulses, however, is a late sign of vascular injury/occlusion.
Capillary refill	Capillary refill time provides an assessment of the perfusion to an area peripheral to an injury/surgical site. It is conducted by applying pressure to the finger-tip/nail or toe-tip/nail for 5 seconds with enough pressure to cause blanching as the blood is pushed out of the tissues. The pressure is then released and the time taken for the tissue to return to the same colour as that around it. Normal refill time is considered to be 2 seconds or less.
Pallor/discoloration/temperature	Paleness, discolouration (blue/mottled) and/or coolness of the skin are signs of vascular compromise indicating that peripheral tissues have no, or insufficient, blood supply. This is also a very late sign of vascular injury/occlusion.
Paraesthesia	Paraesthesia is a patient-reported perception of abnormal sensation, which can include 'pins and needles', tingling, pricking or crawling. This is a potential sign of peripheral nerve damage (neuropathy).
Loss of sensation and/or paralysis	Both loss of sensation and paralysis of a limb or section of a limb indicate lack of nerve supply to bring about movement or sensation.

and unaffected limb as an important method of collecting and comparing data from baseline onwards.

Shields and Clarke (2011) also recommend the use of a dedicated chart to record pain intensity and type alongside warmth, sensation, colour, capillary refill time and the movement of the affected and unaffected limbs as an important method of collecting and comparing data from baseline onwards.

A central aspect of care of the patient with suspected ALCS, or other form of neurovascular compromise, is to treat any suspicion as a medical emergency requiring immediate medical attention. It is essential that the practitioner inform a senior member of medical staff immediately so that intervention can be instigated.

Fasciotomy is the management option of choice for ALCS; the fascia is divided along the length of the compartment to release pressure. The pressure at which fasciotomy is performed is based on the clinical picture/rising pressure. Following the procedure, the wound is usually left open for approximately 5 days until the soft tissues have recovered and swelling has begun to subside. Muscle and skin grafting may be required.

Urinary Retention

A significant reason for urinary catheterisation in the orthopaedic patient is post-operative urinary retention, which is frequently reported following orthopaedic surgery. It has been reported to occur in up to 84% of patients following total hip arthroplasty (Lawrie et al. 2017). It is defined as an inability to pass urine even though the patient has a full bladder. This can result in pain and distress for the patient and can lead to bladder distention. Retention can also lead to UTI, adverse autonomic responses such as vomiting, hypotension and cardiac dysrhythmia, and permanent damage to the bladder with resultant future urinary problems (Baldini et al. 2009). Early recognition of the problem is therefore essential and should be included as an aim for all postoperative care. There is no evidence that routine catheterisation intraoperatively as a method of prevention is beneficial and the risk of haematogenous spread of infection to the surgical site is too great for this to be advised (Crain et al. 2021).

The main symptom of urinary retention is pain and discomfort in the lower part of the abdomen in a patient who is unable to pass urine despite good fluid balance. This can,

however, be masked post-operatively by the effects of general and regional anaesthesia, nerve blockade and analgesia. Bladder catheterisation can also be used as a method of assessing bladder volume and diagnosing retention through measurement of the residual volume of urine. This, however, carries with it the risk of infection associated with per urethral catheterisation. The literature suggests that the use of nurse-led ultrasound to assess bladder volume is a relatively simple and appropriate way for the practitioner to monitor bladder volume and identify retention whilst avoiding unnecessary catheterisation (Crain et al. 2021).

Once retention has been identified, the treatment usually involves bladder catheterisation. It is recommended that this is done as an in-out (intermittent) catheterisation rather than with an indwelling catheter and that antibiotic prophylaxis is essential (Baldini et al. 2009). It may be necessary for the bladder volume to be monitored for up to 48 hours post-operatively, but most problems tend to resolve once the patient begins to mobilise and is able to visit the toilet to void.

Constipation

Constipation is a very common and significant complication that can be either acute or chronic. In the orthopaedic and trauma patient, it is often caused by a decrease in bowel action due to a combination of factors that lead to hard, dry stools that are difficult and/or painful to pass. The problem is defined by the patient and may include what they feel to be 'unsatisfactory' or incomplete defecation. Although it is thought there is a link between the incidence of constipation and increasing age, this is most likely because of the greater incidence of other precipitating factors in older people. Some other common causes of constipation in the orthopaedic and trauma patient include:

- *dehydration*, leading to desiccated stools
- *reduced mobility*, resulting in weakness in the accessory muscles, which help bowel evacuation
- *reduced or changed dietary intake*, resulting in a diet that is depleted or lacking in fibre
- *pharmacological agents*: one of the main side effects of many drugs is constipation and in the orthopaedic and trauma patient both opioid and non-opioid analgesic agents are implicated (known as opioid-induced constipation; Sonneborn and Bui 2019), but other drugs such as antidepressants can also contribute because of the slowing effect on peristaltic action.

Hospitalised patients and those reliant on others for toileting needs often resist the urge to pass bowel movements because of embarrassment or pain associated with the required activity, particularly if they require a bedpan (Cohen 2009). Because of embarrassment, patients may not be willing to inform a health professional of the problem. These are issues that it is essential the practitioner is sensitive to.

Impaction, where faeces become trapped in the lower part of the large bowel, is very distressing for the patient. The most serious consequence of untreated constipation and impaction is bowel obstruction by a volvulus, which becomes a surgical emergency and can be fatal. It is essential that this be avoided through careful assessment and prevention.

The most important aspect of the prevention and management of constipation is the early and continuous assessment of bowel activity. Because nearly all orthopaedic and trauma patients have at least one risk factor for constipation and because of the reluctance of patients to discuss difficulties with the nurse, it is essential that a proactive daily assessment of bowel activity is made. 'Normal' bowel habits vary from one person to another and the practitioner must take this into account when assessing bowel function, considering the patients' normal frequency of opening their bowels. If constipation lasts more than a few weeks and/or is associated with other symptoms such as abdominal mass/pain or blood in the stools, then a medical referral is made to rule out other more serious causes.

Proactive prevention of constipation is an important aspect of the care of the orthopaedic and trauma patient. It is important that this is incorporated into the standard care of all patients at risk of constipation and is not left until constipation has begun to occur. Prevention involves management of the causes and risk factors for constipation. This generally includes helping to provide conditions for toileting routines that are as near normal for the patient as possible with due consideration of privacy and position:

- *monitor*: daily assessment and recording of bowel activity
- *diet*: ensuring the diet contains or is supplemented with foodstuffs high in dietary fibre
- *hydration*: ensuring that the patient takes plenty of oral fluids
- *exercise*: within the limits and abilities of the patient; when unable to walk there may be benefit from abdominal exercise such as pelvic tilt (Joanna Briggs Institute 2008).

It is important that any tendency to constipation be managed as soon as possible after symptoms occur. The management of constipation generally involves the use of pharmacological laxatives. There are several different types of laxative, which work in different ways:

Box 9.11 Evidence Digest: Nurse-led Management and Prevention of Opioid-induced Constipation

Sonneborn, O. and Bui, T. (2019) Opioid induced constipation management in orthopaedic and trauma patients: treatment and the potential of nurse-initiated management, *International Journal of Orthopaedic and Trauma Nursing,* 34, pp. 16–20. https://doi.org/10.1016/j.ijotn.2019.03.002.

Sonneborn and Bui (2019) point out that opioid analgesics, used to treat moderate to severe pain, are associated with common side effects, such as constipation. Orthopaedic and trauma patients are at high risk of developing opioid-induced constipation (OIC) because their mobility is reduced and there is a need for opioids such as codeine to manage pain.

The authors' aim was to 'examine the evidence base to guide clinicians on the most effective or tolerated laxative regimen for the management of OIC and nurse-initiated management of OIC'.

They conducted a review of the literature, searching several databases to identify studies regarding OIC, laxatives and nurse-initiated management.

The review findings concluded the following:

1) Laxatives do not address the underlying cause of OIC and there is currently insufficient evidence to guide clinicians on the most effective or tolerated laxative regimen for the management of OIC.
2) The use of peripheral acting mu-opioid receptor antagonists (PAMORAs) could be considered in those for whom regular use of a combination of laxatives has not been successful, and nurses should take a broader role in the assessment of symptoms and response to treatment.

The authors conclude that there is an important balance between adequate analgesia and minimising OIC symptoms, making this an ongoing challenge for clinicians and an area of patient care where nurses could be leading management.

- *bulking agents* contain fibrous material that absorbs water in the bowel and makes stools softer
- *peristaltic stimulants* are useful where peristaltic action is reduced
- *osmotic laxatives* encourage the absorption of fluid into the stools
- *stool softeners* lubricate and moisten stools.

When constipation is opioid-induced it is advisable to begin treatment with a combination of an osmotic laxative and a peristaltic stimulant (see Box 9.11). If this treatment fails to resolve constipation or faecal impact is suspected, treatment with suppositories or enemas along with peristaltic stimulants may be required.

Further Reading

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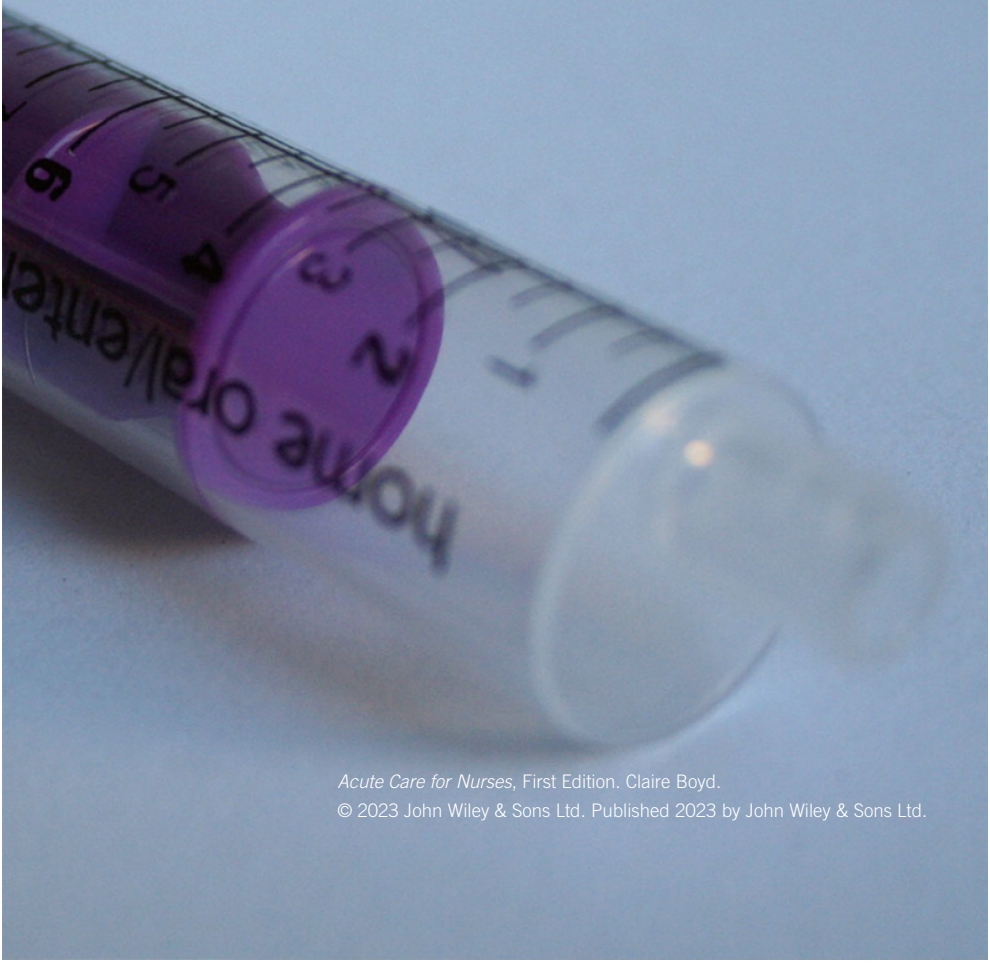
Chapter 2

RECOGNISING SEPSIS



Acute Care for Nurses, First Edition. Claire Boyd.

LEARNING OUTCOMES

By the end of this chapter you will have an understanding of sepsis, including the incidence, physiology, how to recognise it, risk factors, and treatment. You will also have knowledge of the sepsis six and an understanding of blood results used in conjunction with diagnosing sepsis.

To begin this chapter, look at the following medical terms and think for a moment about what they actually mean:

- Bacteraemia
- Sepsis syndrome
- Severe sepsis
- Septicaemia
- Systemic inflammatory response syndrome

Did you know that all these terms mean the same thing and have now been deemed obsolete and replaced by one term – **sepsis**.

Within the healthcare environment (World Health Assembly, National Health Service [NHS], National Institute for Health and Care Excellence [NICE]) there has been a big push to improve the identification, diagnosis, and management of sepsis to improve patient safety outcomes.

Question 2.1 So, what exactly is sepsis? Isn't it just blood poisoning?

Well done – you've just found another term for sepsis! Sepsis is a very serious reaction to an infection. Normally the body's immune system kicks in to fight off the infection, but in the

case of sepsis the immune system response becomes overactive and starts to cause damage to the body itself. NICE has defined sepsis as 'a life-threatening condition that arises when the body's response to an infection injures its own tissues and organs' (NICE, 2016, https://www.nice.org.uk/guidance/ng51).

We can see where sepsis fits into the infection spectrum in Figure 2.1.

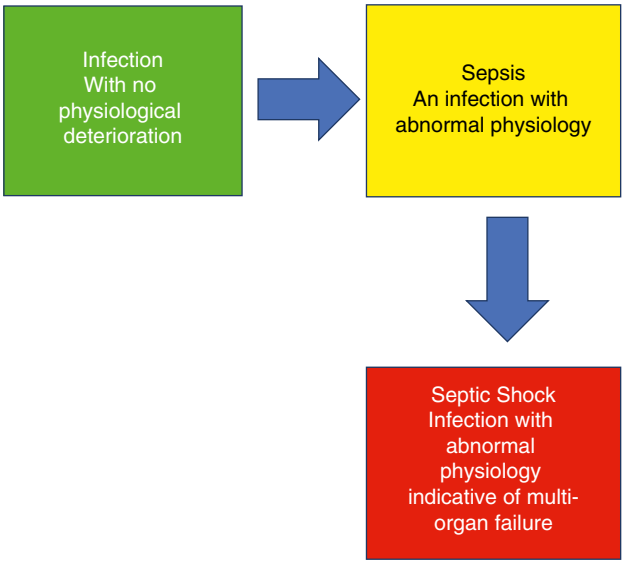


Figure 2.1 Infection criteria.

We can see from Figure 2.1 that sepsis is the worst kind of infection, which can lead to septic shock. To understand why we are so concerned about sepsis, first we need to see some facts and figures.

SEPSIS FACTS AND FIGURES

DID YOU KNOW?

- Sepsis claims 8 million lives each year worldwide – many of whom are children.
- There are approximately 250 000 cases of sepsis in the United Kingdom every year.
- There are approximately 46 000–52 000 deaths in the United Kingdom every year from sepsis.
- Sepsis claims more lives than breast, bowel, and prostate cancers put together and more lives than lung cancer.
- Sepsis costs the NHS approximately £1.5–£2 billion every year.
- Sepsis costs the wider economy approximately £11–£15.6 every year.
- Forty percent of all sepsis survivors suffer permanent life-changing effects.

WHAT HAPPENS TO THE BODY DURING SEPSIS?

The functional changes that occur during sepsis are as follows:

- The body senses that injury has occurred (the infection) and attempts to repair itself by releasing white blood cells (WBCs), platelets, and fibrin. In sepsis this process happens globally across the body.
- Vasodilation occurs as a means to move WBCs, fibrin, and platelets to where they are needed in the damaged tissues.
- The patient will now present as hot and initially warm around the peripheries.
- Capillary leakage occurs and the WBCs, fibrin, and platelets need to get inside the blood vessels and interstitial tissues where the pathogens are.

- The patient becomes oedematous.
- Inflammatory mediator molecules are released, causing vasodilation, pain at the site of infection, and clot formation.

RECOGNISING SEPSIS

More than 70% of cases of sepsis arise in the community, and yet a large proportion of the public do not recognise the symptoms that can lead to a delay in treatment.

One of the problems of sepsis is that individuals may feel unwell but may not have a high temperature so sepsis may be difficult to diagnose, leading to delayed treatment and eventually to septic shock – organ failure and death.

Sepsis is a clinical emergency. For each hour's delay in administering antibiotics in septic shock, mortality increases by 7.6%; therefore, identifying sepsis in the first instance is vital in saving lives. This is more difficult in those who are unable to communicate just how unwell they are feeling, for example, babies and adults with learning difficulties. This is where our observation skills come to the fore, and we need to ask, 'Are they acting differently from normal?' This is due to the fact that there is no one sign to diagnose sepsis. Also, symptoms present differently in adults and children.

Table 2.1 shows how to spot symptoms in adults; if they develop any of the symptoms, it may be sepsis.

Table 2.1 How to spot sepsis in adults.

S	**=**	**Slurred speech or confusion**
E	**=**	**Extreme shivering or muscle pain**
P	**=**	**Passing no urine (in 24 hours)**
S	**=**	**Severe breathlessness**
I	**=**	**It feels like you are going to die**
S	**=**	**Skin mottled or discoloured**

Source: Adapted from Sepsis Trust.

Table 2.2 shows how to spot symptoms in children. If they develop any of the symptoms, it may be sepsis, but if ever in doubt, act fast and dial 999 or go straight to accident and emergency department.

Table 2.2 How to spot sepsis in children.

1. Is the child breathing very fast?	**Y/N**
2. Has the child had a 'fit' or convulsion?	**Y/N**
3. Does the child look mottled, bluish, or pale?	**Y/N**
4. Does the child have a rash that does not fade when pressed?	**Y/N**
5. Is the child very lethargic or difficult to wake?	**Y/N**
6. Does the child feel abnormally cold to the touch?	**Y/N**
A child under five years old might have sepsis if they:	
Are not feeding	**Y/N**
Are vomiting repeatedly	**Y/N**
Have not passed urine for 12 hours	**Y/N**

RISK FACTORS TO SEPSIS

Anyone can get sepsis if they have an infection, but some people have a higher risk than others, for example, the immunosuppressed. Those at higher risk for sepsis than others include:

- Babies, younger than one year old
- People older than 75 years
- People who are 'frail'
- People with diabetes
- People with weak immune systems
- People who are undergoing chemotherapy and/or radiotherapy treatment
- Women who have just given birth or recently been pregnant (including those who have had a miscarriage or abortion)
- People who have recently had a serious illness

Babies are also at greater risk if they were born prematurely or their mother had an infection whilst pregnant, including a mild infection.

OBSERVATION CHARTS

One of the first assessments we can perform when suspecting sepsis is to perform a set of observations and plot the results on the National Early Warning Score 2 (NEWS2) observation chart. This chart enables us to assess our patients and care for them before their condition becomes critical or, at the very least, any worse. It is important we understand how to use the NEWS2 chart as gathering the vital signs recordings as part of this assessment, so here's a quick overview.

The NEWS2 observation chart can be seen in Appendix 1 and records six vital signs (or physiological measurements) in the order of A, B, C, D, and E on the chart:

A and B	**Relates to the respiration rate (RR)**
A and B	**Also relates to the patient's oxygen saturations (SpO_2)**
C	**Relates to the systolic blood pressure**
C	**Also relates to the pulse rate (also referred to as the heart rate)**
D	**Relates to the patient's consciousness or any new confusion**
E	**Relates to the patient's temperature**

This observation chart (NEWS2) can be used on:

- All adult in-patients (and care home residents)
- Occasionally children in adult wards
- Pregnant mothers up to 20 weeks

There are also adapted NEWS2 charts for patients requiring neurological observations (with the Glasgow Coma Scale incorporated into the chart). There are also adapted charts for use in maternity (for pregnant mothers >20 weeks' gestation, children, babies, and neonates) and for patients requiring-end-of-life care.

The NEWS2 chart is brightly coloured, whereby each vital sign generates a score of 0–3, depending on which colour zone the recording is plotted:

White	**Scores 0**
Yellow	**Scores 1**
Orange	**Scores 2**
Red	**Scores 3**

Once all the vital sign scores have been collected and plotted on the chart and totted up to give a score, we are directed as to what to do with this information, such as:

- Score of 0–4: Ward-based response
- Score of 3 in any individual parameter: Urgent ward-based response
- Score of 5–6: Urgent response
- Score ≥7: Emergency response

Patients with sepsis often present with a NEWS2 score >5, which is made up due to a persisting hypotension requiring vasopressors to maintain a MAP >65 mmHg. After serum lactate levels have been obtained (via venepuncture or arterially) with sepsis, these levels are >2 mmol/l (despite adequate volume resuscitation). We will look at lactate in detail later in this chapter.

Vasopressors
A drug or other agent that causes the constriction of blood vessels.

Before we continue with this chapter, we first need to have an understanding of some of the abbreviations we may use in relation to sepsis:

Activity 2.1

What do these healthcare abbreviations and/or terminology mean?

1. FBC
2. U's & E's
3. LFT
4. INR
5. APTT
6. MSU
7. CSU
8. Hb
9. WBC
10. CRP
11. Vasodilation
12. Vasoconstriction
13. AKI
14. CXR

SEPSIS SIX

The sepsis six consists of six recommendations to be delivered within **one hour** of the initial diagnoses of sepsis.

TOP TIP

The sepsis six is often remembered by stating 'three things in and three things out.'

IN: Administer oxygen.
Administer IV antibiotics.
Administer IV fluids.

OUT: Take blood cultures.
Take serum lactate levels and blood tests.
Measure urine output and start fluid balance chart.

Table 2.3 breaks down the whys and wherefores for each of the sepsis six recommendations.

Table 2.3 The sepsis six recommendations breakdown.

Sepsis six	Recommendations
IN: Oxygen	**Give 100% oxygen (15 l/min via face mask with reservoir bag unless oxygen restriction necessary (e.g. in chronic CO_2 retention – aim for an oxygen saturation of 88%–92%)**
IN: IV antibiotics	**Use as per own healthcare provider protocol. Prescribed first dose on the front of the prescription chart. Delay in administration increases mortality.**
IN: IV fluids	**Give a 500–1000 ml bolus of Hartmann's solution. Larger bolus may be required, e.g. if systolic blood pressure is <90 mmHg or lactate >4, consider 1500–2000 ml.**
OUT: Blood cultures	**Take as per Trust guidelines. Culture other sites as clinically indicated, e.g. sputum, wound swabs, cerebrospinal fluid. Consider chest X-ray.**
OUT: Lactate and bloods	**Lactate on atrial or venous sample. Also request full blood count, ureas and electrolytes, C-reactive protein, liver function test, clotting (International Normalised Ratio and activated partial thromboplastin) and glucose. Consider blood transfusion if haemoglobin is <7 (or above this with comorbidities).**
OUT: Urine output	**Monitor urine output hourly. Consider catheter. Perform urinalysis and send off midstream urine/catheter sample of urine.**

DID YOU KNOW?

LACTATE: Normal blood lactate concentration = 0.5–1.3 mmol/l. Patients with critical illness can be considered to have normal lactate concentrations <2 mmol/l.

The next sections will look at lactate and WBCs and their relation to sepsis. But first we can see from the sepsis six that a recommendation for 'bloods' is to consider a blood transfusion if haemoglobin (Hb) is >7 by some NHS Trusts. The next section looks at full blood count (FBC) normal values.

FULL BLOOD COUNT NORMAL VALUES

Red blood cells (RBCs) contain a substance called Hb, which transports oxygen around the body. The amount of oxygen that is delivered to the body tissues depends on the number of RBCs we have and how well they work. An RBC count is usually carried out as part of an FBC count blood test. The blood is obtained via venepuncture in an EDTA (ethylene diamine tetra acetic acid) additive tube, with a sample of blood consisting of approximately 2–4 ml. A normal RBC count is as follows, with women generally having a lower RBC than men:

Male adult: 4.7–6.1 million cells per microlitre (cells/µl)
Female adult: 4.2–5.4 million cells per microlitre (cells/µl)

An FBC comprises the following tests:

- Hb
- WBC count
- Platelet count (Plt)
- RBC count
- Haematocrit (HCT)
- Mean cell volume – red cell (MCV)
- Mean cell haemoglobin (MCH)
- WBC count:
    - Neutrophils
    - Lymphocytes
    - Monocytes
    - Eosinophils
    - Basophils

Table 2.4 lists normal values for an FBC.

Table 2.4 Full blood count normal values.

FBC test	Range	Units
Haemoglobin		
Adult male	**130–180**	**g/l**
Adult female	**115–165**	**g/l**
Red blood cell count		
Adult male	**4.50–6.50**	**$\times 10^{12}$/l**
Adult female	**3.80–5.80**	**$\times 10^{12}$/l**
Haematocrit		
Adult male	**0.40–0.54**	**1/l**
Adult female	**0.37–0.47**	**1/l**
Mean cell volume		
Adult	**80–100**	**fl**
Mean cell haemoglobin – adult	**27–32**	**pg**
White cell count – adult	**3.6–11.0**	**$\times 10^{9}$/l**
Neutrophils – adult	**1.8–7.5**	**$\times 10^{9}$/l**
Lymphocytes – adult	**1.0–4.0**	**$\times 10^{9}$/l**
Monocytes – adult	**0.2–0.8**	**$\times 10^{9}$/l**
Eosinophils – adult	**0.1–0.4**	**$\times 10^{9}$/l**
Basophils – adult	**0.02–0.10**	**$\times 10^{9}$/l**
Platelet count – adult	**140–400**	**$\times 10^{9}$/l**

FBC, full blood count.

Haematocrit
The ratio of the volume of red blood cells to the total volume of blood.

You may be interested to view a more complete list of normal Hb values, which includes values for babies and children. As you can see, results vary because of age and also vary slightly from pathology lab to pathology lab, but they generally fall within these parameters, as shown in Table 2.5. **NOTE:** You will need to check your own pathology lab's values.

Table 2.5 Haemoglobin normal values.

Age	Conventional units	SI units
Cord blood	13.5–20.7	135–207 g/l
0–1 week	15.2–2.6	152–236 g/l
2–3 weeks	12.7–18.7	127–157 g/l
1–2 months	9.7–17.3	97–173 g/l
3–11 months	9.3–13.3	93–133 g/l
1–5 years	10.4–13.6	104–136 g/l
6–8 years	10.9–14.5	109–145 g/l
9–14 years	11.5–15.5	115–155 g/l
15 years to adult Male Female	 13.2–17.3 11.7–15.5	 132–173 g/l 117–155 g/l
Older adult Male Female	 12.6–17.4 11.7–16.1	 130–180 g/l 115–161 g/l

Therefore, if a 35-year-old male's haematology result came back stating 119 g/l, this would be cause for concern.

WHITE BLOOD CELL COUNT

WBCs make up less than 1% of all the blood cells in the body, but play a vital role in keeping the body healthy. They are developed in the bone marrow, inside the bones, and are part of the immune system.

DID YOU KNOW?

Ants never get sick because of the fact that they have little ANTi-bodies!!

As part of the FBC in Table 2.4, we saw the normal ranges of five different types of WBC: neutrophils, lymphocytes, monocytes, eosinophils, and basophils. And I knew you desperately wanted me to give a quick run-down of these cells!

- **Neutrophils** – Fight bacteria and fungal infections. They make up 50%–70% of all WBCs.
- **Lymphocytes** – Fight infection, make antibodies, and destroy tumours. They are in both your blood and your lymph tissues.
- **Monocytes** – Remove damaged or dead cells. They travel into tissues such as the lungs and liver and become another kind of cell that eases inflammation.
- **Eosinophils** – Fight infections caused by parasites. They also respond to allergic reactions and inflammation.
- **Basophils** – Release a chemical called histamine during an allergic response. This triggers symptoms like a running nose and watery eyes (rhinitis).

If a WBC count is higher than the normal range – known as **leukocytosis** – this may be caused by:

- Infection
- Inflammation
- Leukaemia
- Burns
- Use of steroids
- Cigarette smoking
- Pregnancy

If a WBC count is lower than normal range – known as **leukopenia** – this can be caused by:

- A blood or bone marrow disorder
- A side effect of a drug
- An autoimmune system disorder
- A side effect of chemotherapy or radiation treatment
- A viral infection

Symptoms of leukopenia may include fever, body aches and pains, headaches, chills, night sweats, swollen lymph nodes, and/or an enlarged spleen (which may also reduce the WBC count).

Drugs that can lower your WBC count = chemotherapy, antiseizure medication, and antibiotics.

Drugs that can raise your WBC count = **albuterol** (used to widen airways in the treatment of asthma and other breathing problems), **lithium** (used to stabilise moods in the treatment of manic depression and bipolar disorder), and **heparin** (used to prevent blood clots).

LACTATE

Lactate is an essential test in the assessment of sepsis in acutely unwell patients and is obtained by venepuncture in fluoride-oxalate tubes in the amount of approximately 2 or 0.25 ml for neonates. Samples of lactate can also be obtained by the arterial route.

High levels of lactic acid could indicate:

- Sepsis
- Myocardial infarction
- Congestive heart failure
- Severe lung disease
- Respiratory failure
- Fluid buildup in lungs
- Severe anaemia

So what exactly is lactate? Lactate is the end product of anaerobic metabolism; is generated mainly in skeletal muscle, brain, erythrocytes, the skin, and intestine; and is disposed of by gluconeogenesis in the liver and by complete oxidation. Lactate acidosis can be caused by excessive lactate formation or decreased removal.

Type A lactic acidosis is primarily due to an increase in lactate formation as a consequence of tissue hypoxia, for example, shock (cardiogenic or haemorrhagic).

Type B lactic acidosis does not involve hypoxia as the primary event but is associated with the ingestion of drugs or toxins, severe liver disease, and certain inherited metabolic conditions.

SEPSIS TREATMENT PATHWAY

Once sepsis is suspected, the sepsis six pathway is initiated (known as the sepsis screening and action tool, see Appendix 2). Part of this pathway assesses whether any one of the 'red flag' indicators are present, which are clinical indicators of a diagnoses of sepsis:

- Responds only to voice or pain/unresponsive
- Acute confusional state
- Systolic blood pressure ≤90 mmHg (or drop >40 mmHg from normal)
- Heart rate >130 per minute
- Respiratory rate ≥25 per minute
- Needs oxygen to keep oxygen saturations ≥92%
- Non-blanching rash, mottled/ashen/cyanotic
- Has not passed urine in last 18 hours per urine output <0.5 ml/kg/h
- Lactate ≥2 mmol/l
- Recent chemotherapy
- Acute kidney injury present

Activity 2.2

Plot this information on the National Early Warning Score (NEWS) chart (Appendix 1). What is the NEWS score?
Respiratory rate = 25 breaths/min
Scale 1 oxygen saturation (SpO_2) = 92%
Supplementary oxygen administered
Blood pressure = 90 mmHg
Heart rate = 130
Responds only to voice and new confusion
Temperature = 38 °C

PRESCRIBED ANTIBIOTICS

The prescribed antibiotics used to treat sepsis vary from Trust to Trust and within the community setting, and you will need to view your own healthcare setting protocols, but some typical examples are shown in Table 2.6.

Table 2.6 Protocol for first-dose antibiotics in sepsis.

Community-acquired sepsis – source unknown	**Amoxicillin 2 g IV + flucloxacillin 2 g IV + gentamicin 5–7 mg/kg IV**
Community-acquired sepsis – suspected source of sepsis = chest/urine	**Amoxicillin 2 g IV + clarithromycin 500 mg IV + gentamicin 5–7 mg/kg IV**
Community-acquired sepsis – penicillin allergy	**Cotrimoxazole 960 mg IV + gentamicin 5–7 mg/kg IV**
Hospital-acquired sepsis – source unknown	**Cotrimoxazole 960 mg IV + gentamicin 5–7 mg/kg IV**
Neutropenic sepsis/gentamicin contraindicated	**Piperacillin/Tazobactam 4.5 g IV**

TOP TIP

Doctors often write up piperacillin/tazobactam as 'Tazocin' on prescription charts – confusing!

Neutropenic sepsis

A life-threatening complication of cancer treatment, the term is used to describe a significant inflammatory response to a presumed bacterial infection in a person with or without fever.

Neutropenia

An abnormally low number of neutrophil granulocytes in the blood.

TEST YOUR KNOWLEDGE

1. How many lives does sepsis claim worldwide?
2. True or false: Individuals 65 years old are at greater risk, if they have an infection, for sepsis.
3. What score does a heart rate of 91 beats/min generate on the NEWS2 observation chart?
4. What does 'MAP' mean? Clue, nothing to do with orienteering.
5. What does the abbreviation CRP mean?
6. What are the sepsis six?
7. What is the normal value of lactate?
8. What is the normal range of basophils in adults?
9. Is a haematology result of 135 g/l within the normal values for adult males?
10. What does leukopenia mean?

KEY POINTS

- Understanding sepsis
- Recognising sepsis
- Risk factors of sepsis
- Sepsis six
- Full blood counts
- Sepsis treatment

WEB RESOURCES

Sepsis Trust: https://sepsistrust.org
NHS – symptoms of sepsis: https://www.nhs.uk/conditions/sepsis
NHS England – sepsis: https://www.england.nhs.uk/ourwork/clinical-policy/sepsis
Sepsis: https://www.nice.org.uk/guidance/ng51

CHAPTER 7
Acute Kidney Injury

By the end of this chapter you will be able to:

- Define acute kidney injury (AKI)
- Understand that AKI is a complex clinical syndrome
- Know the factors that affect renal perfusion
- Appreciate the importance of preventing AKI
- Use a simple checklist for treating most cases of AKI
- Know the indications for renal replacement therapy
- Apply this to your clinical practice

Acute kidney injury (AKI) is common. It is found in at least 20% of patients admitted to acute hospitals and 50% or more of patients admitted to specialist areas such as intensive care and cardiac surgery units.[1] AKI has traditionally been viewed as a single disease, classified according to the anatomy of the kidney (i.e. prerenal, renal, and postrenal). We now know it is a complex clinical syndrome involving several different, often overlapping, diseases. These include the hepatorenal syndrome,[2] cardiorenal syndrome,[3] sepsis,[4] and the use of nephrotoxic drugs.[5] Each appears to have its own unique pathophysiology as well as treatment. A challenge in the diagnosis of AKI and its management is therefore recognising that these conditions often overlap and coexist, as illustrated in Figure 7.1. Because AKI often occurs as a result of other diseases, it can be considered a marker of severity of disease and a predictor of short- and long-term outcomes.

Definitions

The international consensus criteria for AKI were first introduced by the Acute Dialysis Quality Initiative,[6] subsequently modified by the AKI Network,[7] and finally by Kidney Disease Improving Global Outcomes (KDIGO).[8] AKI is defined as *any* of the following:

Essential Guide to Acute Care, Third Edition. Nicola Cooper, Paul Cramp, Kirsty Forrest and Rakesh Patel.

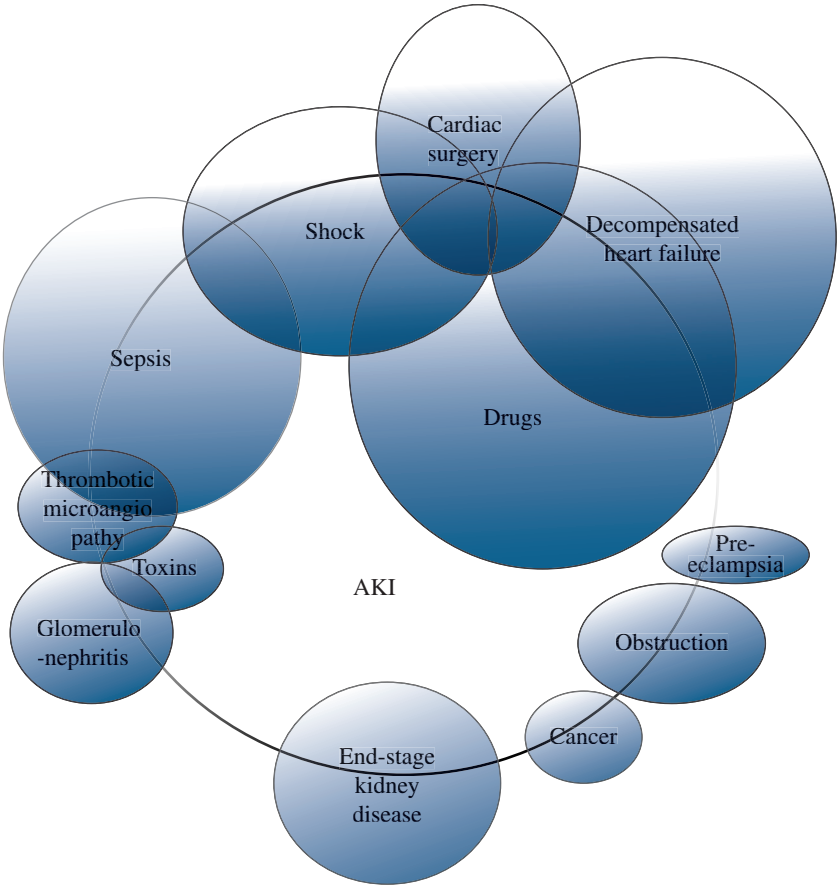


Figure 7.1 Overlapping clinical conditions in AKI.

- An increase in serum creatinine by ≥0.3 mg/dL (≥26.5 μmol/L) within 48 hours
- An increase in serum creatinine to >1.5 times baseline, known or presumed to have occurred within the last 7 days
- A urine output ≤0.5 mL/kg/h for 6 hours

AKI is staged according to its severity, as illustrated in Table 7.1. *Chronic* kidney disease (CKD) is defined as abnormalities of kidney structure or function present for at least 3 months, with implications for health (see Table 7.2). 'Acute kidney disorder' refers to abnormalities present for 7 days to 3 months which could be AKI or CKD.

Single creatinine measurements are unhelpful in deciding whether a person has AKI or CKD. Creatinine is a nitrogenous waste product derived from muscle. Elderly people have less muscle mass and can have a 'normal' creatinine with impaired renal function. Likewise, athletes may have a 'high' creatinine with normal renal function. Therefore, labs in many countries routinely report an estimated glomerular filtration rate (eGFR) alongside creatinine measurements. The eGFR is an *estimate* of creatinine clearance based on creatinine,

Table 7.1 Staging of AKI (KDIGO 2012 definitions).

Stage	Creatinine	Urine output
1	1.5–1.9 times baseline Or Increase of ≥0.3 mg/dL (25 μmol/L)	<0.5 mg/kg/h for 6–12 h
2	2.0–2.9 times baseline	<0.5 mg/kg/h for ≥12 h
3	3.0 times baseline Or Increase to ≥4.0 mg/dL (353.6 μmol/L) Or Initiation of renal replacement therapy	<0.3 mg/kg/h for ≥24 h Or Anuria for ≥12 h

Table 7.2 Definitions of CKD.

Either one of the following present for >3 months, with implications for health:	
Markers of kidney damage (one or more)	Albuminuria (albumin–creatinine ratio ≥30 mg/g) Urine sediment abnormalities Electrolyte and other abnormalities due to tubular disorders Abnormalities detected by histology Structural abnormalities detected by imaging Renal transplant
Decreased eGFR	<60 mL/min/1.73 m^2

Source: Modified from KDIGO Clinical Practice Guideline for Acute Kidney Injury. Notice. Kidney Int 2012; 2:1–141.

age, sex, and race. However, a formal creatinine clearance calculation should be used when prescribing medications, as some drugs should be given in lower doses to patients with reduced renal function. Creatinine clearance can be calculated using the equation:

$$\text{Creatinine clearance} = \frac{(140 - \text{age}) \times \text{weight in kg}}{\text{creatinine in } \mu\text{mol/L}} \ (\times 1.2 \text{ for men})$$

Basic Renal Physiology

Renal blood flow in a 70 kg man is around 1200 mL/min, which is 20–25% of cardiac output, making the kidneys among the most highly perfused organs in the body. Various factors affect renal blood flow, as illustrated in Figure 7.2. There is autoregulation of renal blood flow between a mean arterial pressure (MAP) of 70–130 mmHg in the average healthy person. This is a vital homeostatic mechanism designed to protect the kidney from injury and allow it to maintain a relatively constant GFR necessary to clear waste

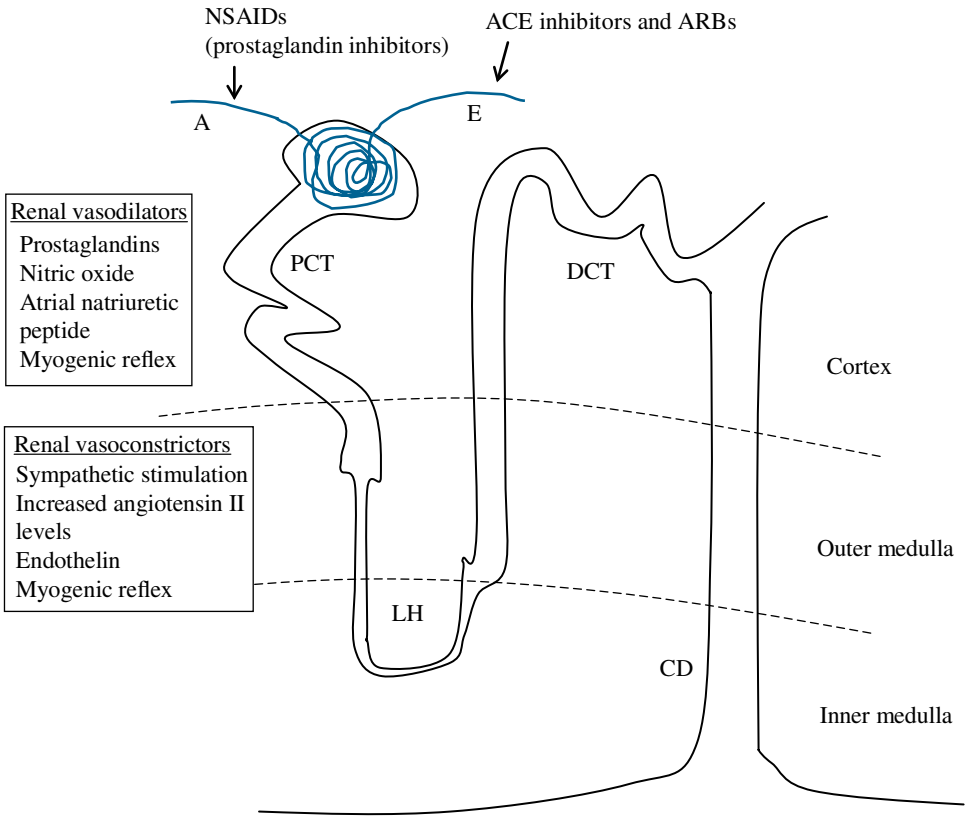


Figure 7.2 Factors affecting renal blood flow. A = afferent arteriole; ACE = angiotensin converting enzyme; ARB = angiotensin receptor blocker; CD = collecting duct; DCT = distal convoluted tubule; E = efferent arteriole; LH = thick and thin descending and ascending Loop of Henle; NSAIDs = non-steroidal anti-inflammatory drugs; PCT = proximal convoluted tubule. Vasodilatory prostaglandins increase renal blood flow and glomerular filtration rate under conditions associated with reduced actual or effective circulating volume, resulting in greater tubular flow and secretion of potassium. Renin is also produced in the kidneys in response to reduced blood pressure or sympathetic stimulation, producing angiotensin II leading to vasoconstriction and an increase in blood pressure. In a person on an ACE inhibitor and an NSAID who is admitted with any condition causing hypovolaemia or low blood pressure, these physiological mechanisms designed to increase renal blood flow are blocked, leading to kidney injury.

products from the body. The ability of the kidney to maintain a relatively constant blood flow, GFR, and glomerular capillary pressure is mediated by the myogenic response of afferent arterioles working in concert with tubulo-glomerular feedback in response to changes in the concentration of sodium chloride reaching the distal tubules. Below a MAP of 70 mmHg, renal blood flow and GFR fall sharply, as illustrated in Figure 7.3. Autoregulation is impaired in people with hypertension, diabetes, and other forms of chronic kidney disease, which is one reason why these people are more susceptible to AKI in acute illness.

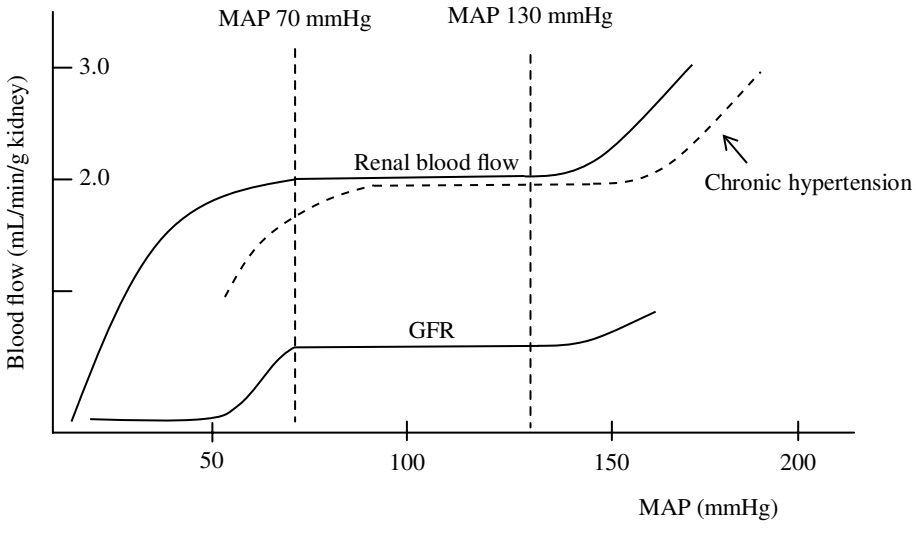


Figure 7.3 Autoregulation of renal blood flow. Elevations in vascular resistance occur in hypertension, especially in the renal circulation. There is also generalised endothelial dysfunction, hypertrophy of pre-glomerular arteriolar walls, and diminished vasodilatory responses. Autoregulation is shifted to the right, and the magnitude of the shift depends on the degree and duration of the hypertension.

The Pathophysiology of AKI

The pathophysiology of AKI varies according to the myriad of conditions associated with it and there is still a lot we do not understand. Most cases of AKI admitted to hospital are due to renal hypoperfusion, sepsis, and cardiorenal syndrome, often in combination with nephrotoxic drugs. Most cases of hospital-acquired AKI are multifactorial, for example, in people admitted for major surgery. 'Renal' causes of AKI (e.g. a rapidly progressive glomerulonephritis) are uncommon but are extremely important not to miss.

Renal Hypoperfusion

Renal hypoperfusion (e.g. due to hypovolaemia, low blood pressure, or reduced blood flow due to other causes) activates protective physiological mechanisms within the kidney in order to maintain GFR. If the hypoperfusion is sustained or the response is inadequate, GFR will decrease, initially without any structural damage. If renal perfusion is not restored within a few hours, then ischaemic necrosis can occur, along with endothelial injury, activation of inflammatory mediators, and further renal damage.

Sepsis-Associated AKI

As described in Chapter 5, in sepsis there is macrovascular and microvascular dysfunction, immunological dysfunction, and abnormal cellular responses. Sepsis leads to an increase in circulating inflammatory cytokines and leucocyte activity which leads to the formation

of capillary microthrombi and a redistribution of intrarenal perfusion. This produces kidney inflammation, oedema, reduced capillary blood flow, reduced oxygen delivery, and increased venous output pressures. Imbalances in reactive oxygen species or nitric oxide production also contribute to endothelial damage, increased vascular permeability, and worsening interstitial oedema.[9]

Cardiorenal Syndrome

Cardiorenal syndrome is the term used to describe a spectrum of disorders involving both the heart and kidneys in which acute or chronic dysfunction in one organ can induce acute or chronic dysfunction in the other. It occurs as a result of 'haemodynamic cross-talk' between the failing heart and the response of the kidneys and vice versa, as well as alterations in neurohormonal markers and inflammatory mediators. The condition known as type 1 cardiorenal syndrome reflects an abrupt worsening of cardiac function (e.g. acute or decompensated congestive heart failure) and can develop because of a low cardiac output, renal vein congestion, or both.[10] The kidney plus cardiac dysfunction affects kidney perfusion pressures and compensatory mechanisms may be insufficient to maintain an adequate renal blood flow. Inflammation, neurohumoral activation plus the effects of drugs, and the presence of pre-existing CKD also contribute to the development of this syndrome.[11] A common scenario is that when patients with decompensated congestive heart failure are admitted to hospital with a deterioration in renal function, their 'nephrotoxic' medications are suspended and they may even be given fluid. We now know this is detrimental and this topic is explored further in the mini-tutorial.

Mini-Tutorial: Treatment of AKI in Decompensated Heart Failure

Cardiorenal syndrome is a spectrum of disorders in which heart failure can cause renal dysfunction *or* renal dysfunction can cause heart failure.[10] When a patient first presents, it may not be possible to identify in which organ the syndrome first originated. However, a common scenario in acute care settings is when patients on treatment for decompensated heart failure develop AKI.

Impaired renal function is common in patients with congestive heart failure and is associated with worse outcomes. Drugs used to treat heart failure, such as ACE inhibitors and angiotensin-receptor blockers (ARBs) improve long-term outcomes. National Institute of Health and Care Excellence (NICE) guidelines[12] recommend that, following the introduction of ACE inhibitors or ARBs, a fall in eGFR of up to 25% or a rise in creatinine of up to 30% is acceptable. Hyperkalaemia may be a reason to reduce or stop drug treatment.

When a patient whose primary problem is heart failure presents with worsening oedema and AKI, more diuretics are required. The kidneys are 'biochemically sacrificed' in order to reduce preload and optimise cardiac output. Guidance on changes in renal function associated with drug treatment in heart failure[13] can be summarised as follows:

- If a patient is 'dry', then diuretics should be stopped or withheld temporarily
- In patients with fluid retention, higher doses of diuretics are needed and a decline in renal function is not an indication to reduce the diuretic dose. If the patient remains congested, more diuretics are required
- The only way to tell whether a patient has fluid retention or is hypovolaemic is to examine them clinically

In patients with decompensated congestive heart failure and AKI, loop diuretics are the diuretic of choice. However, they are dependent on an adequate GFR to exert their effect; therefore, it follows that the worse the AKI, the greater the dose of loop diuretic that is required. If a patient is admitted with fluid overload due to decompensated heart failure, a useful rule of thumb is to find out the usual total daily dose that previously maintained them in euvolaemia and then increase this by 25–50%. For example, if the usual maintenance dose was furosemide 80 mg twice a day, the dose could be increased to 240 mg of furosemide once a day as an intravenous infusion. Once diuresis is stimulated and there is appreciable weight loss, the drug can be divided into a twice daily oral regimen as maintenance.

Box 7.1 Common Nephrotoxic Drugs

- Acyclovir
- Aminoglycosides
- Amphotericin
- ACE inhibitors
- Angiotensin receptor blockers
- Antibiotics (e.g. flucloxacillin)
- Ciclosporin
- Cisplatin
- Methotrexate
- Non-steroidal anti-inflammatory drugs
- Radiocontrast agents
- Sulphonamides
- Tacrolimus

Nephrotoxic Drugs

Drug-induced AKI is important to detect because the offending drug can be stopped or substituted for one that is non- or less nephrotoxic. Nephrotoxic drugs are implicated in roughly one-fifth of critically ill patients with AKI.[14] A list of commonly prescribed drugs that contribute to AKI is given in Box 7.1. Nephrotoxic drugs impact on the kidney in different ways. Antibiotics and endogenous toxins (e.g. myoglobin, uric acid) are filtered and concentrated and can reach toxic levels, having a direct cytotoxic effect on

renal tubular cells, affecting intrarenal blood flow or causing precipitation of metabolites or crystals.[15] In acute interstitial nephritis, drugs or infectious agents can also activate an immune reaction in patients who are genetically predisposed, leading to an interstitial inflammatory cellular infiltrate which in turn stimulates the production of cytokines, eventually (if not interrupted) causing interstitial fibrosis and CKD.[16]

Iodinated radiocontrast agents are an important cause of AKI. Contrast agents are directly toxic to tubular epithelial cells, but there are also vasomotor changes mediated by endothelin, nitric oxide, and prostaglandins. The outer renal medulla has a relatively low partial pressure of oxygen, making it particularly susceptible to the haemodynamic effects of contrast in situations where there is also a high metabolic demand. Patients with pre-existing kidney disease, the lowest levels of kidney function, and those receiving higher doses of contrast are most at risk. Contrast-associated AKI is associated with increased mortality, but it is not clear whether it is a mediator or simply a marker of adverse outcomes, as there are currently no adequately powered trials showing that prevention of contrast-associated AKI reduces mortality.[17] The increments in plasma creatinine levels that are used to define acute kidney injury are common in patients who have undergone procedures using contrast, but this is also true of hospitalised patients in general. However, the incidence of *severe* acute kidney injury due to contrast material (i.e. creatinine >50% of baseline, or requiring dialysis) is very low – 1.2% in one study of elective coronary angiography patients with CKD[18] and 0.3% of patients undergoing contrast CT scans.[19] Intravenous sodium chloride 0.9% in the few hours before and after the procedure (e.g. at a rate of 125 mL/h) probably prevents contrast-associated AKI (although caution may be required in patients with congestive heart failure). There is no evidence that using oral *n*-acetylcysteine offers a protective benefit. Bottom line: if your patient needs an urgent intervention because of a serious (even potentially life-threatening) acute illness – *proceed* with the intervention and explain the risks and benefits to the patient. Stop nephrotoxic medication, administer intravenous sodium chloride 0.9%, use the lowest dose of contrast medium possible, and monitor renal function closely following the procedure.

Hepatorenal Syndrome

Hepatorenal syndrome is the most extensively studied form of AKI in terms of neurohumoral changes. In this syndrome, there is intense renal vasoconstriction due to renin–angiotensin–aldosterone activation, accompanied by fall in systemic blood pressure due to splanchnic vasodilatation. This compromises renal blood flow. In patients with decompensated liver disease and tense ascites, increased intra-abdominal pressure can also contribute to a reduction in renal perfusion. This is why treatment of this syndrome includes: suspending nephrotoxic drugs, fluid resuscitation and correction of hypotension, terlipressin (see Chapter 5), and drainage of tense ascites.

AKI in Major Surgery

AKI is common in patients undergoing major surgery and is associated with poor short- and long-term outcomes. Surgery is a leading cause of AKI acquired in hospital, with an incidence

of 5–7.5% among general in-patients and 50–60% of critically ill patients. Patients most at risk are those who are older with hypertension, diabetes, or CKD. The highest rates of AKI are found after cardiac surgery. In major surgery, fluid losses (e.g. blood loss, insensible losses, and extravasation of fluid in to the third space) and the effects of anaesthetic drugs (e.g. peripheral vasodilation, myocardial depression) are thought to be the main causes of AKI.[20]

Obstruction

Extrarenal (e.g. prostatic hypertrophy) or intrarenal (e.g. stones) obstruction leads to an increase in intratubular pressures in the kidney, leading to impaired blood flow and inflammation that can result in AKI, depending on previous kidney function and the severity of the obstruction.

Preventing AKI

The first and most important principle is to anticipate and mitigate all potential causes or triggers of AKI *before* they happen. The second principle is to ensure that further insults are avoided after AKI occurs.[21] Intravascular volume depletion should be corrected. Mean arterial pressure should be optimised so that renal perfusion is maintained, thereby minimising further damage. Nephrotoxic drugs should be stopped. The assessment of intravascular volume status can be monitored by physical examination (see Chapter 5). Dynamic tests such as the response to fluid challenges and passive leg raising may provide additional information.

In critically ill patients, invasive haemodynamic monitoring and vasopressor drugs may be required to increase mean arterial pressure. The use of hydroxyethyl starch has been shown to result in increased rates of AKI especially in patients with sepsis.[22] The use of sodium chloride 0.9% has been shown to increase the risk for composite death, dialysis, and persistent renal dysfunction compared to more balanced solutions such as Hartmann's.[23]

Theoretically, loop diuretics may protect the loop of Henle from ischaemia by decreasing its transport-related workload. However, no results from double blinded randomised controlled trials of suitable size have shown that these agents reduce the incidence of AKI.[24] Although no specific drug-based intervention has been shown to be of benefit, avoidance of nephrotoxic drugs probably shortens the course of AKI. For those patients at particular risk of contrast-associated AKI (i.e. interventional cardiology and oncology patients), intravenous *n*-acetylcysteine or sodium bicarbonate do not provide additional benefit beyond hydration therapy alone.[25]

Assessment of Patients with AKI

In deciding what has caused AKI, and whether renal impairment is due to AKI, CKD, or AKI on a background of CKD, the patient's history usually gives the answer. In the majority of admissions to hospital with AKI, there is an acute and identifiable cause: hypovolaemia

(e.g. diarrhoea and vomiting), sepsis, decompensated heart failure, often in combination with nephrotoxic drugs. Obstruction causing retention of urine due to an enlarged prostate, cancer, haematuria, or stones can also usually be diagnosed on history, physical examination, and a bedside bladder scan (but remember that basic bladder scanners cannot detect a urine volume in the presence of ascites).

In some cases, AKI is due to intrinsic kidney disease. Of these, vasculitis, glomerulonephritis, and interstitial nephritis are the most common. The clinical features suggesting one of these diagnoses range from non-specific symptoms (e.g. malaise, darker/less urine) to symptoms that point towards kidney disease (e.g. oedema, proteinuria, microscopic haematuria, and hypertension) to systemic manifestations of classical syndromes (e.g. Henoch–Schönlein purpura, scleroderma). There may also be temporal association with starting a drug known to cause interstitial nephritis. When there is no clear 'pre-renal' cause or obstruction, bedside urinalysis to look for proteinuria and/or microscopic haematuria and urine microscopy is important. Urine microscopy can suggest pathological glomerular changes such as fragmented red cells, red cell casts, white cell casts, or granular casts. The urine microscopy score (based on the quantification of tubular cells and casts) correlates with worsening AKI, need for renal replacement therapy, and hospital mortality.[26]

Kidney disease is usually a silent condition and therefore AKI can also be diagnosed in the absence of an acute illness when there is a reduction in kidney function within the past 3 months, with or without a change in urine output. Some patients present with abnormal kidney function of unknown duration and the challenge is to decide whether this is AKI or the first presentation of CKD or both (AKI on CKD). In these situations, the history (e.g. presence of risk factors), the presence or absence of albuminuria, kidney size on ultrasound (small in CKD), and the presence of features of CKD (e.g. normocytic anaemia, hyperphosphataemia, and high parathyroid hormone levels) can be helpful in distinguishing between AKI and CKD.

Management of AKI

Early action can save kidneys. In general, the management of AKI involves six simple steps:

1) Treat hyperkalaemia first (see Box 7.2)
2) Correct volume depletion
3) Treat hypoperfusion
4) Exclude obstruction
5) Stop nephrotoxins
6) Treat the underlying cause

Box 7.2 Treatment of Hyperkalaemia

Hyperkalaemia is defined as:

- Mild 5.5–5.9 mmol/L (meq/L)
- Moderate 6.0–6.4 mmol/L (meq/L)
- Severe >6.5 mmol/L (meq/L)

The principles of treatment of severe hyperkalaemia are:

- Protect the heart
- Shift potassium in to cells (this is only temporary)
- Remove potassium from the body
- Prevention of recurrence

This is done by administering:

- Intravenous calcium
- Intravenous insulin and dextrose
- Nebulised salbutamol can be added but is not effective in up to 40% of patients with end-stage renal disease – the potassium-lowering effect of nebulised salbutamol occurs within 30 minutes and lasts for up to 2 hours. The serum potassium level may fall by between 0.5 and 1.0 mmol/L when 10–20 mg of nebulised salbutamol is delivered[27]
- Stop drugs that may contribute to hyperkalaemia, institute low potassium diet, treat AKI

The patient should have frequent capillary glucose monitoring and repeat potassium levels.

Remember that pseudohyperkalaemia is most commonly caused by *in vitro* haemolysis and the presence of haemolysis is usually reported by the lab. In true hyperkalaemia, both the absolute value of serum potassium and the rate of change are risk factors for electrocardiogram (ECG) changes, and some susceptible individuals may develop cardiac arrythmias at lower levels of hyperkalaemia. The ECG changes in hyperkalaemia include 'tented' (tall) T waves, a prolonged PR interval, and then loss of P waves, QRS widening, and arrythmias.

In critically ill patients with established AKI, fluid overload is often present, and maintaining nutrition and administering crucial drugs often requires the administration of at least 1500 mL of fluid per day. Early renal replacement therapy is the best treatment in this situation, as fluid overload has been recognised as a major contributor to increased mortality in patients with AKI.[28]

Mini-Tutorial: The Use of Intravenous Sodium Bicarbonate in AKI

Current UK guidelines for the management of AKI[29] recommend the administration of intravenous sodium bicarbonate for a severe metabolic acidosis (pH < 7.2). Acute acidosis causes myocardial depression and cellular dysfunction, so in theory correction of acidosis could be beneficial. However, this is not without controversy (see mini-tutorial in Chapter 3) and the few studies that have shown a beneficial effect of sodium bicarbonate in AKI have been in the intensive care population. The first randomised trial involved 389 patients with a pH of <7.2 who received intravenous sodium bicarbonate to raise the pH level to at least 7.3. There was no difference in 28-day mortality overall, but in the subset of patients with AKI, there was a survival benefit, and they also required less vasopressors and renal replacement therapy.[30] The second randomised trial involved patients with sepsis and reported similar results.[31] The use of sodium bicarbonate in non-critically ill patients and less severe metabolic acidosis is unclear.

Oral sodium bicarbonate is sometimes used in patients with severe chronic kidney disease. The kidney is integral to maintaining acid–base balance in normal health and the ability of the kidney to perform this function declines when the GFR falls to less than 30 mL/min/1.73 m^2. The dose of sodium bicarbonate can be increased in the setting of an acute intercurrent illness and given intravenously if necessary. Sodium bicarbonate can cause fluid retention in higher doses which can be important in patients with heart failure or hypertension.

Renal Replacement Therapy

In some patients, AKI is severe enough to require renal replacement therapy. The indications for starting renal replacement therapy in AKI include:

- Oliguria/anuria
- Resistant hyperkalaemia
- Fluid overload
- Severe metabolic acidosis (pH < 7.2)
- Uraemia (urea > 30 mmol/L or BUN > 83 mg/dL) including its complications (e.g. encephalopathy, pericarditis, and seizures)

Three forms of renal replacement therapy are available: continuous, intermittent (either as intermittent haemodialysis or slow low-efficiency dialysis), and peritoneal dialysis. Continuous renal replacement therapy can involve filtration alone (e.g. continuous veno-venous haemofiltration) or diffusion alone (e.g. continuous veno-venous haemodialysis) or both (continuous veno-venous haemodiafiltration). Peritoneal dialysis is rarely used in AKI due to clearance limitations and difficulty with fluid removal; however, it is often used to start patients with established kidney disease safely on renal replacement therapy.

Evidence from various small- and medium-sized trials suggest little difference in patient outcomes between intermittent renal replacement therapy or continuous renal replacement

therapy.[32] There is also little difference in patient survival rates or time to recovery from AKI with increasing intensity of renal replacement therapy.[33]

Prognosis of AKI

Nearly two-thirds of AKI cases resolve within 7 days.[34] When AKI does not resolve, substantially worse clinical outcomes can be expected. Patients with stage 2–3 AKI who resolve within 7 days and remain alive and free of renal dysfunction by hospital discharge have a 1-year survival of more than 90%. In contrast, patients whose AKI never resolves have a 47% hospital mortality and among those who are discharged alive, the 1-year survival is 77%.[34] In the long term, several studies have demonstrated a link between AKI and the subsequent development of CKD.[35] Not all episodes of AKI lead to death or CKD, but patients with risk factors for progression (e.g. diabetes, hypertension, and heart failure) should be followed up long term.

Key Points: AKI

- AKI is common and diagnosed when there is an acute reduction in kidney function, with or without a change in urine output
- AKI is a complex clinical syndrome involving several different, often overlapping, conditions
- Renal blood flow is autoregulated between a MAP of 70–130 mmHg and this is impaired in people with hypertension, diabetes, and other forms of chronic kidney disease
- AKI can be prevented
- A simple checklist can be used to treat most cases of AKI – but treatment of AKI in decompensated heart failure and some other conditions may be different
- Two-thirds of cases of AKI resolve within 7 days but some patients need renal replacement therapy and patients with risk factors for progression to CKD should have long-term follow-up
- The prognosis of unresolved AKI is poor

Self-Assessment: Case Histories

1 A 31-year-old man is admitted to hospital after being found on the floor of his apartment. He had taken intravenous heroin the night before. His vital signs are: drowsy, blood pressure 93/61 mmHg, pulse 108/min, temperature 35°C, respiratory rate 8/min, and oxygen saturations 95% on air. His blood results show a normal full blood count, sodium 131 mmol/L, potassium 6.5 mmol/L, urea 30 mmol/L (BUN 83 mg/dL), creatinine 600 µmol/L (7.2 mg/dL), and calcium 1.9 mmol/L (7.6 mg/dL). He looks dehydrated. What is your management?

2 An 82-year-old year man is admitted with 'general deterioration' and not eating or drinking. He is usually treated for heart failure and is taking the following medications: ramipril 10 mg daily, furosemide 80 mg daily, and allopurinol 300 mg daily. He was treated for a chest infection 1 week previously with amoxicillin and also took ibuprofen for pleuritic chest pain. His vital signs are: drowsy, blood pressure 90/60 mmHg, pulse 90/min, temperature 37°C, respiratory rate 20/min, and oxygen saturations 95% on air. His blood results show: sodium 133 mmol/L, potassium 5.1 mmol/L, urea 29 mmol/L (BUN 80 mg/dL), and creatinine 483 µmol/L (5.7 mg/dL). His last available blood results are from 2 months ago and show a urea of 7 mmol/L (BUN 19.5 mg/dL) and creatinine 120 µmol/L (1.44 mg/dL) with an eGFR of 53.4 mL/min/1.73 m^2. At that time, his blood pressure was 140/80 mmHg. On physical examination, there are no signs of fluid overload and his lungs are clear. He has dry axillae. What is your management?

3 A 34-year-old woman is admitted with breathlessness that started 1 week ago. The chest X-ray shows bilateral patchy shadowing and she reports coughing up small amounts of blood with no sputum. Her vital signs are: alert, blood pressure 181/85 mmHg, pulse 81/min, temperature 37.5°C, respiratory rate 20/min, and oxygen saturations 94% on air. She has no history of vomiting or diarrhoea and appears to be euvolaemic on physical examination. Her blood results show a normal full blood count, sodium 135 mmol/L, potassium 4.2 mmol/L, urea 33 mmol/L (BUN 91.6 mg/dL), and creatinine 451 µmol/L (5.41 mg/dL). What is your management?

4 You are asked to see a 55-year-old man on a surgical ward. He is being treated for ascending cholangitis and had a failed endoscopic retrograde cholangio-pancreatogram (ERCP) today to retrieve a common bile duct stone. His medication chart shows a beta-blocker, calcium channel blocker, and a nitrate for angina. He has no other past medical history. His vital signs are: alert, blood pressure 87/62 mmHg, pulse 85/min, respiratory rate 28/min, temperature 38.1°C, and oxygen saturations 95% on air. He has warm hands and feet. He also has a left nephrectomy scar from 15 years ago. He has been given gentamicin for his infection. The nurse alerts you to his urine output which has been 10 mL/h for the last 3 hours. What is your management?

5 A 63-year-old woman is admitted with diarrhoea and vomiting which she has had for 4 days. Her usual medication comprises bendroflumethiazide and ramipril for hypertension. On admission, her vital signs are: alert, blood pressure 94/67 mmHg, pulse 108/min, temperature 37.7°C, respiratory rate 22/min, and oxygen saturations 98% on air. She reports passing less urine in the last 24 hours. Her blood results show: sodium 145 mmol/L, potassium 4.0 mmol/L, urea 25 mmol/L (BUN 69.4 mg/dL), and creatinine 309 µmol/L (3.70 mg/dL). From her records, her eGFR was normal 1 month ago. What is your management?

6 An 84-year-old woman is admitted after sustaining a fractured neck of femur. Her vital signs are: alert, blood pressure 180/80 mmHg, pulse 75/min, temperature 36.6°C, respiratory rate 14/min, and oxygen saturations 95% on air. On admission, her haemoglobin is

13.5 g/dL, sodium 139 mmol/L, potassium 4.0 mmol/L, urea 6 mmol/L (BUN 16.6 mg/dL), and creatinine 55 μmol/L (0.66 mg/dL). She is prescribed a non-steroidal anti-inflammatory drug for pain. In theatre, she had a 10 minute period of hypotension (85/60 mmHg). Two days postoperatively her blood results are as follows: haemoglobin 10.5 g/dL, sodium 130 mmol/L, potassium 3.8 mmol/L, urea 23 mmol/L (BUN 63.8 mg/dL), and creatinine 254 μmol/L (3.05 mg/dL). What is your management?

7 A 52-year-old man with early diabetic nephropathy is admitted to the coronary care unit with an inferolateral myocardial infarction. He suffers a VF arrest and has no cardiac output for 5 minutes. He has a period of hypotension following this and is treated with inotropes. Although his cardiac condition recovers, his renal function deteriorates. On admission, his urea was 12 mmol/L (33.3 mg/dL) and creatinine 157 μmol/L (1.88 mg/dL). Forty-eight hours later his urea is 27 mmol/L (75 mg/dL) and creatinine 317 μmol/L (3.8 mg/dL). What are the possible reasons for the change in renal function and what is your management?

8 A 56-year-old woman undergoes an elective abdominal aortic aneurysm repair. The aneurysm was located above the renal arteries and the aorta was cross-clamped for 30 minutes. She returns to the intensive care unit from theatre still ventilated. Her vital signs are: pulse 110/min, blood pressure 120/80 mmHg, CVP 10 mmHg, and temperature 36.5°C. Her arterial blood gases on 35% oxygen show: pH 7.2, $PaCO_2$ 4.0 kPa (30.7 mmHg), base excess (BE) 10, and PaO_2 25.0 kPa (192 mmHg). Her urine output has been 20 mL/h for the last 2 hours. What is your management?

9 A 70-year-old man is referred by the community heart failure team because of increasing breathlessness, peripheral oedema, and a reduction in renal function. On admission, his vital signs are: alert, blood pressure 110/60 mmHg, pulse 103/min, temperature 36.7°C, respiratory rate 22/min, and oxygen saturations 93% on air. On examination, he has pitting oedema of his lower limbs and abdominal wall. His chest X-ray shows bilateral pleural effusions and interstitial oedema. His diuretics had been increased recently, but his breathlessness and oedema did not improve and his renal function deteriorated. On admission, his urea is 13 mmol/L (36.1 mg/dL) and creatinine 236 μmol/L (2.83 mg/dL) with an eGFR of 25.3 mL/min/1.73 m^2. His blood results 3 months ago showed an eGFR of 35 mL/min/1.73 m^2 and his renal function has been steadily declining since. What is your management?

Self-Assessment: Discussion

1 Management starts with assessing and treating problems with A (airway), B (breathing), C (circulation), and D (disability). In this case, this will include fluid challenges and intravenous or intramuscular naloxone. Lower doses of naloxone should be given to long-term opiate users.[36] While this patient's previous renal function may be unknown, information from the history suggests acute kidney injury rather than CKD. Normal sized kidneys on ultrasound would support this. A 'long lie' (lying on the floor

for more than 1 hour and unable to get up) and some drug overdoses can cause rhabdomyolysis. Myoglobin and urate from muscle breakdown mediate kidney injury. This can be confirmed by measuring creatinine kinase levels, which are usually in the tens of thousands, and testing the urine for myoglobin (alternatively blood+++ on urinalysis but no red cells on microscopy). The typical picture of severe rhabdomyolysis is a high creatinine relative to urea, hyperkalaemia, high phosphate, and low calcium. Aggressive fluid resuscitation is the single-most important treatment. The patient's limbs should be checked for evidence of potential compartment syndrome caused by crush injury.

2 The history points to AKI on a background of CKD. This case is a multifactorial combination of dehydration (diuretics), the action of ACE inhibitors and NSAIDs on the kidneys, infection, and hypotension. Unlike patients admitted with decompensated heart failure and AKI, this patient has no signs of decompensated heart failure. Treatment would consist of the AKI checklist: correct volume depletion, treat hypoperfusion (stop ACE inhibitor, NSAID, and aim for a MAP of at least 70 mmHg), exclude obstruction, stop nephrotoxins, and treat any other underlying causes (e.g. ongoing infection, and consider penicillin-induced acute interstitial nephritis if his AKI does not resolve quickly). The dose of allopurinol should be reduced in renal impairment.

3 This patient appears to be well, with normal vital signs. The combination of haemoptysis plus AKI in a well patient should make you think of a pulmonary-renal syndrome (e.g. granulomatosis with polyangiitis, formerly known as Wegener's, or anti-GBM disease, formerly known as Goodpasture's). In a sick patient, the common cause of bilateral patchy shadowing on the chest X-ray, haemoptysis, and AKI is pneumonia, although the haemoptysis is usually mixed in with sputum. Urinalysis and urine microscopy are important in this case and she should be referred to a renal specialist as soon as possible. Blood can be sent for ANCA (anti-neutrophil cytoplasmic antibodies) and anti-GBM (glomerular basement membrane) antibodies. Underlying causes of AKI can be suspected when 'classical' clinical patterns present, as illustrated in Table 7.3.

4 AKI is defined as an acute rise in serum creatinine or a urine output $\leq$0.5 mL/kg/h for 6 hours. This patient is at high risk of AKI due to oliguria, cholestasis (which causes renal vasoconstriction), sepsis, gentamicin therapy, and a previous nephrectomy – early action is essential to prevent irreversible damage to his single kidney. Persisting hypotension and oliguria despite adequate volume replacement should trigger a referral to the intensive care unit within a few hours (see Chapter 6). Urinary obstruction should be excluded by urgent ultrasound. His anti-hypertensive medication and any nephrotoxins should be stopped. (Beta-blockers in low doses tend not to lower blood pressure and ideally should not be stopped suddenly as rebound angina or fast atrial fibrillation can occur.) The underlying cause (biliary infection and obstruction) should be treated as soon as possible.

5 This is a typical combination of volume depletion in combination with infection and nephrotoxic medication which should be corrected using the AKI checklist: correct

Table 7.3 'Classical' clinical patterns in renal disease.

Symptom + impaired renal function	Consider this diagnosis
Arthralgia	Any connective tissue disease
Diarrhoea	Haemolytic uraemic syndrome
Diarrhoea in a transplant patient	Cytomegalovirus infection, mycophenolate mofetil toxicity
Haemoptysis	ANCA + vasculitis, anti-GBM disease
Hypercalcaemia and back pain	Myeloma
Long lie	Rhabdomyolysis
Microscopic haemoproteinuria in a well patient	IgA nephropathy
Miscarriages	Lupus, antiphospholipid syndrome
Peripheral oedema	Nephrotic syndrome
Sinusitis	ANCA + vasculitis
Thrombocytopenia	Thrombotic thrombocytopenic purpura (TTP)

These patterns mean the diagnosis should be *considered*, they do not confirm the diagnosis.

volume depletion, treat hypoperfusion (stop ACE inhibitor, bendroflumethiazide, and aim for a MAP of at least 70 mmHg), exclude obstruction, avoid nephrotoxins, and treat the underlying cause. Regular vital signs and fluid balance should be recorded. After correcting volume depletion and stopping her medication, the rest of the management involves ensuring adequate nutrition, monitoring her renal function, and ensuring she is followed up after discharge to ensure no progression to CKD.

6 The perioperative period can be associated with episodes of hypoperfusion because of volume depletion from many causes and hypotension due to anaesthesia. Perioperative medication may precipitate AKI, especially if the patient has predisposing risk factors: old age, diabetes, hypertension, and CKD. Use the AKI checklist: correct volume depletion, treat hypoperfusion (stop any nephrotoxic drugs and aim for a MAP of at least 70 mmHg), exclude obstruction (has she gone in to urinary retention?), and look for any other underlying causes (did she fall because of an infection?).

7 This patient was at risk of developing AKI because of pre-existing kidney disease coupled with a major cardiovascular event. A period of hypoperfusion probably precipitated AKI, but he may have also had a primary coronary angioplasty for his myocardial infarction involving the administration of contrast. As well as paying attention to A (airway) and B (breathing) in this case, management is as per the AKI checklist: treat hyperkalaemia first, correct volume depletion, treat hypoperfusion (stop any nephrotoxic drugs and aim for a MAP of at least 70 mmHg), exclude obstruction, and look for any other underlying causes. If his renal function continues to deteriorate, then renal replacement therapy may be necessary to support the patient through this acute

event. His renal function should be monitored closely with regular blood tests and a urinary catheter should be inserted for close monitoring of urine output.

8 The combination of volume depletion associated with major surgery, transient hypotension due to anaesthesia or blood loss, and cross-clamping of the aorta all put this patient at risk of developing postoperative AKI. The AKI checklist is still relevant in this situation, with the added consideration of whether and when to administer intravenous sodium bicarbonate (see mini-tutorial). Renal replacement therapy may be necessary if her creatinine rises significantly or she develops complications of AKI.

9 The priority for management in this situation is treating the patient's fluid overload. In this particular cardiorenal syndrome, the renal impairment is secondary to decompensated heart failure. His renal function may get worse with diuretic treatment; however, a further deterioration is often accepted as a 'trade-off' in this context. If he were taking 80 mg of oral furosemide twice a day at home, this could be increased by 25–50% and administered as a daily intravenous infusion, with close monitoring of his renal function. Once diuresis and weight loss are established, he can be switched to an oral twice a day regimen. Provided the patient has heart failure with a reduced ejection fraction, there is no hyperkalaemia, and his creatinine does not rise by more than 30%, any renin–angiotensin blockers do not need to be stopped.

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Airway: maintaining airway patency

Part 2

Chapters

8 Upper airway: assessment and management

Figure 8.1 The respiratory tract, showing the upper and lower airway structures. Source: Peate I. and Nair M., 2011. Reproduced with permission of John Wiley & Sons.

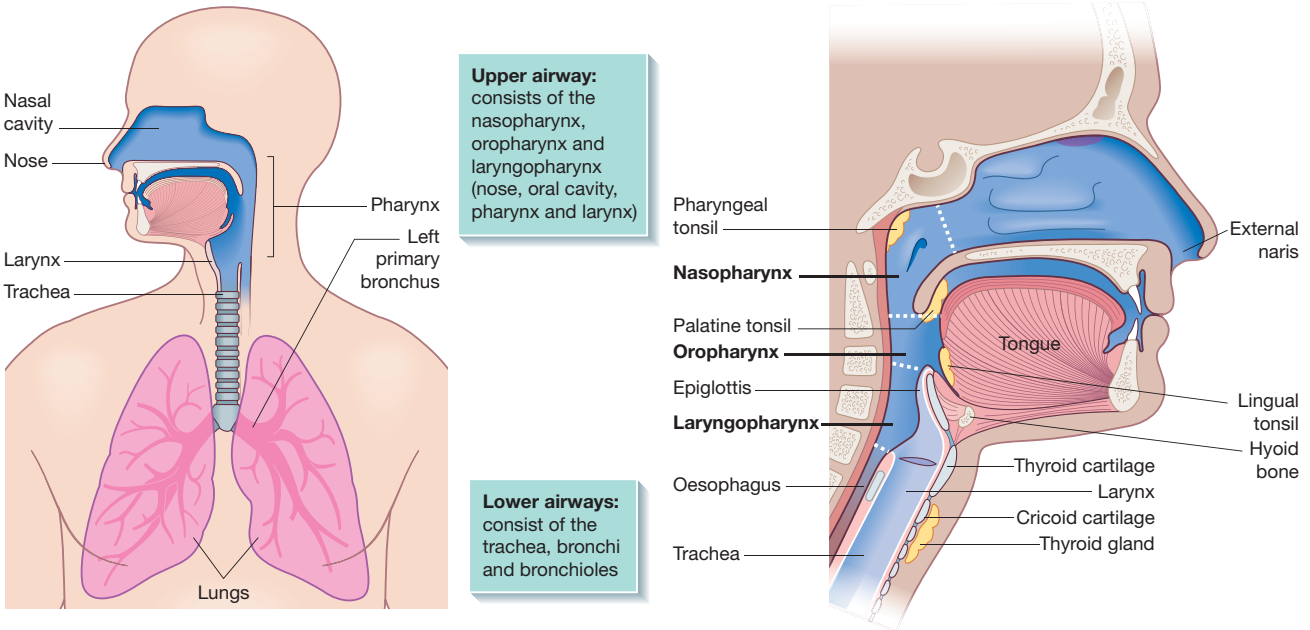


Figure 8.2 Upper airway obstruction. Source: Leach R.M., 2014. Reproduced with permission of John Wiley & Sons.

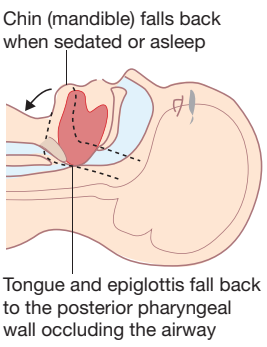


Figure 8.3 Opening the airway (a) head tilt chin lift and (b) jaw thrust

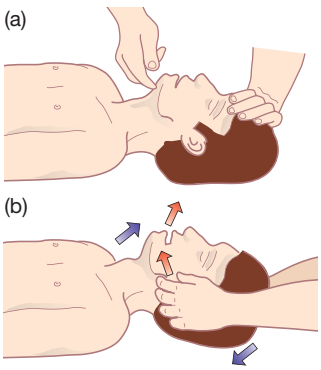


Figure 8.4 Recovery position

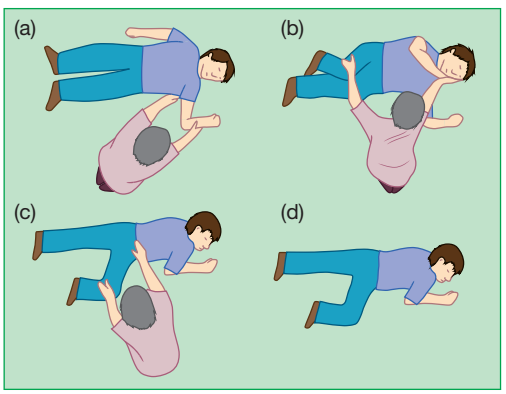


Figure 8.5 RCUK choking algorithm. Source: Resuscitation Council (UK). Reproduced with the kind permission of the Resuscitation Council (UK).

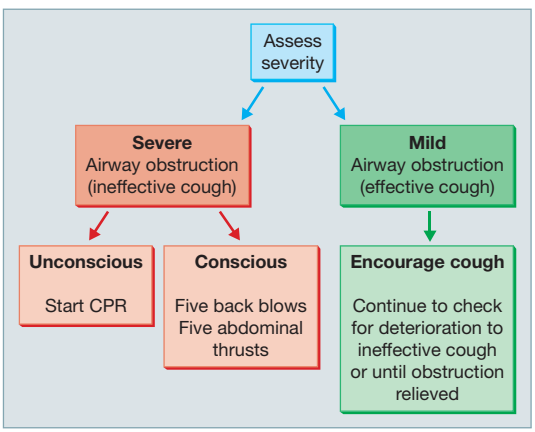
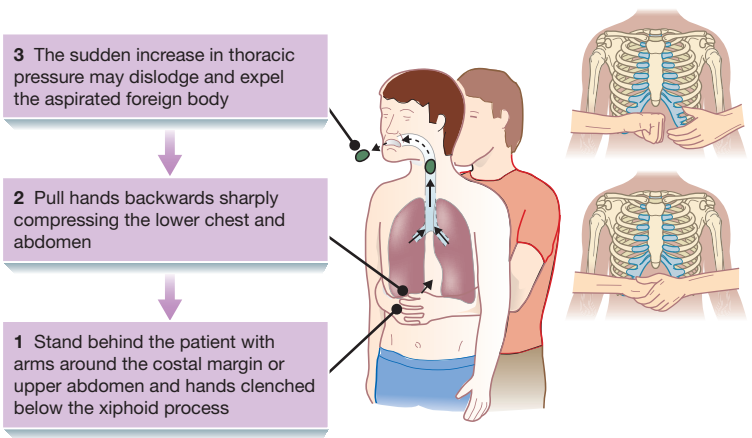


Figure 8.6 Removal of foreign body obstruction. Source: Leach R.M., 2014. Reproduced with permission of John Wiley & Sons.



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Upper airway structure and function

The upper airway is the route by which air first travels from the environment into the trachea, then through the lower airways into the lungs, in order for gaseous exchange to occur (Figure 8.1). Assessment of the upper airway is A of the ABCDE assessment and has priority as airway problems can quickly lead to deterioration, with complete airway obstruction leading to death within a few minutes. Any airway problems must be recognised and dealt with promptly by appropriately trained and competent healthcare practitioners. The upper airway (Figure 8.1) comprises the nose, oral cavity, pharynx and larynx. The primary functions of the upper airway are:

- To conduct air from the environment to the lower airways.
- To warm and humidify inspired air.
- To prevent foreign particles from entering the lower airways.

Airway assessment

If the **upper airway** becomes obstructed due to swelling or the presence of a foreign body, hypoxia, respiratory arrest and death will occur quickly unless prompt action is taken. Assessment of the airway is always the first procedure to be carried out in any acute situation. The airway may become partially obstructed or completely obstructed. Accurate assessment and recognition of the problem will determine the appropriate and effective action to take.

The best approach to airway assessment is first of all to **look** at the person: their pattern of breathing and whether they are conscious. If they are conscious, are they able to speak? Ability to speak indicates the airway is patent as air is able to pass over the vocal cords. Whilst reassuring, the ability to speak does not necessarily mean all is well, as there may be a degree of partial obstruction. Similarly, a lack of verbal response does not always indicate an obstructed airway. Take note of any abnormal skin colour and use of accessory muscles that could indicate partial obstruction. Assess further for partial obstruction and presence of normal breathing by **listening** for breath sounds.

Partial airway obstruction is ***noisy*** and could include:

- Inspiratory stridor – caused by obstruction at, or above, the larynx.
- Gurgling – suggests liquid in the upper airway.
- Snoring – the pharynx is semi-occluded by the tongue.
- Crowing or stridor – caused by laryngeal spasm or obstruction.
- Expiratory wheeze – suggests constriction or spasm of the lower airways.

Complete airway obstruction is ***silent*** and may result in paradoxical or 'see-saw' breathing, as attempts are made to draw in air: the chest is drawn in and the abdomen distends. The opposite occurs on exhalation.

Causes of airway obstruction

The airway can become obstructed either by external pressure or internal blockage of some kind, often requiring surgery. These more uncommon causes of external obstruction include:

- Swelling of the soft tissues of the neck.
- Tumours of the neck.
- Enlarged thyroid or local lymph glands.
- Physical pressure on the neck (strangulation).

It is more usual for the cause of the acute obstruction to be internal for example:

- Blockage by vomit, blood secretions or an inhaled foreign body (removed by suctioning, removal of foreign body and/or positioning).
- Swelling of the airway such as in allergic responses.
- Blockage by the tongue in the unconscious casualty.

One of the most common causes of acute airway obstruction is the patient's tongue. When unconscious, if the patient is supine the tongue will slip backwards occluding the airway (Figure 8.2) due to the fact that the muscles which hold the tongue in place are completely relaxed and the normal reflex responses which protect the airway are absent. In this situation the airway can be maintained by simple airway manoeuvres.

Maintaining the airway

With any upper airway problems, whether complete or partial obstruction, help must be summoned urgently as this is a medical emergency.

The unconscious patient

The head-tilt-chin-lift manoeuvre is the easiest to perform. Bringing the head back, and the chin up as shown (Figure 8.3a), results in the tongue moving away from the back of the throat. This action opens the airway enabling the patient to breathe spontaneously, or for rescue breaths to be performed.

By moving the lower jaw forward, the jaw thrust technique has the same effect (Figure 8.3b), but is achieved without flexing the neck. It is therefore the technique of choice in suspected neck injuries. Two people will be required to perform the jaw thrust technique and rescue breaths, whereas one person can perform rescue breaths and maintain the airway with the head-tilt-chin-lift manoeuvre.

Maintaining an open airway with these techniques is effective but needs to be applied continuously to the unconscious breathing casualty. The risk of regurgitation of stomach contents with secondary airway blockage and/or aspiration remains. Therefore, in a conscious but breathing casualty, in absence of any other injury or contraindication, the recovery position is the safest way to protect the airway (Figure 8.4). The recovery position allows maintenance of the airway whilst at the same time allowing drainage of any fluid from the mouth.

Airway obstruction due to a foreign body

The inhalation of a foreign body, causing a physical obstruction, is another acute cause of airway obstruction. With foreign body airway obstruction (FBAO) the correct response is outlined in the Resuscitation Council UK's choking algorithm (Figure 8.5).[1] This involves assessing the severity of the problem initially. If the patient is able to cough, the first action should be to encourage coughing with the person sitting or standing upright and leaning slightly forward. If the casualty is unable to cough, but is still conscious, then up to five back blows should be given. If these do not relieve the obstruction then abdominal thrusts should be performed as shown in Figure 8.6. This inward and upward motion with the fists positioned just underneath the diaphragm increases intrathoracic pressure and hopefully results in the foreign body being forcibly expelled. If at any stage the patient is unconscious then immediate help should be summoned, and if breathing also ceases, cardiopulmonary resuscitation should be commenced.

9 Advanced airway management

Figure 9.1 Oral suction using a wide bore suction device

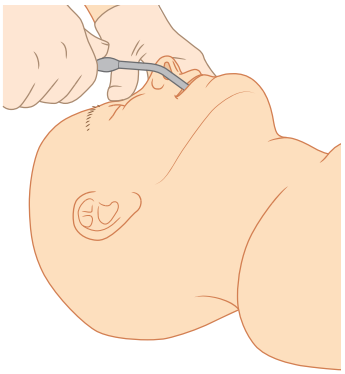


Figure 9.2 Oropharyngeal airway

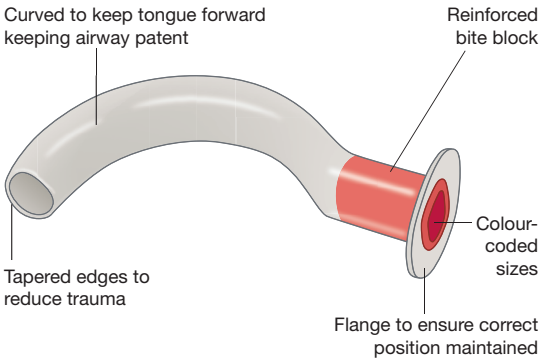


Figure 9.3 Nasopharyngeal airway

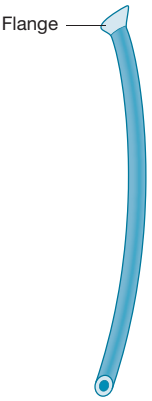


Figure 9.4 Sizing of oropharyngeal airway

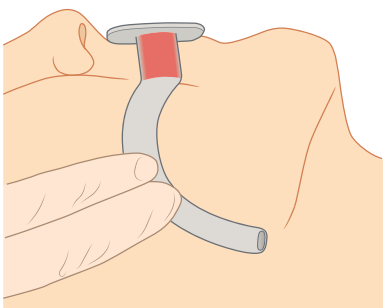


Figure 9.5 Oropharyngeal airway (OPA) insertion

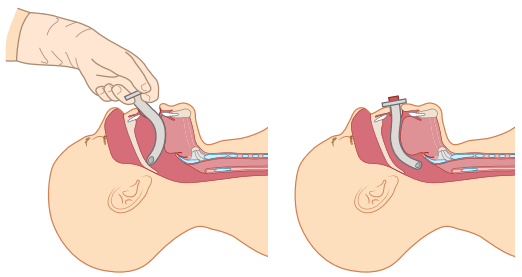


Figure 9.6 The nasopharyngeal airway (NPA) sits just above the epiglottis. Source: Leach R.M., 2014. Reproduced with permission of John Wiley & Sons.

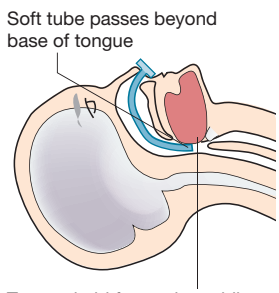


Figure 9.7 The laryngeal mask airway

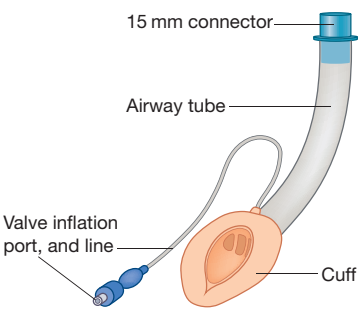


Figure 9.8 The laryngeal mask airway *in situ*

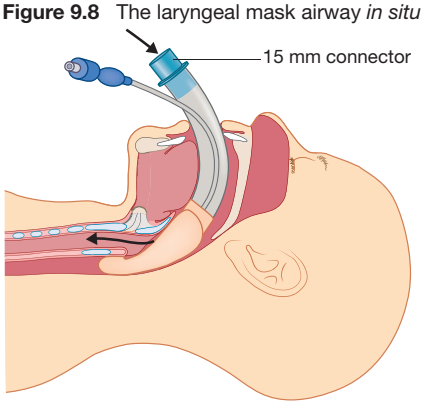
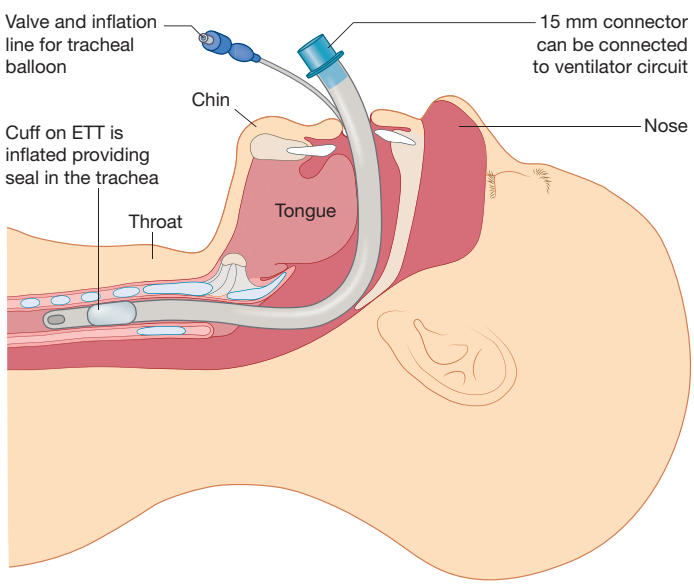


Figure 9.9 Endotracheal tube (ETT) *in situ*



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Assessment and maintenance of a safe and open airway remains the top priority in all acute situations.[1] Systematic assessment continues throughout the acute phase of deterioration and beyond, to enable recognition and prioritise management of ongoing and new problems. Once the airway is open it must be maintained effectively in order to ensure adequate ventilation for oxygenation of vital organs and tissues.

Ensuring a clear airway

If the patient is unable to maintain a clear or safe airway independently, it should be opened using the techniques described in Chapter 8. If the airway is obstructed or at risk of obstruction due to blood, vomit or secretions, gentle oral suction should be applied using a wide bore oral suction device (Figure 9.1). When carrying out this procedure do not advance the catheter into the oropharynx as this causes gagging and vomiting, further compounding the problem.

Maintaining the airway with adjuncts

Simple airway adjuncts such as the oropharyngeal airway (OPA) (Figure 9.2) and nasopharyngeal airway (NPA) (Figure 9.3) can be helpful in maintaining an open airway, but should only be inserted by healthcare staff who are trained and competent to do so. Once inserted they help maintain airway patency and are used in conjunction with the pocket mask or bag valve mask devices as an aid to ventilation as necessary.

Oropharyngeal airway

Oropharyngeal airways come in a variety of sizes from infant to adult and ensuring the correct size is important. If it is too big it can obstruct the airway or cause trauma. OPAs should only be used in an unconscious patient, as in a conscious person their insertion can stimulate the gag reflex and induce vomiting.

To ensure the correct size, the bite block should be placed at the level of the incisors and it should reach to the angle of the jaw (Figure 9.4). The airway is inserted upside down and then turned 180° once contact has been made with the back of the throat (Figure 9.5). Once *in situ* the OPA can assist with the maintenance of the airway and enables access to the oropharynx with a fine bore flexible suction catheter, to clear secretions if required.

Nasopharyngeal airway

The NPA is useful for awake or conscious patients as it does not stimulate the gag reflex. It is inserted into the nasal passageway and sits just above the epiglottis, separating the soft palate from the wall of the oropharynx and maintaining airway patency (Figure 9.6).

- Select the correct size of NPA by measuring from the patient's earlobe to the tip of the nostril.
- The nostril should be inspected for polyps prior to insertion, and if necessary the other nostril used.
- A water-based lubricant is used prior to insertion.
- Insert gently, as trauma and bleeding can occur in around 30% of insertions. If resistance is felt do not continue, try the other nostril.
- When inserted the flange should rest just below the patient's nostril.

NPAs can facilitate removal of secretions in patients who have a weak cough, as a suction catheter can be passed down into the lower airway. NPAs should not be used in the case of head trauma until the possibility of a fractured base of skull has been ruled out. **Even with an OPA or NPA *in situ* the airway can obstruct if the head is not correctly positioned.**

Artificial airways

It is sometimes necessary to insert a more long-term device if airway maintenance and management is likely to be prolonged or there is a need to overcome an airway obstruction. OPAs ensure a patent airway, but give no protection from aspiration of vomit or secretions. Aspiration is defined as the inhalation of either oropharyngeal or gastric contents into the lower airways. Gastric acid causes inflammation of the lung tissue or pneumonitis. Bacteria aspirated from the oropharynx cause a bacterial pneumonia. Both of these lung problems are associated with significant mortality. The insertion of an artificial airway ensures a secure airway and protection from aspiration. Artificial airways enable tracheobronchial suction, removal of secretions and artificial ventilation.

Laryngeal mask airway

The laryngeal mask airway (LMA) is a supraglottic airway device (Figure 9.7). This means it is placed above the level of the **glottis** (Figure 9.8). It is often used in the emergency setting to enable establishment of a secure airway relatively quickly and easily. It is also used for more prolonged airway management, in addition to tracheal suction to remove secretions, and effective ventilation. The LMA consists of a large tube with an elliptical mask on the distal end. This mask's inflatable cuff covers the tracheal opening covering the supraglottic structures and allows isolation of the trachea from the oesophagus, reducing risk of aspiration. There are a number of benefits of LMAs:

- They are relatively easy to use and quick to place, even for the less experienced clinician. Despite this they should only be inserted by healthcare staff trained and competent in their use.
- LMA use results in less gastric distension when used in conjunction with bag-valve-mask as opposed to a facemask for ventilation.
- Due to the isolation of the trachea, the risk of aspiration is significantly reduced although not entirely eliminated.

LMAs are used in patients who are unconscious or heavily sedated.

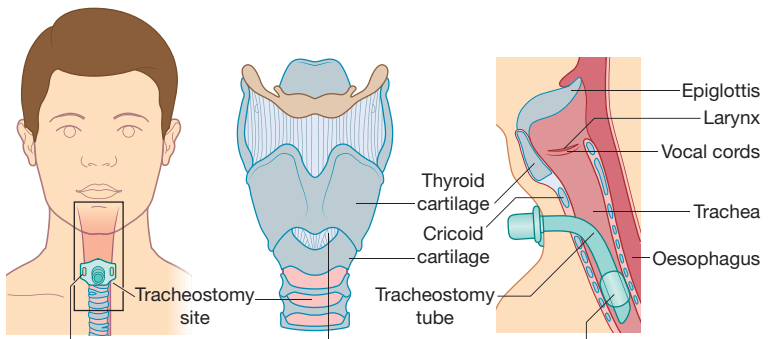
Endotracheal tube intubation

The placement of an **endotracheal tube** (ETT) is considered the 'gold standard' in terms of establishing a secure artificial airway (Figure 9.9). The inflated cuff on the ETT provides a seal to prevent gastric or oropharyngeal contents entering the lung, so the airway is kept patent and protected from the risk of aspiration. Endotracheal suction removes secretions. ETTs may be inserted nasally or orally. The oral route is common in an acute situation but may be contraindicated in situations of oral trauma. Nasal intubation is used when required over a longer period of time, as it enables effective oral hygiene to be carried out. Intubation with an ETT enables positive pressure ventilation to be established. Intubated patients will be cared for in an ICU by staff who are competent in caring for ventilated patients.

Insertion of an ETT is highly skilled and should only be attempted by those trained and experienced in the technique. Both LMAs and ETTs are used in patients who are unconscious or heavily sedated. Therefore, the long-term management of both requires specialist skills and knowledge to ensure patient safety.

10 Airway management: tracheostomy

Figure 10.1 Location of tracheostomy site, tube and anatomical landmarks



Box 10.1 Common reasons for a tracheostomy

- Airway maintenance
- Airway protection
- To enable suctioning of secretions
- Weaning from ventilation

Figure 10.2 Cuffed tube

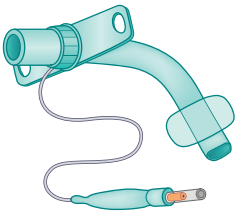


Figure 10.3 Fenestrated tube with inner cannula. Inner tube (cannula) requires cleaning 4-hourly

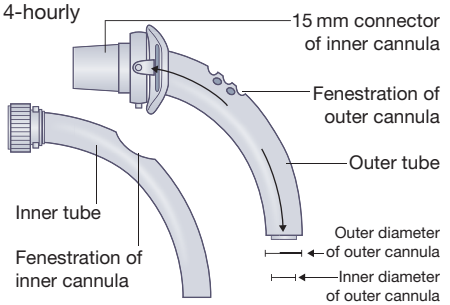
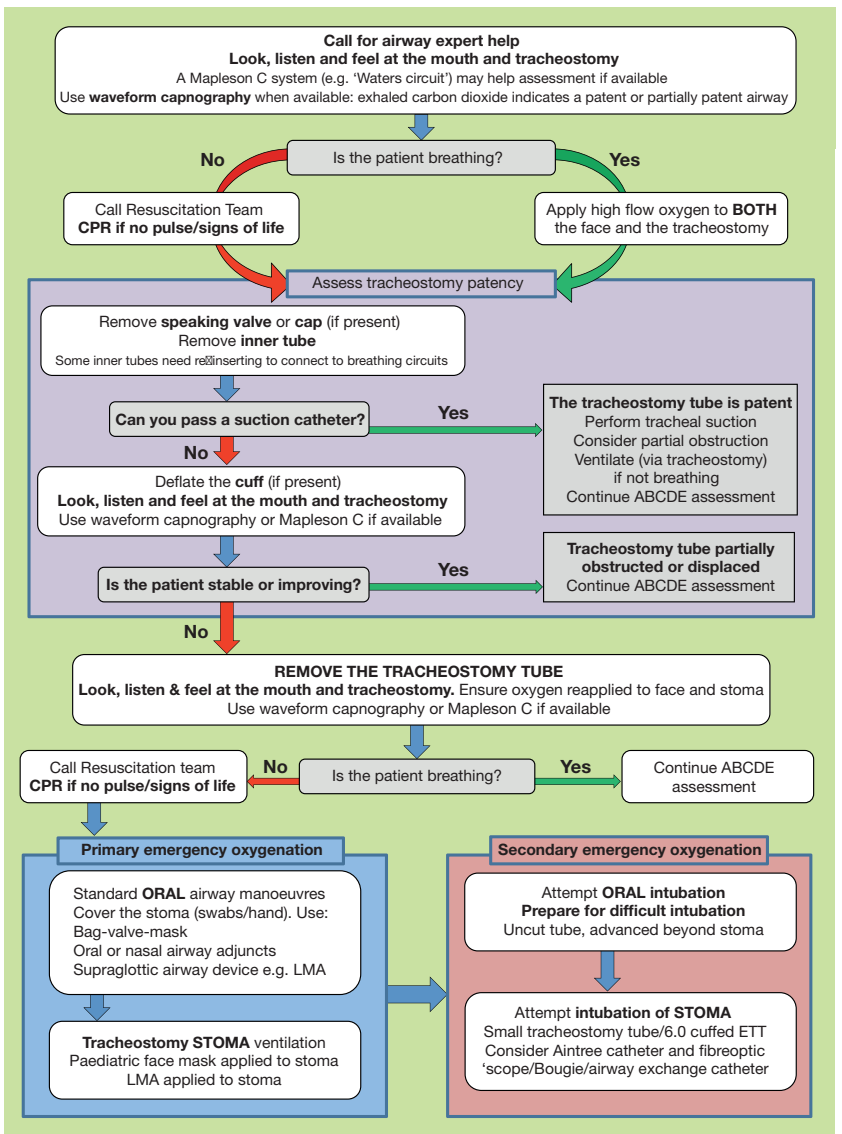


Figure 10.4 Emergency tracheostomy management – patent upper airway.
Source: McGrath B.A., *et al*., 2012. Reproduced with permission from Blackwell Publishing Ltd.



Box 10.2 Bedside equipment for all tracheostomy patients (and for patient transfer). Source: Intensive Care Society, 2014.

- Suction unit, Yankauer sucker and suction catheters
- Tracheostomy disconnection wedge
- Non-powdered latex free gloves, aprons and eye protection
- Rebreathe bag and tubing, catheter mount or connector
- Spare tracheostomy tubes, same size and size smaller
- 10 mL syringe if tube cuffed
- Tracheal dilators
- Resuscitation equipment

Ongoing care equipment

- Humidification equipment
- Scissors if tube sutured
- Call bell
- Clean pot for spare cannula
- Water-soluble lubricating jelly
- Communication aids
- Sterile water for cleaning suction tube
- Sterile dressing pack
- Bedside equipment checklist

Hints and tips: tracheostomy wound care

- Secretions that collect above the cuff ooze out of the stoma site leading to possible excoriation and infection. The site should be assessed an stoma cleaned once a day using a clean technique
- Red, excoriated or exuding stomas should have swabs sent for culture

Red flag events

- Pain at tracheostomy site
- Visibly displaced tube
- Bleeding
- Suction catheter not passing easily into trachea
- Cuffed tracheostomy patient being able to talk, or bubbles coming from upper airways
- Frequent need to reinflate cuff to prevent air leaks
- Respiratory distress, difficulty in breathing
- Surgical emphysema (air in the soft tissues)
- Aspirating feed from trachea (cuff not functioning)

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A tracheostomy is a surgical procedure to create an artificial opening (stoma) in the anterior wall of the trachea, just below the cricoid cartilage. A small and curved tracheostomy tube is placed into the trachea, via the newly created stoma, sitting just above the level of the **carina** (Figure 10.1). Up to 15 000 tracheostomies are performed each year in England with common indications given in Box 10.1. Most patients have their tracheal tubes removed (known as decannulation) prior to discharge from ITU/HDU to the ward environment. However, this is not always possible. The Intensive Care Society (2014)[1], informed by the National Confidential Enquiry into Patient Outcome and Death (NCEPOD) (2014)[2], published standards and guidelines for the care of adult patients with a temporary tracheostomy, to inform safe practice in any care setting.

Tracheostomies

The majority of tracheostomies will be planned, using a variety of approaches, depending on the reasons for tracheostomy formation:

- A **surgical tracheostomy** is performed in a theatre environment by a surgeon for patients with difficult anatomy who require permanent or long-term airway protection. This type of tracheostomy can be permanent or temporary.
- A **percutaneous tracheostomy** is temporary. This procedure is usually performed in critical care by an anaesthetist. A small hole is made and dilated using specialised equipment, until the tracheostomy tube can be inserted. Usually the site heals more quickly and with less scarring than surgical tracheostomy.

A **cricothyroid/mini-tracheostomy** is formed in the relatively avascular cricothyroid membrane (Figure 10.1). A small diameter tube, which is uncuffed, is inserted using a guidewire and dilator. A mini tracheal tube is sufficient to remove excessive bronchial secretions with a narrow 10F suction catheter, thereby relieving sputum retention.

Types of tracheostomy tubes

Staff caring for a patient with a tracheostomy **must know the type of tube in place** and this should be **clearly documented in the patient's notes** and care plan. **Cuffed tracheostomy tubes** (Figure 10.2), when inflated, provide a seal in the trachea to protect the lungs. Air does not pass through the upper airway, but through the tube. An **uncuffed tracheostomy tube** allows air to move through and around the tracheostomy tube. **Fenestrated tracheostomy tubes** have holes in the outer cannula (Figure 10.3) allowing air to pass through vocal cords in the upper airway, and the patient to speak. Most tracheostomy tubes will have an inner tube that is regularly removed for cleaning (Figure 10.3).

Tracheostomy tube: care issues

- **Emergency protocols must be in place to maintain a patent upper airway: nurses must be familiar and competent with guidelines (Figure 10.4)** in the case of a blocked upper airway or tracheostomy tube.[3]
- **Essential tracheostomy equipment** must be at the patient's bedside at all times (Box 10.2).
- **Humidification is essential** for patients with a temporary tracheostomy, using regular nebulisers, humidifier systems or heat moisture exchanger (HME). Inadequate humidification may lead to **life-threatening blockage** of **the tracheostomy tube**.
- **Inner cannulae** must be regularly changed and cleaned 4-hourly (Figure 10.3) according to trust guidance, to maintain patency.
- **Cuff pressure** should not exceed 25 cm H_2O, as above this permanent damage to the trachea can occur. An air leak may be heard as a squeaking sound. If this occurs with the cuff pressure at 25 cm H_2O, the tube may have become displaced or require changing, and immediate expert review is required.
- **Communication**. Following a tracheostomy procedure, the patient will be temporarily unable to speak. Communication boards and signing can be frustrating and tiring, so time and encouragement is required. Verbal communication is possible with a speaking valve placed on either an uncuffed tube, or a fenestrated tube with the cuff down. It is essential that the **cuff is deflated prior to speaking valve placement** as failure to do so will cause airway occlusion. If the patient exhibits **signs of respiratory distress** or they are unable to vocalise, the **speaking valve must be removed immediately** and the cause of the problem identified.
- **Regular suction** removes secretions which can be aspirated from the end of the tube in shallow suctioning. Deeper tube suction requires an appropriately trained and competent practitioner.
- The **tracheostomy site** must be cleaned and dressed daily (see hints and tips box).
- **Tracheostomy tube change** must occur as necessary as a planned procedure; those with inner tubes can remain for a maximum of 30 days. Only **staff trained and competent to change tracheostomy tubes** should do so. Nurses must adhere to trust policies and procedures when carrying out any clinical procedures.

Cuff deflation

An inflated cuff compresses the oesophagus making swallowing difficult. The decision to allow feeding with a deflated cuff should be made after a swallowing assessment, with the patient monitored for any evidence of aspiration. Before cuff deflation, the patient is warned about the alterations in tracheal airflow sensation and that they may need to cough. If coughing persists, and is not resolved with suction and reassurance, the cuff must be re-inflated. On cuff deflation the tracheostomy tube should be occluded briefly with a clean, gloved finger to check that air is flowing around the tube.

Tracheostomy red flags

Red flags are used as a warning signal that a problem needs to be urgently dealt with (see red flag box). All staff caring for patients with a tracheostomy need to be aware of these signs.

Weaning

Weaning is a planned process completed under the instruction of a clinical expert, involving increasing the periods of time the cuff is deflated. A **decannulation cap** is placed on a fenestrated tracheostomy tube with deflated cuff, effectively blocking the tracheostomy tube opening. The decannulation cap should only to be used with a fenestrated inner tube in place. The decision to remove the tube is made by the multidisciplinary team, with one person having overall responsibility. This is very important for ward-based patients who may not always be under the direct care of the critical care team.

The normal healing process: acute wounds

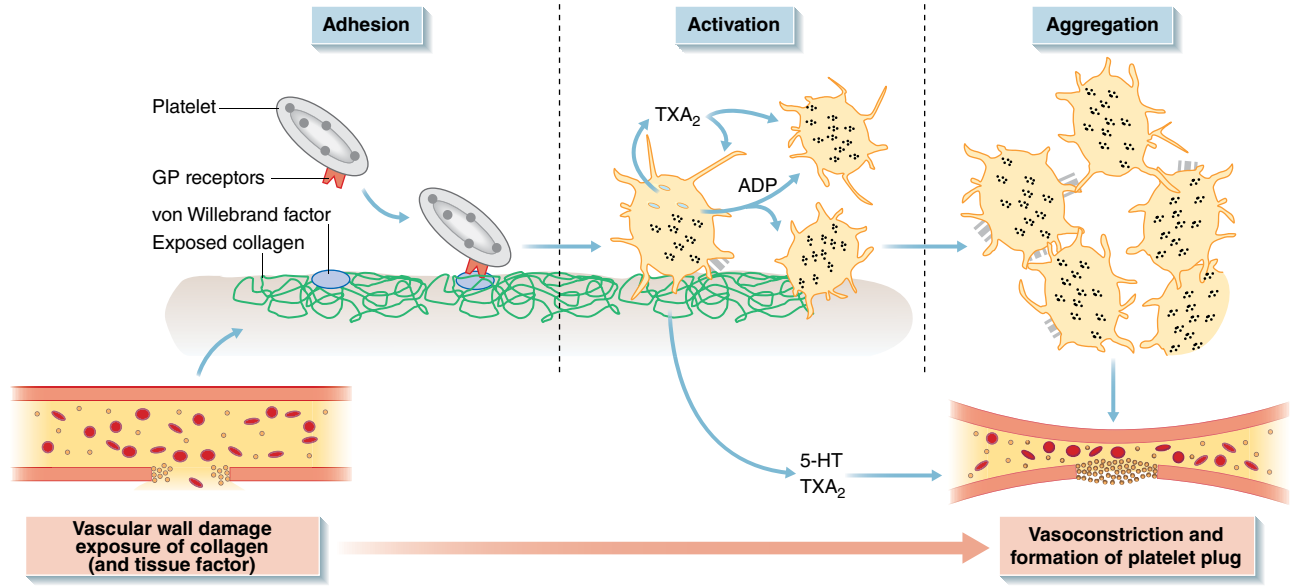
Part 2

Chapters

Visit the companion website at **www.ataglanceseries.com/nursing/woundcare** to test yourself on these topics.

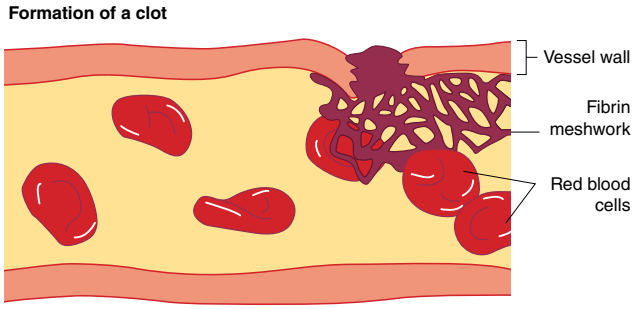
6 Haemostasis

Figure 6.1 A platelet plug.



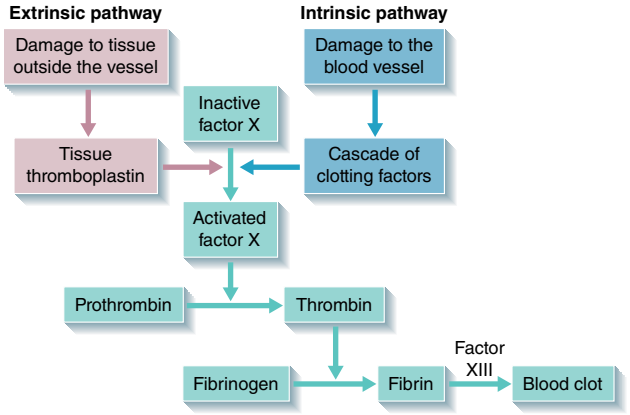
Source: Ward and Linden 2013, figure (a), Chapter 9, p. 28. Reproduced with permission of Wiley & Sons, Ltd.

Figure 6.2 Fibrin meshwork.



Source: Nair and Peate 2013, Figure 6.16, p. 167. Reproduced with permisson of Wiley & Sons, Ltd.

Figure 6.3 Extrinsic and intrinsic pathways.



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Physiology of haemostasis

The haemostatic mechanism is complex and delicately balanced. Haemostasis is an aspect of the wound-healing process. The wound-healing process passes through a number of phases, including inflammation, granulation and maturation.

Haemostasis is a process that causes bleeding to stop, keeping blood within a damaged blood vessel; the opposite of haemostasis is haemorrhage, and it is the first stage of wound-healing.

The body's initial response to wounding is to control the loss of blood from the area. Following damage to blood vessels and endothelial cells, platelets become sticky and adhere to the wall of the blood vessel and to each other, forming a platelet thrombus. The platelet thrombus acts as a temporary plug that reduces blood flow out of the wound (see Figure 6.1). The platelets also release serotonin and other chemical mediators, which results in a short period of vasoconstriction. Haemostasis is also achieved by the activation of the clotting cascade, initiated by damage to the endothelium.

Platelet function

Platelet function is essential in haemostasis. Platelets are vesicle-like fragments; they are incomplete cells, approximately 2–4 μm in diameter, discoid in shape and arise from larger cells, the megakaryocytes, in the bone marrow. The normal platelet count is approximately 150–400 million per millilitre of blood.

Five events occur when there is injury to a blood vessel:

1 Local vasoconstriction
2 Adhesion and aggregation of platelets
3 Activation of the clotting cascade
4 Activation of coagulation inhibitors
5 Fibrinolysis.

When damage to a blood vessel occurs, constriction occurs through direct action and also indirectly through the release of vasoconstrictors from platelets; these actions play a central role in limiting blood loss. Damage to the endothelial lining of the blood vessel also triggers platelet activity; when this happens, the platelets aggregate forming a plug. These actions along with the transient vasoconstriction are generally responsible for the cessation of bleeding. Platelets, when activated, also liberate a number of vasoconstrictor substances, revealing a phospholipid that is essential for the creation of a blood clot.

Generally, platelets do not adhere to the smooth endothelial lining of the blood vessels. However, when the vasculature is damaged, this exposes the blood to subendothelial collagen and microfibrils. The platelets stick to the collagen in the damaged vessel via glycoproteins (GPs) (von Willebrand factor enhances the adhesion) that are located on the surface of the platelets. Activation results in the platelets changing shape along with the production of pseudopodia (these are temporary protrusions), producing thromboxane A_2 (TXA_2) – an enzyme (a lipid with prothrombotic properties) – along with the release of 5-hydroxytryptamine (5-HT or serotonin) and ADP (adenosine diphosphate). Further vasoconstriction occurs caused by TXA_2 and 5-HT; ADP recruits more platelets and they aggregate to each other cross-linking with fibrinogen. A soft plug forms that is held together by fibrinogen molecules that create bridges between adjacent platelets. The aggregated platelets occlude the wound, which eventually stops bleeding. This is an unstable primary plug, a loosely aggregated plug, and has to be consolidated into a more stable plug.

Clotting cascade

The clotting cascade provides this more stable consolidated plug and ultimately results in the conversion of the plasma protein fibrinogen to fibrin, which forms a meshwork providing a seal to the damaged vasculature (see Figure 6.2). The cascade is a sequence of interactions between proteins that cause fibrin depositions at the location of tissue injury and is initiated by its interaction with activated factor VII. See Figure 6.3 for the extrinsic and intrinsic pathways.

The initial phase (this used to be known as the extrinsic pathway) involves several factors. Tissue factors VII and VIIa convert factor X to its active form, factor Xa. Tissue factor VIIa also converts factor IX to its activated form, factor IXa; further generation of factor Xa is inhibited by the tissue factor pathway inhibitor. At this point, the amount of factor Xa produced is insufficient to sustain coagulation. Further, factor Xa to allow haemostasis from progress to completion can now only be generated by the factor IXa pathway; by this stage though, enough thrombin has been generated by factor Xa to activate factors VIII and V. Both of these act as potent catalysts. Factor VIIIa increases the capacity of factor IXa to activate factor X to Xa many thousand times. The increased factor Xa produced in this way along with its own cofactor, activated Va, forms a complex that promotes the efficient conversion of prothrombin to thrombin. Thrombin then allows the conversion of fibrinogen to fibrin. The final result of the cascade is the production of fibrin, the biological glue that eventually seals the haemostatic plug ensuring haemostasis. As soon as a small amount of thrombin is formed, the clotting process accelerates and provides more and more thrombin into the wound. At high concentrations, this thrombin quickly converts fibrinogen to fibrin on the surface of the platelet aggregate to stabilise the haemostatic plug. Over the course of the subsequent 7–12 days, the process of fibrinolysis dissolves the fibrin in the wound, as the site of injury heals and the cell layer in the vessel wall is restored. Scar formation occurs, and the wound is completely healed within a few weeks.

Wound healing

Wound healing is a complex and dynamic process that varies according to the location and type of wound and occurs as a systemic process, which follows in stepwise fashion and involves the stages of haemostasis, inflammation and repair. Generally, from injury to resolution, wounds go through four phases:

1 Haemostasis
2 Inflammation
3 Proliferation
4 Remodelling.

As the fibrin formation occurs, a protective wound scab is formed. Scab formation provides a surface beneath which cell migration and movement of the wound edges can follow. The inflammatory process brings nutrients to the area of the wound, removes debris and bacteria and makes chemical stimuli available to start the wound repair. Repair begins instantaneously after wounding and proceeds quickly through the processes of epithelialisation, fibroplasia and capillary proliferation into the healing area. Different tissues possess their own normal rates of growth as the healing process occurs. The ideal rate of healing happens when there are factors present that are advantageous to healing, and the factors that have the ability to disturb or hinder the healing processes are controlled or absent.

7 Inflammation

Figure 7.1 Causes of the inflammatory response.

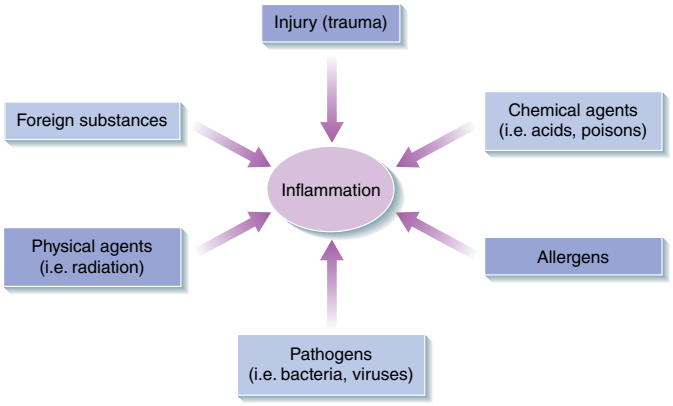
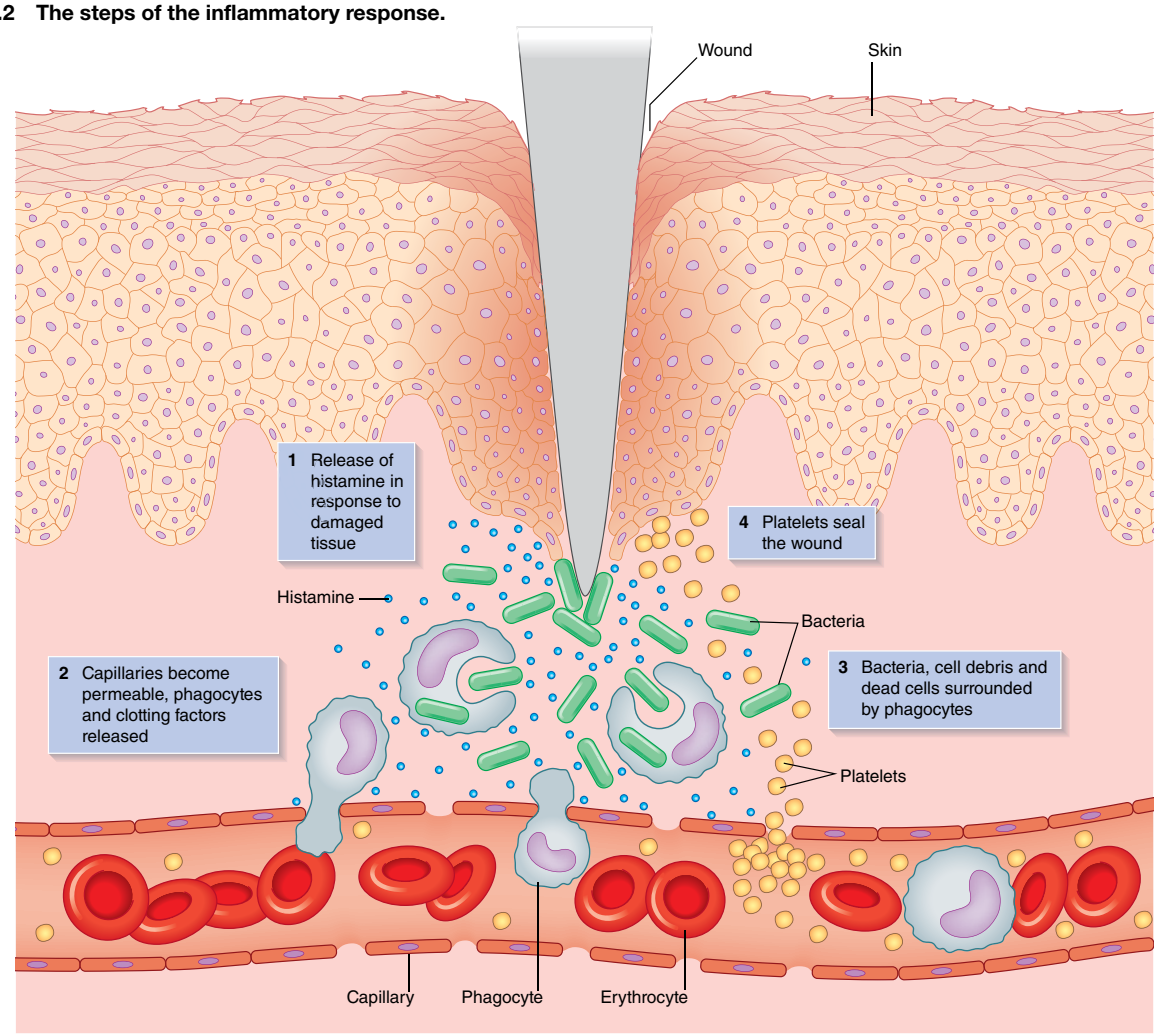


Figure 7.2 The steps of the inflammatory response.



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Inflammation

When there is an insult to the body, for example, a trauma or any intentional injury resulting in damage to the blood vessels, the first response is to arrest the haemorrhage. The prevention of blood loss and the formation of clots and scabs are the parts of the haemostatic process in an attempt to provide a protective covering when the skin is broken.

The body has effective mechanisms to block potential pathogens from entering the body, including the skin and mucous membranes as physical barriers. Enzymes, such as lysozymes in tears and sweat, have the capacity to destroy many potential pathogens chemically; however, even with these external barriers available to fight potential pathogens, there are occasions when pathogens enter the body. When this occurs and infection is present, the body sets the inflammatory response into action. This response is non-specific and attacks any and all foreign invaders. This inflammatory response is a universal reaction to tissue damage. See Figure 7.1 for the causes of the inflammatory response.

The inflammation attempts to rid the body of microbes, toxins or other foreign material at the site of injury with the intention of preventing their spread to other tissues; it also begins to prepare the site for tissue repair.

The inflammatory response

Inflammation is an innate response to tissue damage, with four phases (or characteristic signs) associated with the inflammatory response:

1. Redness (rubor)
2. Swelling (tumour)
3. Heat (calor)
4. Pain (dolor).

Loss of function is the fifth phase added to the original four. Inflammation can result in a loss of function; for example, the inability to detect sensation depending on the site and extent of the injury.

In case of an injury, pathogens such as bacteria, virus or fungus gain entry into the body. Almost immediately, a number of events are caused by the damaged cells, such as:

1. Vasodilation
2. Messenger molecules are released
3. Activation of complement
4. Extravasation of vascular components
5. Phagocytosis
6. Pain.

The injured mast cells in the connective tissue release histamine. The arrival of histamine at the site of injury has an immediate impact on blood vessels in the region. Arterioles dilate and venules constrict, causing an increase in blood flow. Vasodilation is brought about by four main mechanisms.

1. The kinin system in the cell produces bradykinin, a vasodilator, which is also responsible for pain.
2. Damaged plasma membranes release arachidonic acid, a fatty acid and a precursor to prostaglandins. Prostaglandins are also vasodilators and have hyperalgesic properties (they increase pain).
3. Release of histamine from the degranulated mast cells increases the pore size between the capillary cells, permitting movement of proteins and other micromolecules into interstitial spaces.
4. Vascular epithelial cells release nitric oxide, a vasodilator. Macrophages also release large amounts of nitric oxide.

Increased blood flow explains the redness and heat associated with the infection. Capillaries within injured tissue dilate, becoming permeable; this is essential for an appropriate inflammatory response, providing an opportunity for some of the blood components to be released into the damaged area and the site of infection (Figure 7.2).

Platelets and clotting factors

Platelets and their associated clotting factors (see Chapter 6) (for example, thrombin and fibrinogen) exit through the leaky capillary walls and migrate towards the site of injury. The clotting factors serve a dual purpose when this occurs; they help to plug the wound and seal damaged blood vessels; they also confine infectious agents to the wound site slowing their systemic spread.

Chemokines

A second line of defence also becomes activated. Cells close to the injury release a series of chemical signals radiating from the site of inflammation; these signals are known as chemokines. A very high concentration of chemokines immediately surrounds the infection. High levels of chemokines attract or provide a signal for the attraction of phagocytic white blood cells including the neutrophils.

Phagocytosis

As the chemokine concentration continues to increase, phagocytes leave the capillary entering the site of infection and macrophages arrive 24 hours later. Phagocytes engulf and destroy the present pathogens, recognising the pathogen as a non-self-matter and sending out the pseudopodia surrounding the pathogen. The wound site begins to heal. After the white blood cells engulf the pathogen particles, they begin to die; they eventually form the pus associated with infected cuts. The key molecule released is interleukin 1, attracting neutrophils and macrophages to the site of injury and helping to clear away debris from the injured area.

Phagocytosis results in a metabolically intensive activity and is responsible for some of the heat associated with inflammation. Pyrexia occurs during infection accompanying the inflammation. Bacterial toxins elevate the body temperature, releasing cytokines from macrophages causing the increase in temperature. The presence of pyrexia exaggerates the impact of interferons, hindering the growth of some microbes speeding up the reactions that aid repair.

When the pathogen particles are destroyed and the damaged tissue is repaired, histamine signals fade and blood vessels return to their normal size.

The sensation of pain in inflammation is increased by the action and interaction of bradykinin and prostaglandins. Other chemicals are also involved in the stimulation of pain after the injury; lactic acid produced by anaerobic cellular respiration is one example. Hydrogen ions and potassium released from damaged cells also stimulate the pain receptors.

Inflammation lasts for approximately 4–5 days. The process requires energy and nutritional resources for efficacy. With large complex wounds, demands made on the body are considerable. Inflammation has a protective function helping to eliminate the cause(s) of tissue damage. Extending the inflammatory stage – for example, where there is infection, the presence of a foreign body or damage that has been caused by an inappropriate dressing – can have an adverse effect on the person's health and well-being.

8 Proliferation (granulation and epithelialisation)

Figure 8.1 Wound-healing time scales.

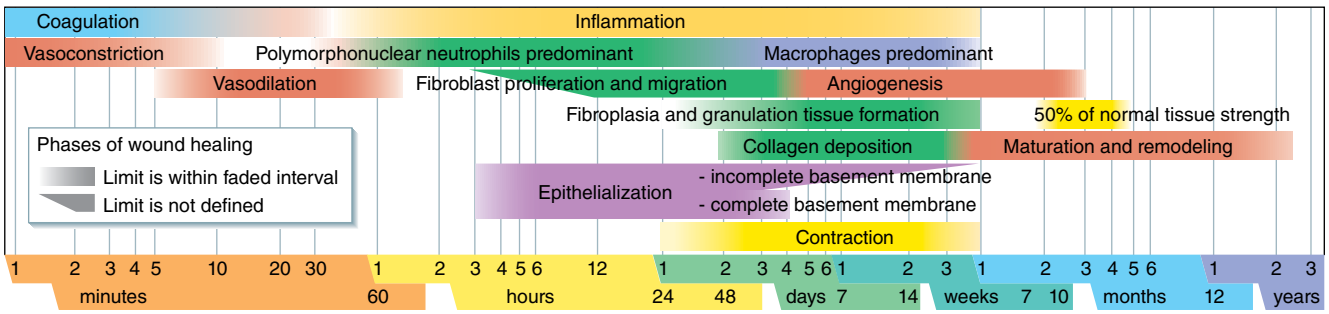
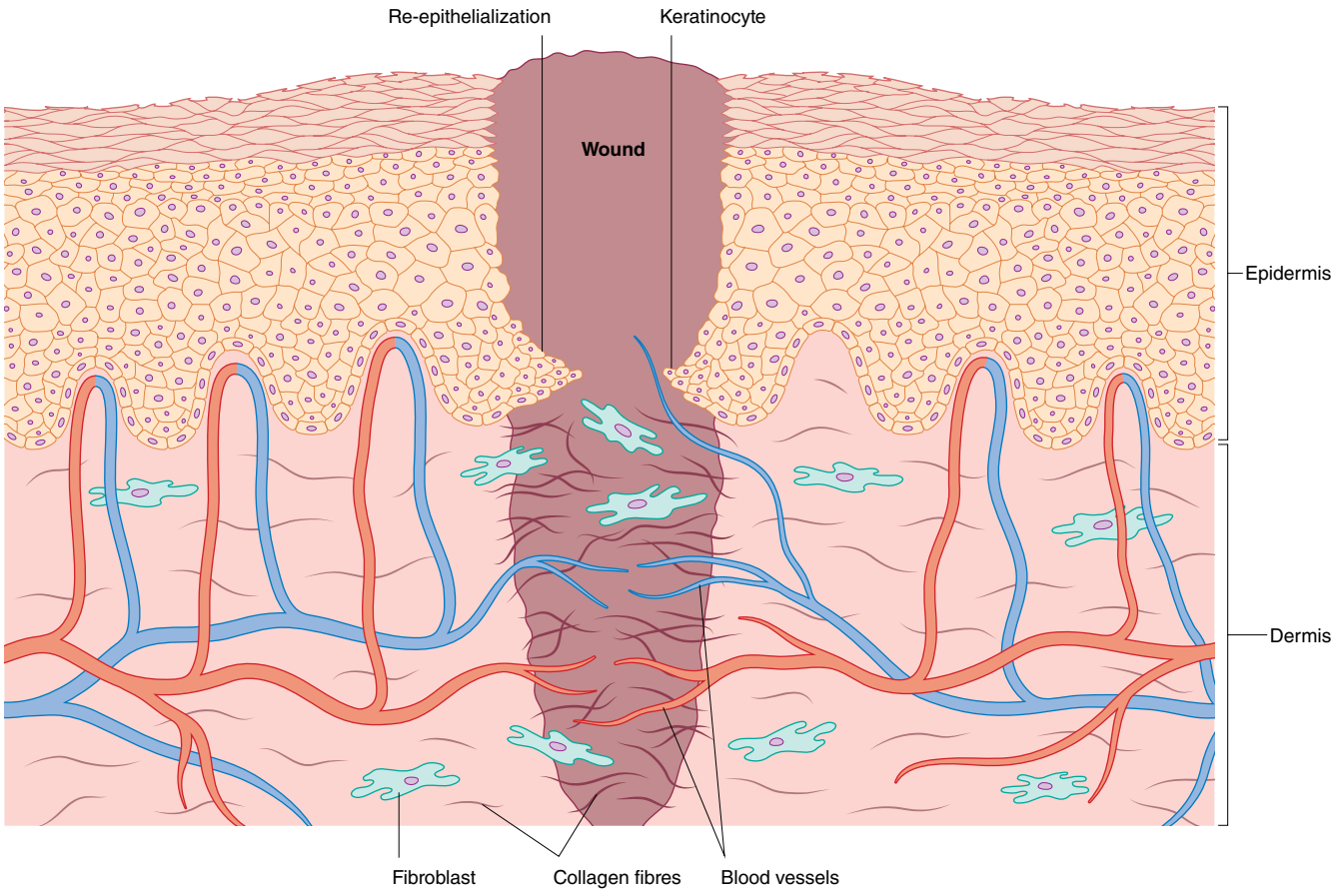


Figure 8.2 Proliferative phase of wound-healing showing fibroblasts producing extracellular matrix and re-epithelialisation by keratinocytes.



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Wound-healing consists of filling the gap created by tissue destruction followed by restoration of the structural continuity of the injured part through three phases of healing (Figure 8.1); they are:

1 The inflammatory phase
2 The proliferative phase
3 The remodelling phase.

Proliferation

The proliferation phase overlaps with the inflammation stage (see Chapter 7), as this phase starts to end. The focus of the proliferative phase is associated with the building of new tissue to fill the wound space. As the inflammation diminishes, the process to repair the injury starts. About 3 days after the injury, fibroblasts start to enter and assemble in the wound; this is the start of the transition from inflammatory phase to proliferation phase. The fibroblasts are the connective tissue cells that synthesise and secrete collagen. The secretion of growth factors that induce the growth of blood vessels starts through the process of angiogenesis along with promoting endothelial cell proliferation and migration. As the fibroblasts grow and form, they produce a new, provisional extracellular matrix that comes about by excreting collagen and fibronectin.

The fibroblasts and endothelial cells form granulation tissue that acts as the foundation for scar tissue development (Figure 8.2).

Granulation

Granulation tissue contains newly developed capillary buds. Granulation tissue can be seen in the wound around the end of the first week; this tissue continues to grow until the wound is healed. This tissue is rich in new blood vessels and other components that are required to fill in the injured tissue. Granulation tissue is usually bright red or pink, moist, soft in touch and has a bumpy appearance, it is fragile and bleeds easily. The masses within the injured tissue keep growing and contracting depending on the wound type; this takes approximately 8 weeks for a standard open healing excision wound and 4 weeks for a closed (sutured) wound.

Around day 5 post injury, exudate appears in the wound (this is the by-product of healing and is a sticky greenish-white substance resembling pus, but is not). If too much exudate is produced, wound healing may be slowed; increased exudate may be an indication of infection and increased oedema.

Epithelialisation, maturation and remodelling

The final feature of the proliferative stage is epithelialisation; this is the regeneration, migration, proliferation and differentiation of epithelial cells at the wound's edge forming a new surface area similar to that destroyed by the injury. Just after the injury, cytokines are released from platelets and they activate keratinocytes. As the migration of keratinocytes occurs, re-epithelialisation begins as early as 2 hours after wounding. Growth factors, such as keratinocytes growth factor (KGF) and epidermal growth factor (EGF), provoke the proliferation and migration of keratinocytes. The main sources of migrating keratinocytes during re-epithelialisation process are basal keratinocytes from the wound edges, dermal appendages, such as hair follicles, sweat and sebaceous glands and bone marrow–derived keratinocyte stem cells. Keratinocytes secrete proteases and plasminogen activator that activates plasmin. Migration of keratinocytes over the wound site is also enhanced by the lack of contact inhibition and the release of nitric oxide from polymorphonuclear leucocytes (PMNs), keratinocytes and fibroblasts. Epithelial cells continue migrating across the wound bed until cells from different sides meet in the middle; at this point, the contact between keratinocytes inhibits further migration. New layers of keratinocytes differentiate, and this gives rise to a stratified epidermis.

As the proliferative stage ends, white bloods cells leave the area and oedema diminishes; the wound begins to blanch as the small blood vessels begin to thrombose and degenerate.

The maturation and remodelling phases overlap with the proliferation phase, as the healing begins to come to an end. The remodelling phase begins after about 3 weeks and can continue for 6 months or longer. Final scar tissue starts to form by the simultaneous synthesis of lysis and collagen. Collagen is fibrous in character, and connects and supports tissues and organs, such as skin, bone, tendons, muscles and cartilage. It is often referred to as the glue that holds the body together; it is collagen that provides tensile strength. There are over 25 types of collagen that occur naturally in the body; collagen can be found both inside and outside cells, contributing to the structure of cells.

At this stage, the process of remodelling of the collagen fibres is laid down. The scar becomes avascular.

The wound is made smaller by the action of myofibroblasts, as the edges of the wound are drawn closer together. This establishes a grip on the edges of the wound, causing them to contract using a mechanism that is similar to that in smooth muscle cells. When the role of myofibroblasts is close to completion, cells that are no longer needed undergo apoptosis. Myofibroblastic activity can persist, contributing to fibrosis and scarring in the skin.

Nerve endings now start to redevelop, and the tissue starts to rearrange itself. The scar tissue may achieve 70–80% of tensile strength by the end of 3 months. Within the tissue, there is much physiological activity left even after the surface wound-healing. This final phase continues for up to 18 months after the wound is closed.

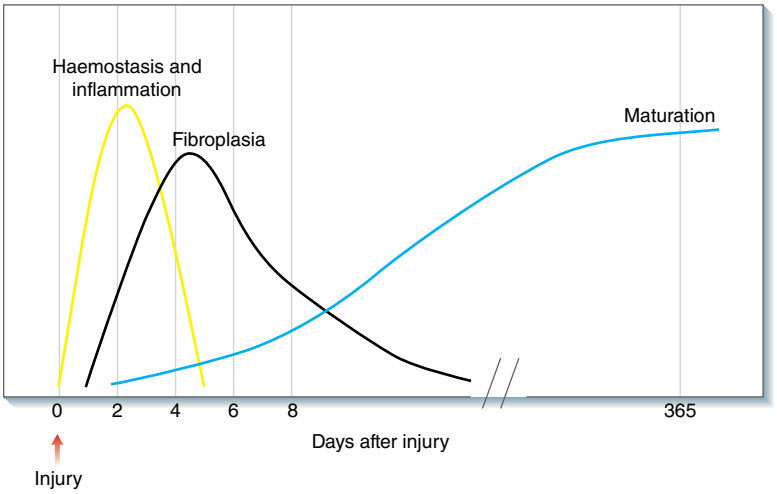
Rapid keratinocyte migration along with re-epithelialisation can often lead to better wound-healing outcomes and decreased scar formation. However, exposure to air and/or lack of moisture will result in a delay in the healing process.

This whole process is complex, and the skin is fragile and prone to interruption or failure. The outcome of this is the formation of non-healing chronic wounds. There are factors that may contribute to the interruption or failure, such as:

- Diabetes
- Venous or arterial disease
- Infection
- Ageing.

9 Maturation

Figure 9.1 The phases of wound-healing.



Box 9.1 Wound–healing.

- The stages of wound-healing are complex.
- This is a fragile process.
- Failure to progress in the stage of wound-healing has the potential to lead to chronic wounds.
- Careful, patient-centred wound care can accelerate the wound-healing process.

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Wound healing

This text explains that there are many factors associated with the healing of wounds, for example, internal (coexisting disease, the ageing process and nutritional status) and external (pressure, friction and sheer) factors. Wound-healing is a natural response to the tissue injury, a restorative response. Healing occurs as a result of the interaction of a complex cascade of cellular events that produces resurfacing, reconstitution and restoration of the tensile strength of the skin that has been injured. There are also a number of phases of wound healing (Figure 9.1):

- The vascular response (this is where haemostasis takes place)
- The inflammatory response (here the body puts in place a variety of inflammatory responses)
- The proliferation phase (the active growth phase with granulation and epithelisation occurring)
- The maturation phase.

Whilst healing is a systematic process, the four classic phases mentioned above often overlap with each other (as is the case here). For the sake of discussion and understanding, the process of wound- healing is usually presented as a series of separate discrete events. In reality, it must be noted that the whole process is much more complicated, as cellular events that lead to scar formation overlap. There are many aspects of wound-healing that are still to be understood.

The maturation phase

Collagen

During the maturation phase, collagen remodelling depends on the continued collagen synthesis in the presence of collagen destruction. In the wound, collagenases and matrix metalloproteinases (an enzyme) help with the removal of excess collagen, as the synthesis of new collagen persists. Tissue inhibitors of metalloproteinases limit these collagenolytic enzymes, helping ensure that a balance exists between the creation of new collagen and the elimination of old collagen.

As the remodelling occurs, collagen becomes more progressively organised. A number of complex interactions take place with fibronectin gradually disappearing and hyaluronic acid and glycosaminoglycans replaced by proteoglycans. The types of collagen change, and type III collagen is replaced by type I collagen. Water is resorbed from the scar. These events allow the collagen fibres to lie closer together, enabling collagen cross-linking and ultimately reducing scar thickness. Intramolecular and intermolecular collagen cross-links result in increased wound bursting strength (tensile strength). Approximately 21 days after injury, the remodelling begins when the net collagen content of the wound is stable; this can continue indefinitely. Wound-healing is a complex process; see Box 9.1 for details.

The measurement of the tensile strength of a wound is its load capacity per unit area. The bursting strength of a wound is associated with the force needed to break a wound regardless of its dimension. Bursting strength differs according to skin thickness. The peak tensile strength of the wound is achieved approximately 60 days after the injury. When the wound heals, it only reaches approximately 80% of the tensile strength of the unwounded skin.

Cytokines

These are the significant mediators of wound-healing events. A cytokine is a protein mediator that is released from numerous cell sources, binding to cell surface receptors with the purpose of stimulating a cell response.

Cytokines can reach their target cell via various routes. The first cytokine described was the epidermal growth factor, which is a potent mitogen (a substance that encourages cell division) for epithelial cells, endothelial cells and fibroblasts. Epidermal growth factor stimulates other activities in the wound-healing factors, such as fibronectin synthesis, angiogenesis, fibroplasia and collagenase activity. Fibroblast growth factor stimulates angiogenesis. This factor also stimulates wound contraction, epithelialisation and the production of collagen.

Platelet derivative growth factor (PDGF) is released from the platelets and is responsible for the stimulation of neutrophils and macrophages. It is a mitogen and chemotactic agent for fibroblasts and smooth muscle cells, stimulating angiogenesis, collagen synthesis and collagenase.

Transforming growth factor-β is an important stimulant for fibroblast proliferation and the production of proteoglycans, collagen and fibrin. The factor promotes accumulation of the extracellular matrix and fibrosis; it has the ability to reduce scarring and to reverse the inhibition of wound-healing.

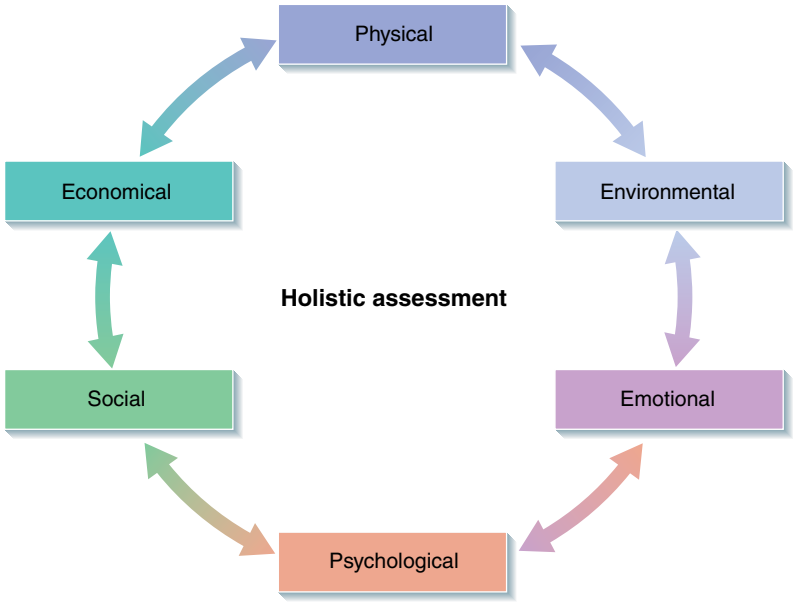
Tumour necrosis factor-α is produced by macrophages and stimulates angiogenesis and the synthesis of collagen and collagenase. This factor is a mitogen for fibroblasts.

During the maturation phase (also known as the 'reconstruction phase'), remodelling of the scar continues for approximately 1 year. Scar tissue regains about two-thirds of its original strength; it will never be as strong as the original tissue it replaces. Maturation is the final phase occurring once the wound is closed. This phase includes the remodelling of collagen from type III to type I. Cellular activity reduces, and the number of blood vessels in the wounded area regress and decrease.

The ability to closely approximate uninjured tissue is very much dependent on the size, depth, location and type of wound, as well as on a person's nutritional status, wound care and overall health.

10 Factors affecting wound-healing

Figure 10.1 Components of holistic assessment.



Wound Care at a Glance, Second Edition. Ian Peate and Melanie Stephens.

Companion website: http://www.ataglanceseries.com/nursing/woundcare/

Holistic assessment

There are many factors that influence the healing process and healing rates of wounds; these include physical, environmental, emotional, psychological, social and economical factors. In order to ensure that a wound progresses, as far as it is possible, through the normal healing process within a reasonable time frame, it is essential that these factors are considered when carrying out a wound assessment. Simply assessing the wound in isolation will not identify any issues that may be preventing a wound from healing, or which could be slowing down the rate at which it will heal. Therefore, a holistic assessment must be completed as regularly as a wound assessment in order to identify and rectify potential delays in the wound-healing rates. The holistic and wound assessment process will be discussed later in Chapter 17, and, in the meantime, Figure 10.1 shows how each factor can impact the other factors. Let us briefly consider some of these factors and how they can affect the patient and healing rates.

Physical

This factor considers the patient in the physical sense; for example, age, gender, underlying medical conditions, medications, physique, weight, height and lifestyle. In other words, all intrinsic factors associated with the patient. The failure to identify and address relevant physical issues, where it is possible to address them, could not only impact on the wounds' ability to heal but also on the following holistic issues.

Many aspects of the patient cannot be altered, such as age; however certain medical conditions, could be controlled to optimise the patient's health and healing prospects, such as diabetes.

Environmental

External factors can impact on a wound's ability to heal; therefore, consideration must be given to this aspect when assessing the patient; for example, the environment the patient is nursed in. It is important to consider who (if anyone) is providing care to the patient. Is the care appropriate? Are the staff/carer knowledgeable about the standards of care? Is the patient's clothing or footwear appropriate and not ill-fitting? Ill-fitted clothing could reduce circulation or add pressure to the patient's skin, resulting in wound deterioration or pressure damage. Is the surface on which the patient is sitting or lying comfortable? Are the medical devices that are being used safe enough? Surfaces and devices can easily cause pressure damage or affect the circulation of blood to a wound, if kept in the same vicinity. Is the patient getting access to good nutrition? Who is providing the food, and is it of good quality? The environment as a whole must be considered when assessing the patient (more on this later).

The wound environment must be considered a priority. There is always the possibility of an inappropriate dressing applied to a wound that does not maintain a moist wound-healing environment that is conducive to optimum healing. Also, if wound dressings are being tampered with by the patient or by others (such as family, carers and other untrained, unskilled persons). These factors should be addressed and dealt with.

Emotional

What is affecting the patient's feelings? Is the patient worried about issues such as income? Does the patient have family worries or stressors? Does the patient have any kind of pain or anything else that could affect his/her emotions, such as bereavement or loneliness? Emotional issues are known to impact on the patient's physical well-being and skin integrity, and this can be evidenced in some people who are prone to developing rashes or herpes simplex (cold sores) when under a lot of emotional strain.

Psychological

This factor can be linked quite closely with the symptoms resulting from emotions, which can impact on the patient's psychological status and can lead to psychological breakdown. It is important to consider factors such as psychological illness and neurological impairments, such as dementia, all of which could affect the patient's mental capacity (either permanent or temporary). Psychological issues can impact on the patient's physical well-being in a similar way to any emotional burdens they may have, whilst exacerbating those emotions.

Social

The examples of social issues could be loneliness and isolation, poor housing and/or living conditions, poor nutrition, poor standards in hygiene, overcrowding and lifestyle. This factor can also be linked to emotional and psychological status of a patient, and if left unaddressed could impact on their physical well-being. For example, a patient with a malodorous wound could become isolated due to the embarrassment about the odour emanating from their wound. In time, the patient could become housebound, lose their independence and become lonely due to the lack of socialising.

Economical

This factor could indicate financial issues with the patient that could impact on the type and quality of nutrition the person is able to afford. The cost issue with regard to the lack of appropriate bedding, seating, clothing or footwear could impact on the patient's wound and skin integrity as a whole. This factor could also have a bearing on all of the aforementioned factors.

The type of dressing must also be considered for use on the wound; whilst cost-effectiveness must always be aimed for, it is not always the case that the cheapest or most expensive product is the most appropriate.

Whilst cost must be a consideration in all of the above economical aspects, much of it is out of the control of the nurse and may be dependent on the patient's own budget. However, where possible, due consideration must be given to the patient's needs, and finance must not be the main driving force when selecting any products or devices.

Acute complications

Part 4

Chapters

22 Recognizing and treating hypoglycaemia

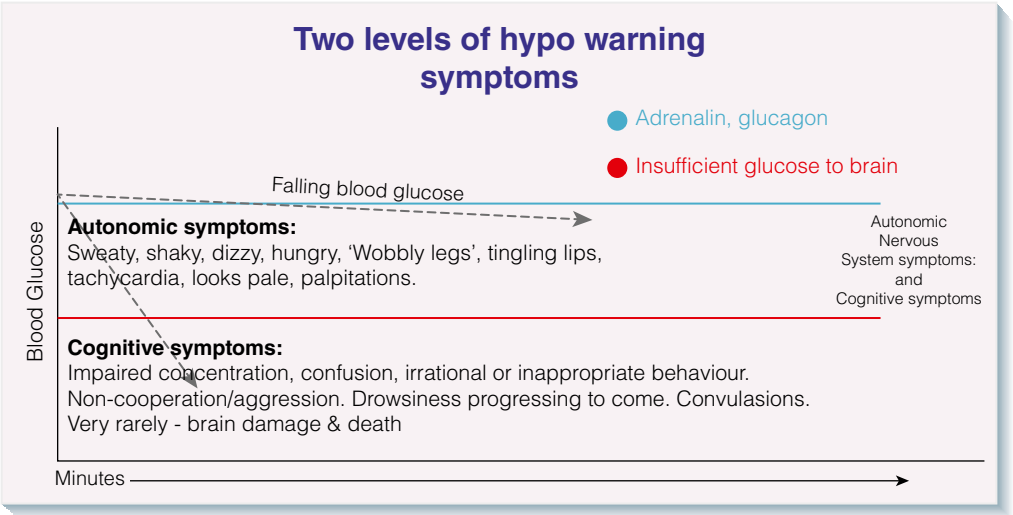
Figure 22.1 Factors which affect blood glucose levels during the day and night.

- Type of food consumed
- Timing of medication and meals
- Physical activity
- Medications
- Age
- Stress
- Dehydration
- Temperature in the environment
- Illness
- Menstrual periods
- Alcohol
- Renal function

Figure 22.2 Symptoms of hypoglycaemia.

- Lightheadedness
- Dizziness
- Confusion
- Irritability
- Lack of concentration
- Nervousness
- Shakiness
- Anxiety
- Pallor
- Sweating
- Clamminess
- Palpitations and a fast heart rate
- Hunger
- Sleepiness
- Fainting
- Tingling lips and tongue
- Or no symptoms at all to the individual if they have hypo unawareness

Figure 22.3 Progress of hypoglycaemia.



Diabetes Care at a Glance, First Edition. Edited by Anne Phillips.

This chapter is to help you understand what hypoglycaemia is but also to help you treat hypoglycaemia effectively. The Hypobox, which is available in many NHS trusts, GP practices and schools, is a an important tool to help people who may be potentially or actually experiencing low blood glucose (known as hypoglycaemia) and who may need your help.

Someone with either type 1 or type 2 diabetes treated with glucose-lowering medications (e.g. insulin or sulfonylureas such as gliclazide) can experience low blood glucose or hypoglycaemia. The usual blood glucose levels for people without diabetes range from 3.5 to 6.5 mmol/l (63–117 mg/dl). Blood glucose varies with food intake and measurements are usually taken before eating (preprandial) and after eating (postprandial). For people with diabetes the aim is for the lowest blood glucose to be 4 mmol/l (72 mg/dl) and to treat with fast-acting glucose if the blood glucose is below 4 mmol/l. This is commonly known as '4 is the floor'. Various factors affect blood glucose levels and these are listed in Figure 22.1.

The brain is dependent on glucose to function effectively. Because the brain cannot store glucose, it needs a constant supply; consequently, as blood glucose levels start to fall, the brain is unable to function as normal, a condition known as neuroglycopenia. If blood glucose starts to fall below 4 mmol/l (72 mg/dl), then the individual begins to experience different signs and symptoms and their behaviour may change (Figures 22.2 and 22.3). Initially the individuals affected may appear confused, flustered or quite disorientated and may not recognize these symptoms themselves.

Affected individuals will also develop pallor, but sometimes a bluish tinge appears around their lips and tongue. They will also start to sweat very suddenly and profusely, called a cold sweat; this is different from the normal sweating that occurs after physical activity or exercise. Individuals may start shaking and lack coordination so that they are unable to carry out your instructions or requests for them to sit down; if this occurs, you will be required to guide them to a place of safety.

The lack of glucose also causes an increase in heart rate and individuals can experience palpitations and consequent release of adrenaline, which in some people may induce anxiety, discomfort and fear. As the blood glucose drops even further, individuals start to become sleepy. Not everybody will exhibit the signs and symptoms in the same way, as it depends on the individual and also on how rapidly the blood glucose is falling.

It is important to remember that these signs and symptoms are simply indications of what people might present with and that hypoglycaemia is only one cause. If the individual's signs and symptoms cannot differentiate between hypoglycaemia and hyperglycaemia, start treatment for low blood glucose because it is very important to avoid the risk of fitting (seizures).

In the first instance, individuals should be encouraged to treat themselves and hopefully they will have some fast-acting glucose stores on their person. However, low blood glucose causes some people's characters to change completely, and they can become very aggressive, very vocal, or may cry. This is worth bearing in mind especially with those who have lived with diabetes a long time and have avoided taking fast-acting glucose, so may resist attempts to give glucose to them.

It is recommended to use pure glucose sources first, for example dextrose tablets or a glucose-type drink. The reason for this is because pure glucose is rapidly absorbed and released into the bloodstream to increase blood glucose. Combinations of glucose and fructose (a fruit sugar found in smooth orange juice, jelly beans or small bags of sweets) are absorbed slower. If this is the only option available, then it should be used, but try to treat with the quickest option first.

Avoid using anything containing chocolate, for example a chocolate bar or a chocolate bar with toffee, because they contain a lot of fat and fat is very slow to be absorbed, so is not a good treatment for hypoglycaemia. In particular, avoid hot drinks containing sugar because there is a risk of burns if they are spilt.

The person should recover within about 5–10 minutes. Once recovery occurs and the blood glucose level has returned to above 4 mmol/l (72 mg/dl), encourage the individual to eat some carbohydrate-rich food, for example a sandwich or a banana or a couple of biscuits. Reassure them that they are recovering because they will be very frightened and potentially a bit embarrassed about what has happened.

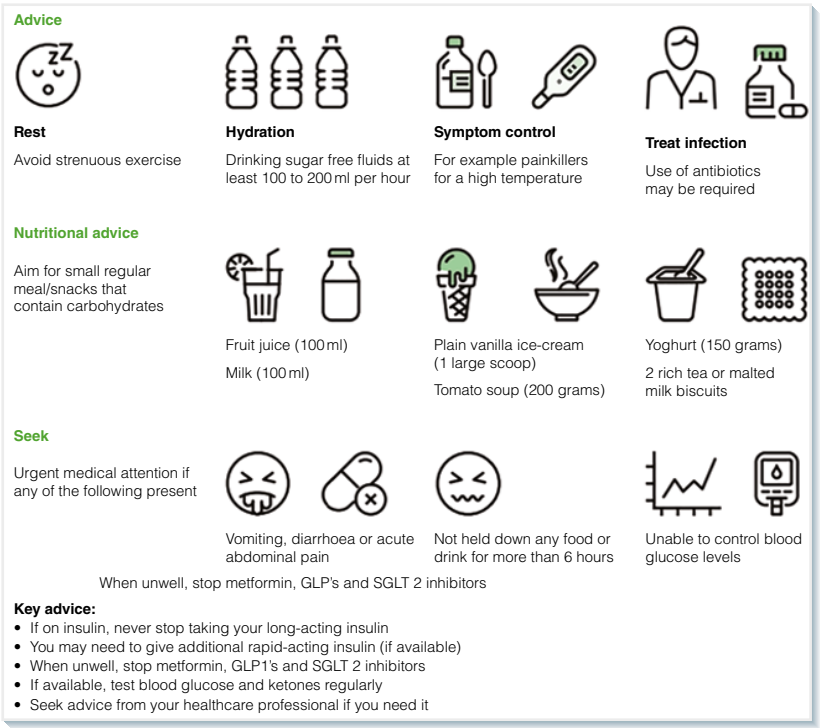
If recovery has not occurred and the blood glucose is still less than 4 mmol/l (72 mg/dl) after 5–10 minutes, then repeat the treatment and try to use a quick-acting glucose store. However, if recovery has still not occurred after the repeat treatment, and the individual is becoming unconscious or fitting, then help is required immediately. Place the person in the recovery position and ring the resus bell in hospital or call 999 in the community.

Any intervention is for the individual's own safety and so the most important aims are to treat the hypoglycaemia, treat the low blood glucose, enable the person to recover, and then follow up with a carbohydrate source of food. Document the hypoglycaemia and seek medical or diabetes specialist nursing review.

23 Sick day advice

Figure 23.1 Sick day management advice for type 1 diabetes. *Source:* Adapted from Down (2020), Diabetes UK (2020) and Trend UK (2020a).

Figure 23.2 Sick day management advice for type 2 diabetes. *Source:* Adapted from Down (2020), Diabetes UK (2020) and Trend UK (2020b).



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Like most people, those living with diabetes occasionally experience intercurrent illness, which has the potential to cause acute complications. Any intercurrent illness can cause glucose levels to rise and these include the common cold, diarrhoea and vomiting, and urinary tract infections. Intercurrent illness is the development of a new disease or illness occurring during the progress of another condition.

The recent COVID-19 outbreak has added to the list of illnesses and the need to reiterate self-management advice to people living with diabetes. Recent studies have linked an increased prevalence and poor outcomes with COVID-19 in people with both type 1 and type 2 diabetes. If blood glucose levels are not well controlled before hospital admission, people with diabetes and COVID-19 generally take longer to recover or have long-term effects from the condition.

How illness affects management of diabetes

The stress response due to illness results in the body preparing itself by ensuring that energy (glucose) is readily available. Insulin levels fall and hyperglycaemia results from the release of stress hormones such as glucagon and adrenaline (epinephrine), which oppose the action of insulin. The action of glucagon leads to increased glucose release by the liver and a rise in growth hormone and cortisol levels, causing body tissues (muscle and fat) to become less sensitive to insulin (called insulin resistance). As a result, more glucose is present in the bloodstream and although the individual may not have missed their dose of medication, the effect of illness increases their insulin requirements. Previous chapters have shown that insulin is required by the body to move excess glucose from the bloodstream to the cells that need glucose for energy. Ketones are formed by the liver as an alternative source of energy when the cells cannot access the glucose because of a shortage of insulin (see Chapter 24).

The symptoms that a person can experience when unwell are:

- feeling thirstier than usual
- feeling nauseated or vomiting
- elevated glucose levels
- elevated ketone levels
- passing more urine than usual.
- Urgent medical attention should be sought if the individual is drowsy or is vomiting or is experiencing abdominal pain and fast deep breathing as these are very serious symptoms.

Acute complications

People living with diabetes may be able to self-manage hyperglycaemia and a minor degree of ketonaemia; however, there is always a risk of developing acute complications such as diabetic ketoacidosis (DKA) and hyperosmolar hyperglycaemic state (HHS). Occasional hyperglycaemia (elevated blood glucose levels) is a common occurrence in both type 1 and type 2 diabetes and is usually resolved by adjusting either carbohydrate intake or insulin dosage.

However, if diabetes is not managed well during illness it can escalate and result in more serious conditions such as DKA and HHS, which would require emergency hospital admission. DKA and HHS are discussed in Chapter 24.

What people need to know when managing their diabetes during illness

People living with diabetes need to know how to manage blood glucose during periods of illness (called sick-day management). This includes specific information about frequency of blood glucose monitoring, blood glucose targets, checking for ketones, what ketone testing results in urine or blood indicate, taking extra quick-acting insulin, appropriate adjustment of insulin doses, identifying early signs and symptoms of DKA or HHS, and knowing when to contact the diabetes specialist team. Additionally, advice should be given about what medications should be stopped in episodes of vomiting and diarrhoea and when to escalate their care accordingly.

People with diabetes need to have adequate access to repeat prescriptions of the equipment that will enable them to manage their condition during illness. This includes glucose strips, ketone testing strips, and lancets for those who usually use them (mainly people taking insulin or diabetes medication that can cause hypoglycaemia or who are pregnant).

Organizations such as Trend Diabetes UK have produced handy, easy-to-follow leaflets regarding sick-day management for individuals with type 1 and 2 diabetes that healthcare practitioners can not only use themselves, but signpost to people with diabetes.

Figures 23.1 and 23.2 summarize the sick day management advice for people living with diabetes.

Summary

Whilst most people living with diabetes are successfully self-managing when they are well, illness can have a variety of effects on a person's glycaemic control and overall well-being. The overall effect of illness could result in temporary erratic glucose levels but could also lead to serious complications such as DKA and HHS.

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Diabetes UK(2020). Diabetes when you're unwell. www.diabetes.org.uk/guide-to-diabetes/life-with-diabetes/illness (accessed 1 July 2022).

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24 Diabetes-related ketoacidosis

Figure 24.1 Comparison between DKA and HHS.

• Signs and symptoms of DKA:	• Signs and symptoms of HHS:
Remember...	**Remember...**
• Signs and symptoms of DKA often develop quickly, sometimes within 24 hours	• HHS occurs gradually over a few days
• DKA is potentially life-threatening and requires urgent hospital admission	• Can overlap with symptoms of DKA
	• HHS is potentially life-threatening and requires urgent hospital admission
• Polydipsia (excessive thirst)	• Elevated blood glucose levels of above 30 mmol/l
• Polyuria (excessive or an abnormally large production of urine)	• Confusion – new confusion or worsening of pre-existing confusion
• Dehydration (dry mouth, tongue, skin)	• Polyuria
• Laboured breathing	• Polydipsia
• Abdominal pain	• Dehydration
• Nausea and vomiting	• Nausea
• Drowsiness	• Gradual drowsiness
• Confusion	• Loss of consciousness
• The smell of ketones on breath (pear-drops)	
• Ketones in urine dipstick or blood ketone test	

Diabetes-related ketoacidosis (DKA) is a potentially life-threatening condition when there is severe lack of insulin. This means the body's cells cannot access glucose for energy and start to use fat instead. The liver breaks down fats, and compounds called ketones are produced by this process. When ketones are released, they can be a useful source of energy in emergency situations. However, if left unchecked, ketones can accumulate and make blood acidic, hence the term 'acidosis' (JBDS-IP 2021).

Who can develop it?

DKA is a serious condition that affects people with type 1 diabetes, and occasionally those with type 2 diabetes. In some cases, DKA may be the first sign that someone has diabetes. Some people have ketosis-prone type 2 diabetes. DKA can develop very quickly over several hours.

How is DKA diagnosed?

There are three blood tests used in the diagnosis of DKA.

1 Elevated blood glucose levels (>11 mmol/l): if there is not enough insulin in the body to allow glucose to enter the cells, blood glucose levels will rise (hyperglycaemia). As the body breaks down fat for energy, blood glucose levels will continue to rise.
2 Elevated ketone levels (blood ketones above 3 mmol/l or urine ketones above 2+ on a dipstick): when the body breaks down fat for energy, ketones enter the bloodstream.
3 Blood acidity (pH <7.3 on blood gas analysis; normal pH is 7.35–7.45): excess ketones in the blood cause it to become acidic

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(acidosis) and this can change the normal function of organs throughout the body.

Signs and symptoms of DKA

- High blood glucose levels.
- Rapid breathing.
- Being very thirsty.
- Needing to pass urine more often.
- Feeling tired and sleepy.
- Confusion.
- Stomach pain.
- Feeling or being sick.
- Sweet or fruity-smelling breath (like nail polish remover or pear drop sweets).
- Passing out.

What are the triggers that can lead to DKA?

DKA can be triggered by an illness or infection that can cause the body to produce higher levels of certain hormones, such as adrenaline or cortisol. Unfortunately, these hormones act in opposition to the effect of insulin and sometimes trigger an episode of DKA. Examples include chest and urinary tract infections. Sometimes a stressful event such as a heart attack or stroke can be a trigger.

Another issue can be a problem with insulin therapy. Missed insulin doses, not taking enough insulin or a faulty insulin pump can leave a person with insufficient insulin in the body, triggering DKA.

How is DKA treated?

Treatment usually involves the following.

- **Fluid replacement**: most likely intravenous (IV) fluids to replace those lost through excessive urination; also help dilute the excess glucose in the blood.
- **Electrolyte replacement**: electrolytes are salts/minerals (e.g. sodium, potassium, chloride) in the blood that carry an electrical charge. The absence of insulin can lower the level of several electrolytes in the blood. These are initially given intravenously to help keep heart, muscle and nerve cells functioning normally.
- **Insulin therapy**: insulin reverses the processes that cause DKA, and this will initially be given intravenously as well. When blood glucose levels fall to around 11.1 mmol/l and the blood is no longer acidic, the person may be able to stop IV insulin therapy and resume their normal subcutaneous insulin injections.

The underlying cause of the DKA also needs to be managed, for example treating an infection or educating the person about sick day management.

How to avoid DKA

Diabetes can be a stressful condition to live with, but the following are some steps that can reduce the chances of developing DKA.

- Taking diabetes medications or insulin as prescribed and adjusting insulin dosages as needed with the support of diabetes healthcare professionals.
- Medication doses often need adjusting in relation to factors such as blood glucose levels, food, activity and illness.
- People using insulin pumps need to ensure that they have a back-up supply of insulin pens in case the pump stops working.
- Following sick-day management advice is also important. Blood glucose levels should be monitored frequently, and more often during illness or stress. Careful monitoring is the only way to ensure that glucose levels remain within target range.
- Checking ketone levels when hyperglycaemic and during illness or stress. If ketone levels are not reducing with sick-day management, individuals should contact their primary or secondary healthcare professional or seek emergency care.

Hyperosmolar hyperglycaemic state

Hyperosmolar hyperglycaemic state (HHS) is a serious condition that occurs in people with type 2 diabetes who experience very high blood glucose levels (often over 30 mmol/l). It can develop over a course of weeks through a combination of illness (e.g. infection) and dehydration (JBDS-IP 2022). There are some similarities to DKA, but the main symptoms include urination, thirst, nausea, dry skin, disorientation and, in later stages, drowsiness and a gradual loss of consciousness.

Figure 24.1 shows the main features of DKA and HHS.

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JBDS-IP (2022). The Management of Hyperosmolar Hyperglycaemic State (HHS) in Adults. Joint British Diabetes Societies for Inpatient Care, London. Available at https://abcd.care/resource/jbds-06-management-hyperosmolar-hyperglycaemic-state-hhs-adults-diabetes.

25 Diabetes and steroids

Figure 25.1 Different steroids, doses and duration of action.

Steroid	Potency (equivalent doses)	Duration of action (half-life, in hours)
Hydrocortisone	20 mg	8
Prednisolone	5 mg	16–36
Methylprednisolone	4 mg	18–40
Dexamethasone	0.75 mg	36–54
Betamethasone	0.75 mg	26–54

Figure 25.2 The effect of corticosteroids on blood glucose.

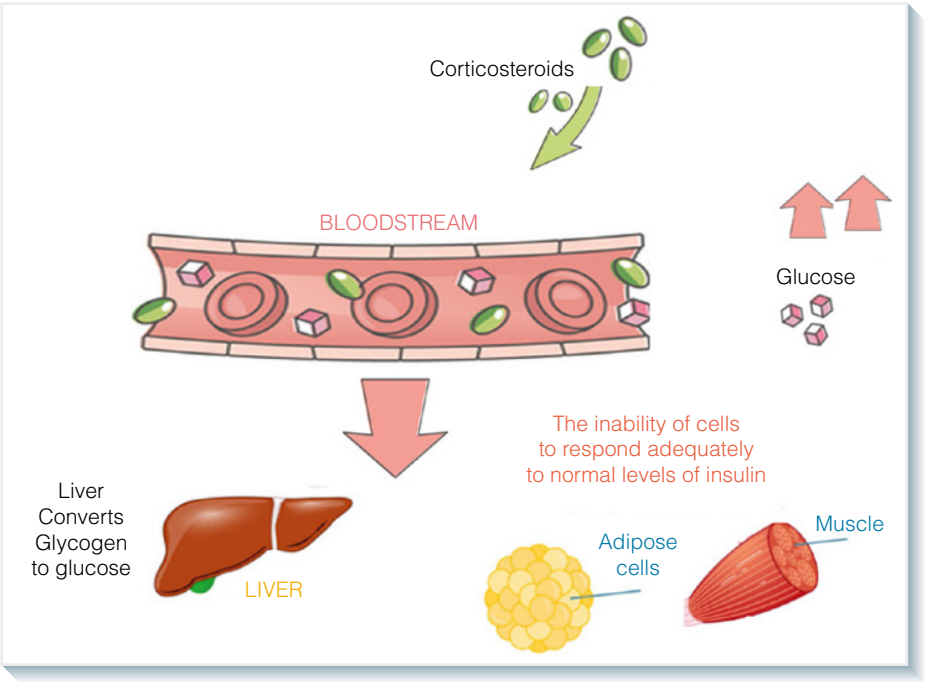

Figure 25.3 Risk factors for steroid-induced diabetes.

Risk factors of steroid-induced diabetes

There are some factors that may mean you are more likely to develop diabetes if you are taking steroids. These include if you:

- are over 40 and white, or over 25 and African-Caribbean, Black African or South Asian
- have a close family member with type 2 diabetes
- are of African-Caribbean, Black African or South Asian descent
- have had high blood pressure
- are living with obesity.

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What are steroids?

Steroids are also known as corticosteroids. They are artificial versions of hormones that are naturally produced by the body. They reduce inflammation and can help to treat a wide range of conditions, including:

- severe asthma
- cystic fibrosis
- arthritis
- inflammatory bowel disease
- some types of cancers.

(Note that these are different from anabolic steroids.)

Different steroids

There are different types of steroids, which can be prescribed in many forms. High doses of steroids are often taken orally or as an injection and are more likely to affect blood glucose levels. Some people with diabetes are also being treated for various inflammatory conditions with steroids such as dexamethasone, hydrocortisone, prednisolone and intravenous methylprednisolone. More recently, dexamethasone has been used in the management of COVID-19 (Diabetes UK 2020; National Institute for Health and Care Excellence 2021). Figure 25.1 provides a list of widely used corticosteroids and their equivalent doses and duration of action.

Steroid-induced hyperglycaemia or steroid-induced diabetes

A rise in glucose related to steroid therapy occurring in people without a known diagnosis of diabetes is termed steroid-induced diabetes. The use of steroid treatment in people with pre-existing diabetes will undoubtedly result in worsening glucose control; this may be termed steroid-induced hyperglycaemia.

How do steroids cause steroid-induced diabetes?

Steroids increase blood glucose levels in two ways.

- They reduce the body's sensitivity to insulin by reducing the ability of muscle and fat (adipose) cells to absorb glucose from the blood (known as insulin resistance).
- They cause the liver to release stored glucose, even when the body does not need additional glucose.

Figure 25.2 shows how steroids impact blood glucose levels.

Is steroid-induced diabetes permanent?

Many people will find that their blood glucose levels return to a healthy range when they stop taking steroids. For others, however, steroid-induced diabetes can continue even after stopping their treatment. This is more likely if they are at higher risk of developing type 2 diabetes. The risk factors for type 2 diabetes are shown in Figure 25.3.

Signs and symptoms of steroid-induced diabetes

Patients taking steroids, but who are not monitoring their blood glucose, will need to be aware of the symptoms of hyperglycaemia. These are the same as the symptoms of undiagnosed diabetes (discussed in Chapter 3), which include:

- excessive thirst
- excessive urination
- feeling very tired
- possibly losing weight unintentionally.

People who are already monitoring their blood glucose may need to increase the frequency or adjust their medication with the support of their diabetes primary or secondary care teams. If no action is taken, the individual may develop acute and long-term complications associated with hyperglycaemia.

How is it diagnosed?

The diagnostic criteria for steroid-induced hyperglycaemia do not differ from those for other types of diabetes and include a confirmed fasting venous blood glucose of 7 mmol/l or above, a random venous glucose level of 11.1 mmol/l or above, or two or more capillary blood glucose levels above 12 mmol/l. When people are in hospital, there will be specific guidelines on how to identify and manage steroid-induced diabetes (Joint British Diabetes Societies for In-patient Care 2021).

Treatment of steroid-induced hyperglycaemia

Treatment needs to be individualized, for example length of the course of steroids and the steroid dose. Sulfonylureas (SUs) such as gliclazide and glipizide are often used to tackle mild steroid-induced hyperglycaemia (Chapter 14 discusses different oral diabetes therapies). SUs stimulate the pancreas to release insulin and are best suited to manage the peaks in glucose caused by steroids. People who start taking SUs need to be provided with a blood glucose monitor and receive education in how to use it. They need to monitor their blood glucose levels regularly and be in contact with diabetes primary or secondary care teams for advice on whether they need to adjust their dose. Monitoring also helps them to detect hypoglycaemia (low blood glucose levels). There is an increased risk of hypoglycaemia with SUs, especially when steroid doses are tapered or if meals are skipped. Part of their education therefore needs to include how to manage hypoglycaemia.

In those with significant hyperglycaemia, some people may need to start insulin. Insulin has the benefit of having an immediate onset of action and doses can be increased regularly to an effective dose. These people will require education regarding insulin injection technique, blood glucose monitoring, adjustment of insulin, and recognition and management of hypoglycaemia. The insulin type will depend on the steroid and frequency of dose. For example, morning administration of an intermediate-acting insulin such as Humulin I may closely fit the glucose peaks during late afternoon to evening induced by a single dose of oral steroid in the morning. A basal analogue insulin such as Lantus may be appropriate if hyperglycaemia is present throughout the day and into the evening.

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