Watch the following video to ease you into this chapter. If you are using the eBook just click on the play button. Alternatively go to https://study.sagepub.com/essentialpatho/videos

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**LEARNING OUTCOMES**

When you have finished studying this chapter you will be able to:

1. Understand the principles of public health and the relevance to person-centred nursing.
2. Recognise the importance of epidemiology in recognising causes of diseases and how this understanding can be used to promote person-centred care.
3. Discuss how epidemiology contributes to diagnosis and treatment of disease and to prevention through public health.
4. Describe the main approaches used in epidemiological studies to confirm the causes of disease and to control its development.
5. Understand the value of cohort and case-control studies in following up progression of people with particular conditions.
6. Illustrate how epidemiology integrates with ethical and political considerations.
INTRODUCTION

Most of this book is about disorders affecting individuals and their management. However, this chapter has a much broader perspective: public health building on the science of epidemiology. This is currently defined by WHO as:

the science and art of preventing disease, prolonging life and promoting health through the organized efforts of society. (Acheson, 1988 cited by WHO Europe, 2012)

This is also described as:

the branch of medicine which deals with the incidence, distribution, and possible control of diseases and other factors relating to health. (Oxford Dictionaries, 2018)

More concisely:

Epidemiology is the study of health and disease in populations. (Saracci, 2010: 2)

Together these definitions identify the key concepts with which this chapter is concerned: epidemiology is the basic science underpinning public health.

Epidemiology enables us to begin to understand the range of factors that influence the distribution of disease through the population and how it spreads between populations. Understanding the principles of epidemiology enables us to recognise the relationship between different factors – genetic, social class, environment, behaviour, nutrition, education and others – and the presentation of disease. It has a key role in identifying necessary public health measures and assists in identifying the appropriate person-centred care. Central to this is facilitating individuals in making appropriate choices about lifestyle and thus influencing disease incidence in the population.

PERSON-CENTRED CONTEXT: THE BODIE FAMILY

Some members of the Bodie family provide examples of epidemiology in practice. George and Maud are examples of generally healthy elderly people with medical conditions that epidemiological research has identified as relatively common in their age group and public health interventions are prescribed.

George was diagnosed with raised cholesterol (hypercholesterolaemia) when he was 73 (9 years ago), since then he has been taking statins. He has also received guidance on lifestyle issues to reduce the risk of heart disease.

GO DEEPER

Atherosclerosis

Atherosclerosis is very common among the population at large. The consensus statement of the European Atherosclerosis Society (Nordestgaard et al., 2013) identifies that 1 in 500 of the general population are heterozygous for the relevant gene for familial hypercholesterolaemia, which increases the risk of
ischaemic heart disease due to atherosclerosis (Humphries et al., 2006). We do not know whether George’s form runs in the family or not.

However, there is evidence that there is considerable underdiagnosis and undertreatment of this condition in a number of developed and less developed countries (Ahn et al., 2015; Nordestgaard et al., 2013). For example, Pearson (2004) identified a considerable gap between those with a diagnosis of this condition and those receiving treatment in the USA. To improve this situation, OTC (Over-The-Counter) statins were being encouraged.

In the UK, only low-intensity OTC statins can be purchased. However, on-line pharmacies enable individuals to access statins via medical practitioners employed by these pharmacies. These doctors question people at a distance and tend to prescribe moderate intensity statins delivered by post. Most GPs will carry out a more comprehensive assessment, including blood tests, before prescribing appropriate treatment, which is likely to be higher density statins than OTC drugs. The majority of those taking statins in the UK are diagnosed and treated in the National Health Service by their General Practitioners and local pharmacies.

Maud also has some conditions which are more common in older people, including heart failure, which is being managed effectively, and hypothyroidism. She is an example of the incidence of hypothyroidism in areas which have adequate iodine, where it is higher among the elderly and 10 times more common in women than men (Vanderpump, 2011). These results are mainly from Caucasian populations (McGrogan et al., 2008).

Jack Garcia lives in New York and has a generally healthy lifestyle. However, his father had testicular cancer which was treated effectively. This condition is the commonest malignancy worldwide among young men (15–34 years), particularly Caucasians, and is usually curable, particularly when diagnosed early. In addition, epidemiological research with families has found that men with fathers with a history of testicular cancer are four times more likely to develop this than men in the general population (Manecksha and Fitzpatrick, 2009). Jack, at 28, is wisely performing self-examination monthly to ensure rapid treatment if anything abnormal is detected.

The siblings Derek and Margaret Jones both have conditions associated with atopy: Derek has asthma and Margaret hay fever. Atopy is the genetic tendency to develop the classic allergic diseases: atopic dermatitis, allergic rhinitis (hay fever) and asthma. Familial grouping of phenotypes is found, although different conditions may present – in this case one has asthma, the other hay fever. There are several chromosomal regions associated with atopic responsiveness (Koppelman et al., 2002), which may explain that two siblings are affected but neither parent is.

Danielle has been receiving the immunisations recommended at this stage in her life. George, Maud and Derek receive their influenza immunisations annually because they are all at increased risk of developing complications from flu. Other family members receive immunisations when going abroad. Epidemiological research has provided the knowledge identifying the value of these interventions and helped promote public health.

EPIDEMIOLOGY

The overall role of epidemiology has been outlined above. In addition, however, understanding the statistical analysis is important in being able to use the findings to plan future services. Statistics helps us to understand and interpret the world, including individuals with disorders with whom we work, and thus enhance the quality of care. However, the detail of statistics is not studied in this book.
Three main types of study within epidemiology can be identified: descriptive, analytical and intervention studies each contribute to our understanding of disease within populations (Carr et al., 2007). The findings contribute to understanding public health and planning health and other services to promote the health status of the community.

In addition, the field of epidemiology has been described in five areas, as shown in Table 2.1.

| Table 2.1  Five major areas in epidemiology |
|-----------------|--------------------------------------------|
| Descriptive epidemiology | Health and disease and their trends over time in specific populations |
| Aetiological epidemiology | Searches for factors (hazardous or beneficial) influencing health status (e.g. toxins, poor diet, pathogenic microorganisms, health-promoting behaviours) |
| Evaluative epidemiology | Evaluates preventative interventions, estimates risks of specific diseases for people exposed to hazards |
| Health services epidemiology | Describes and analyses work of health services |
| Clinical epidemiology | Describes natural course of a disease in patient population and evaluates effects of diagnostic procedures and treatment |

Source: Saracci, 2010: 11

Descriptive epidemiology

This branch of epidemiology examines the incidence of disease in relation to person, place and time. It aims to understand the patterns of health and disease within a population and to generate possible explanations for the findings. The sorts of questions asked in relation to a particular disorder are as follows:

- **Person**: what are the demographic details of those affected, i.e. age, gender, occupation, ethnic background, socioeconomic status, lifestyle factors such as smoking, diet, exercise? This sort of study can provide information about diversity within the population and enable the specific areas to be identified which need consideration with different groups (Lowth, 2015). For example, behaviours associated with certain religious and cultural practices may have potential health implications (Laird et al., 2007). Some examples include:
  - ritual fasting and potential implications for those with chronic diseases such as **diabetes mellitus**
  - potential exposure to infectious diseases when participating in the Haj (the major pilgrimage to Mecca that all Muslims are expected to undertake during their lifetime)
  - a high incidence of consanguineous marriage (i.e. marriage between relatives) increasing the risk of some genetic disorders
  - female genital mutilation (also known as cutting), in which a surgical procedure damages the female genital organs with no medical benefits, and possibly bleeding, infections and other complications including in childbirth (WHO, 2018a), and childhood marriage occur in some groups. While illegal in many countries (including the UK) both of these are often undertaken when girls whose families are living in Western countries are taken back to their ancestral homes and families during long holidays for the surgery and/or to marry (often) a relative whom they have never met. Efforts to minimise this practice include preparing teachers and training airport staff to identify potential girls at risk and putting ‘stickers’ up in toilet cubicles with advice on
how to get help (Iqbal, 2015). Nursing and midwifery professionals need to be able to recognise potential health issues associated with diversity and to intervene appropriately.

- **Place**: is there a difference in the incidence of the condition in some areas, e.g. related to geography, the condition of various parts of a city, the availability of local facilities?
- **Time**: does the incidence of the disease being considered vary over a number of years, at different times of the year, in different weather/climatic conditions, etc.?

Routinely available data

A significant amount of data related to diseases is available through routinely collected information; for example, death certificates must be completed by the attending doctor and the data becomes available to the Office for National Statistics (in the United Kingdom) and can be used to review changes in the incidence of specific diseases over time. A range of administrative data sets are also used and the Office for National Statistics coordinates the national decennial (10-yearly) census, next due in 2021.

**GO DEEPER**

Cholera and epidemiology

An example of an early epidemiological study was carried out by John Snow in 1854 when an area of London supplied by two water companies (the Lambeth Water Company and the Southwark and Vauxhall Company) had a cholera epidemic. Snow identified that the relative proportion of cholera deaths in parts supplied by the Southwark and Vauxhall Company was significantly higher than elsewhere, in particular in those areas where people got their water from the Broad Street Pump. Snow approached the Board of Guardians and reported that he believed that the cholera was spreading in the water from the Broad Street Pump. While this was an incredible suggestion in those times, they agreed to remove the pump handle. The cholera epidemic died down. His hypothesis that cholera was transmitted by contaminated water is now generally accepted by epidemiologists and his action recognised by the installation of the John Snow Memorial marking the epicentre of the outbreak (Rossignol, 2007).

Data on cardiovascular incidence and mortality is collected in numerous countries and parts of the world and demonstrates improving results. The epidemiological statistics enable the changes occurring and the effects of interventions on morbidity to be identified.

**APPLY**

Changes in cardiovascular morbidity

These are diseases of the heart and blood vessels, causing a range of disorders through central and peripheral parts of the circulatory system and are the primary cause of death worldwide. Over three-quarters of these deaths occur in low- and middle-income countries where they cause 37% of premature deaths (under 70 years) from non-communicable diseases (WHO, 2018b). (Continued)
However, it is also important to recognise the developments occurring and approaches to reducing morbidity (illness rate) and mortality (death rate) since they peaked in the 1960s (Luepker, 2016). In Europe these disorders have been diminishing in incidence since the 1980s but they are still the main cause of mortality and major cause of morbidity, but with considerable variability across the region. Central and Eastern Europe have higher death rates from these conditions than in Northern, Southern and Western Europe. These results tend to be linked with minimal levels of primary health care related to smoking and other lifestyle factors and inadequate approaches to early detection and treatment. While smoking has diminished across Europe, the highest rates are found in the countries of the old Soviet Union which also have high levels of cardiovascular diseases (CVDs) (Wilkins et al., 2017).

There is still progress to be made through health promotion activities to promote an appropriate diet to reduce obesity, limit smoking, encourage exercise and moderate drinking. In addition, conditions which increase risk, and therefore should be identified and treated effectively, include diabetes mellitus, hypertension and hypercholesterolaemia (WHO, 2018c).

Cross-sectional surveys

In this type of study, information is collected about the health status and other factors of interest in the population being studied. The aim is to identify the proportion of the population which has the disease being studied (i.e. the prevalence of the disorder is determined). This enables adequate planning of appropriate health services to be carried out.

Analytical studies

Analytical studies aim to identify associations between the diseases of interest and possible causes. There are four types of study in this group.

Ecological studies

These studies are different from most others considered here as data is collected on the whole population. Routinely collected disease data are compared with other factors in the population which could be causes of these diseases.

Cross-sectional studies

These have been mentioned above. In this context they are trying to identify the prevalence of disorders and association with various factors. For example, this type of study has demonstrated a relationship between obesity and diabetes mellitus, and behaviour.

Case-control studies

In these studies, one group of cases (who have the condition of interest) is compared with a group of controls (who do not have the condition). The individuals involved are screened by interview, survey or
previous records to determine their exposure. The exposure to potential cause of those in the two groups is compared to see if the exposure of the group of cases differs from that of the control group. If so, these variables may influence the progression and eventual disease outcomes (Rossignol, 2007).

Cohort studies

Two groups similar in characteristics relevant to the study (except for the condition of interest) are selected and the outcome of interest is compared. Cohort studies can assess a range of outcomes, allowing an exposure to be rigorously assessed for its impact in developing disease, although these studies are lengthy and expensive, especially if the follow-up period is extensive. They are useful for studying exposure to rare conditions such as radiation from the Chernobyl nuclear power plant in 1986. The study can be retrospective or prospective; in either case the groups are similar apart from the condition being studied.

Retrospective

In this case both groups have been exposed to similar conditions except in relation to the key issue being studied and outcomes have occurred. One group has been exposed to the condition being studied while the other has not. The data has already been collected and the results will be studied.

Prospective

In these studies, all the participants come from the same study population and are divided into two groups, one of which will have been exposed to the characteristic under study, and the other will not. As far as possible, the two groups are comparable on other relevant characteristics. Those in both groups are then followed for a specified period to identify the individuals who develop the expected outcome/disease.
Intervention (or experimental) studies

The difference between these and the previous types of study is that in this situation the researcher intervenes to change the exposure of the participants to the factor being studied.

Clinical trials

In these studies, one group of people with a particular condition receive a specified treatment and their progress is compared with a second control group who are not receiving the active intervention. One of the key issues is to prevent bias so that the participants are randomly assigned to a group in what is known as a randomised double-blind controlled trial. The ‘double-blind’ part of this term indicates that neither the participant nor the person managing the trial knows which group the participant is in.

When a drug is being tested, the control group of participants normally receives a tablet that appears similar to the active treatment but is not active; it is what is known as a placebo. In some circumstances the study is aiming to compare a new treatment with the one currently in use.

Community trials

In these studies the study is examining the effect of an intervention on a community as the unit of study, not individuals. Because social, cultural and environmental conditions may be key factors in the incidence of disease, a community trial tries to alter these conditions community-wide and then evaluate the effect. However, it is not usually feasible to undertake double-blind controlled trials.

Contribution of epidemiology

Epidemiology plays an extremely important role in identifying conditions that contribute to health or disease, and in providing you with an enhanced understanding of how to contribute to person-centred care. In addition, it provides the knowledge and understanding that politicians and officials need to undertake the planning and implementation of conditions for optimum health for individuals and the community through public health. An example is the identification of particular conditions which are more common in those from particular ethnic groups or geographical areas than other members of the population.

PUBLIC HEALTH

Public health is underpinned by the science of epidemiology and the WHO definition has already been presented. An earlier US description, still widely quoted and as relevant as ever, is below.

GO DEEPER

Public health

In 1920, Winslow, an American public health specialist of his time, defined public health as follows:

Public Health is the science and the art of preventing disease, prolonging life, and promoting physical health and efficiency through organized community efforts for the sanitation of the
environment, the control of community infections, the education of the individual in principles of personal hygiene, the organization of medical and nursing services for the early diagnosis and preventive treatment of disease, and the development of the social machinery which will ensure to every individual in the community a standard of living adequate for the maintenance of health.

(C.E.A. Winslow, 1920, cited in Evans, 2011)

The key issue in relation to public health is that it is ‘public’ and focused on enhancing the health of the whole population. The World Federation of Public Health Associations (WFPHA) developed a Global Charter for the Public’s Health (GCPH) (Lomazzi, 2016), which identifies core services (protection, prevention, promotion) and functions to enable these services (governance, advocacy, capacity and information) (Figure 2.1). These largely overlap with the areas considered below.

![Figure 2.1 Illustration of the Global Charter for the Public's Health (Lomazzi, 2016)](image)

Within each country the health system is determined by government involving public, private and voluntary sector organisations contributing to health maintenance, and activities influencing health behaviour and status. The structure and interaction within such organisations vary across different countries of the world. In the UK, the four countries of England, Scotland, Wales and Northern Ireland manage most of the public health issues independently, although with some coordination from national government.

**Areas of public health practice**

Organisations with public health remits have identified three areas of public health practice, shown in Figure 2.2. These are largely the same for different organisations but with some differences (FPH, 2010; Gray et al., 2006).
Health protection

This area of public health practice is concerned with actions to reduce exposure to factors that can impact on the development of ill-health. In general, these requirements require tackling at different levels. While normally there are adequate emergency services which undertake rescue and safety activities as necessary, circumstances can arise in which additional support is required and members of the public may contribute as they are able. The box below discusses unusual conditions in the UK which increased the health risks to the population and in which members of the public contributed to **health protection**.

**APPLY**

**Health protection provided by the public**

In the winter of 2017/18 some parts of the UK had very severe snow with potential public health risks. This sort of extreme weather is uncommon in the UK and resources for dealing with these conditions...
National government officials and ministers deal with major social issues such as war and severe social disorder, and ensure that suitable emergency response equipment and facilities are available. Regulations are introduced to ensure that hazards such as chemicals, poisons and radiation are dealt with in industry or elsewhere, in a way that limits the exposure of members of the public, and that those working with these risks are trained and equipped appropriately to maintain their safety. Local authorities have responsibility for ensuring the provision of clean air, water and food. Medical authorities carry the responsibility for ensuring that appropriate measures are taken to limit exposure to or to be immunised against infectious diseases.

Health improvement

This aspect of public health aims to improve the health and wellbeing of individuals or communities through enabling and encouraging healthy lifestyle choices as well as addressing underlying issues such as poverty, lack of educational opportunities and other such areas. (NHS Scotland, 2018)

Much of the work in this area is guided by regional government through organisations with different areas of expertise and responsibilities. The key issue is to reduce inequalities at individual, family and community level through a range of different initiatives which enhance the quality of life, life opportunities and well-being:

- **Housing:** is it possible for individuals and families to find accommodation that provides enough water, heating, space for children to play, and access to shops selling affordable items central to optimal health? Is there a library nearby? These are key issues for a satisfactory quality of life. Policies are often determined at government level and implemented locally.
- **Education:** is essential for development in life. The local education authority carries overall responsibility for the education services at primary and secondary level. Third-level education...
(i.e. post-18) is mainly independent and carries a major responsibility for preparation for the professions and skilled trades. Clearly this is central to the opportunities available for life through employment. In addition, friends made at this stage in life can influence lifestyle choices later. Education for health is also valuable although introduced in very different ways in schools of various types.

- **Employment**: an adequate income is essential for a satisfactory lifestyle. Are there opportunities in the area? What is local government doing to encourage additional employment in the area?
- **Lifestyles**: the factors considered above all influence the scope for lifestyle choices in relation to social, recreational, cultural and literary activities.
- **Specific diseases and risk assessment**: this aspect of public health is managed under the direction of central and local health authorities. It aims to carry out a range of activities to identify conditions associated with differing work conditions, such as asbestos in buildings being demolished being a risk factor for mesothelioma. The local building authorities are responsible for ensuring safe asbestos disposal. The local health services arrange influenza and other immunisations, breast cancer screening, maternal and child health clinics.

The range of issues above can influence quality of life, minimise risks to health and provide necessary information about health risks and how to promote health. Education, housing and traffic conditions, and lifestyles can all contribute to undertaking adequate exercise and limiting obesity. Some key factors need consideration which are central to the quality of health. Some particular examples are:

- Obesity has already been mentioned but is one of the key factors in health. Obesity has a major influence on the risk of disorders including **cardiovascular disease** and diabetes mellitus, which both have the potential to cause serious physical damage.
- Mental ill-health can have a major impact on the quality of life and the provision of mental health services for those at all ages with such problems is essential.
- Air quality is also crucial. Air pollution is identified as the world’s largest environmental health risk (WHO, 2018d). Of those living in urban areas, more than 80% are exposed to air quality levels that exceed the WHO limits, with those in low-income cities in contact with higher levels. Developments in vehicle manufacture with reduced impact on air quality are receiving considerable attention with the aim of reducing this factor.

**Service improvement**

This component of public health is concerned with the provision of a range of services which contribute to health in different ways.

- Research, audit and evaluation is concerned with identifying risks to health and the incidence, morbidity and mortality concerned with different disease processes. Advances in treatment are developed and resulting changes in epidemiological data are identified and used in decision-making about implementation in new treatments. The research carried out must be available for service managers.
- Service planning involves the research outputs evaluated by first-rate researchers and efficient managers who plan implementation to ensure clinical effectiveness and equity in access to care. The Cochrane Collaboration exists to improve health care decisions by gathering and summarising the best evidence involving a worldwide network of researchers, professionals and others involved in health care (Cochrane, 2018).
- Clinical governance has been described as ‘the system through which NHS organisations are accountable for continuously improving the quality of their services and safeguarding high standards of care by creating an environment in which clinical excellence will flourish’ (Public Health England, 2018).
Value of public health and health promotion

The discipline of public health has tended to be seen as having two major directions:

- ‘a broad focus on the underlying social and economic causes of health and disease and their variation in populations’
- ‘a narrower medical focus with treatment of ill health at its centre’ (Carr et al., 2007: 6).

The list of achievements in public health (Table 2.2) includes important examples from both these directions and also relates to the three areas of public health practice discussed above.

Health promotion is a key element in the three aspects of public health discussed above. It has been defined as enabling people to increase control over their own health. It covers a wide range of social and environmental interventions that are designed to benefit and protect individual people’s health and quality of life by addressing and preventing the root causes of ill health, not just focusing on treatment and cure. (WHO, 2016a: 1)

Table 2.2  Ten important public health achievements

<table>
<thead>
<tr>
<th>Vaccination</th>
<th>Eradication of smallpox; polio almost eliminated; immunisation against a wide range of diseases</th>
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<tbody>
<tr>
<td>Control of infectious diseases</td>
<td>Control of typhoid, cholera through clean water and sanitation Tuberculosis and sexually transmitted disease controlled by antibiotics</td>
</tr>
<tr>
<td>Decline in mortality from coronary heart disease and stroke</td>
<td>Risk factor modification (cessation of smoking, control of B/P) Improved access to early detection and treatment</td>
</tr>
<tr>
<td>Healthier mothers and babies</td>
<td>Better hygiene and nutrition, access to health care and technological advances in neonatal and maternal health care</td>
</tr>
<tr>
<td>Family planning</td>
<td>Altered socioeconomic role of women, reduced family size, improved maternal and child health, barrier contraceptives, reduced unwanted pregnancies and STD transmission</td>
</tr>
<tr>
<td>Safer and healthier foods</td>
<td>Decreased microbial contamination, increased nutritional content, nutritional deficiency diseases (e.g. rickets, goitre, pellagra) almost eliminated</td>
</tr>
<tr>
<td>Fluoridation of drinking water</td>
<td>Leading to reduction in tooth decay and loss of teeth</td>
</tr>
<tr>
<td>Recognition of tobacco use as health risk</td>
<td>Changes in social norms leading to reduced smoking and mortality from smoking-related disease</td>
</tr>
<tr>
<td>Safer workplaces</td>
<td>Reduction in fatal occupational injuries; control of pneumoconiosis and silicosis</td>
</tr>
<tr>
<td>Motor vehicle safety</td>
<td>Fall in motor vehicle-related deaths due to engineering improvements, vehicles and roads Changed behaviours - use of seat-belts, reduction in drink-driving, lowered use of mobile phones</td>
</tr>
</tbody>
</table>

Source: Gray et al., 2006, after Center for Communicable Disease Control, Atlanta, GA
Three key elements for health promotion have been identified (WHO, 2016a), which are discussed below:

- **Good governance for health**: involves health becoming central to government policy; being considered in relation to all decisions and policies aiming to prevent illness and injuries. Regulations introduced need to relate private activities with public goals, e.g., tax policies to dissuade unhealthy behaviours such as eating high salt food products, or seat-belt and other safety regulations. Local authorities have requirements laid upon them to promote healthy living conditions.

- **Health literacy**: people need knowledge, abilities and understanding to be able to make choices that promote health, for example about healthy food and accessing health care appropriately. They also need to be in a setting in which members of the community can influence policy.

- **Healthy cities**: play an important role in facilitating population health, contributing to healthy countries and, thus, a healthy world. High-quality functioning of local government makes an important contribution to urban planning and developing measures to promote community health and primary and emergency health care.

Public health activities are considered necessary in all parts of the world, although application will vary according to environmental, government and economic factors. The WHO Regional Office for Europe has identified essential operations for the implementation of public health (Table 2.3).

**Table 2.3** Ten essential public health operations (EPHOs)

<p>| | |</p>
<table>
<thead>
<tr>
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<tbody>
<tr>
<td>1</td>
<td>Surveillance of population health and well-being</td>
</tr>
<tr>
<td>2</td>
<td>Monitoring and response to health hazards and emergencies</td>
</tr>
<tr>
<td>3</td>
<td>Health protection including environmental, occupational, food</td>
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<tr>
<td></td>
<td>safety and others</td>
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<tr>
<td>4</td>
<td>Health promotion including action to address social determinants</td>
</tr>
<tr>
<td></td>
<td>and health inequity</td>
</tr>
<tr>
<td>5</td>
<td>Disease prevention, including early detection of illness</td>
</tr>
<tr>
<td>6</td>
<td>Assuring governance for health and well-being</td>
</tr>
<tr>
<td>7</td>
<td>Assuring a sufficient and competent public health workforce</td>
</tr>
<tr>
<td>8</td>
<td>Assuring sustainable organisational structures and financing</td>
</tr>
<tr>
<td>9</td>
<td>Advocacy, communication and social mobilisation for health</td>
</tr>
<tr>
<td>10</td>
<td>Advancing public health research to inform policy and practice</td>
</tr>
</tbody>
</table>


### Worldwide public health

We have been looking at some of the principles of public health and now we are examining some issues around public health worldwide or global health. This is defined as:

> an area for study, research, and practice that places a priority on improving health and achieving health equity for all people worldwide. (Koplan et al., 2009: 1995)

An important role in promoting worldwide health has been identified by WHO (2016b) in the Global Strategic Directions for Strengthening Nursing and Midwifery, as outlined in Figure 2.3.
Ensuring an educated, competent and motivated nursing and midwifery workforce within effective and responsive health systems at all levels and in different settings

Optimizing policy development, effective leadership, management and governance

Working together to maximize the capacities and potentials of nurses and midwives through intra and interprofessional collaborative partnerships, education and continuing professional development

Mobilizing political will to invest in building effective evidence-based nursing and midwifery workforce development

Available, Accessible, Acceptable, Quality and Cost-effective nursing and midwifery care for all, based on population needs and in support of UHC and the SDGs

Figure 2.3  Global strategic directions for strengthening nursing and midwifery, 2016-2020

Source: WHO (2016b)
Vision, thematic areas and principles are identified which aim to achieve high-quality nursing care for all to meet population needs through supporting UHC (Universal Health Coverage) and SDGs (Sustainable Development Goals). Clearly nurses and midwives have the potential to make substantial contributions to these goals.

Key areas of inequalities, challenges and health disorders are discussed below.

Inequalities

One of the key issues in considering global health is about inequalities beginning early in life. The effects of relative poverty during pregnancy are marked and long-lasting, with deleterious influences on childhood illness and behavioural problems. In particular, the limited resources diminish inputs that influence child development, including reduced reading and stimulation limiting language and cognitive development and, often, less value placed on education. Overall, these children frequently achieve lower educational outcomes and socioeconomic status (Larson, 2007). It is abundantly clear that poverty is a major determinant of the quality of health within and between developed and less developed countries (Stuart and Soulsby, 2011a).

The differences in health between those living with greater or lesser social and economic resources are marked, with a wide range of such inequalities influencing health and well-being. The social, psychosocial, material and biological factors all influence behaviours and position in society which, in turn, are modified by education, occupation, income and political beliefs. The term ‘structural violence’ is sometimes used in describing the social factors that cause harm to individuals and populations. This term was originally devised by Galtung (1969, cited by Farmer et al., 2006): they are described

as structural because they are embedded in the political and economic organization of our social world; they are violent because they cause injury to people. (Farmer et al., 2006: 1686)

In general, health care professionals are not prepared to deal with these factors which are primarily the responsibility of government and regional officials plus those working within the relevant local organisations. However, health professionals do need to be able to understand the relevance of these issues and collaborate with those responsible for these areas.

Challenges

Much of the effort in reducing health inequalities worldwide is about broadly identifying the issues involved and planning across a range of organisations. For example, housing, agriculture, social services, education, infrastructure and engineering, all involving finance, are outside the responsibility of health ministries. Although countries may organise them differently, they are usually within government departments without a health remit. However, planning to enhance the health status of a country has to take account of all these issues (Clift, 2013), which fall into a number of categories:

* **Infrastructure:** in considering developing countries, this group of concerns includes health-related developments but also more general concerns such as roads, dams and power stations, schools and universities.
* **Population growth:** uncontrolled population growth, with the related increased need for food, antenatal and postnatal care, increases the high risks associated with childbirth (see Chapter 18, Female reproductive system), especially in circumstances where midwives and obstetricians may be in short supply. Family planning is crucial.
• Agriculture and livestock: if agriculture and livestock are not managed effectively, then poverty will limit access to adequate nutrition, clean water and sanitation, and education. Livestock may be exposed to diseases, which limits healthy animals for nutrition and for providing a living for families (Stuart and Soulsby, 2011b).

Health disorders

Another major area in enhancing global health is collaboration and funding in dealing with various specific health disorders, falling into two main groups – infectious diseases and non-communicable disorders (Stuart and Soulsby, 2011c).

• Infectious diseases: these are caused by organisms (bacteria, viruses, fungi or parasites), many of which compose the microbiome living in or on our bodies and are mainly harmless. However, in certain conditions they can cause disease and be transmitted between individuals. Some conditions are known as zoonoses in which the infecting agent is transmitted from animals to humans: 61% of human pathogens are said to be zoonotic and up to 75% of recently identified pathogens are in this group (WHO, 2018e). Terms used to describe distribution include:
  - Endemic: a condition that is generally present in a group or area, such as a cold
  - Epidemic: a widespread distribution of a condition in an area at a specific time, such as influenza
  - Pandemic: a condition that spreads worldwide. When a new influenza virus emerges, it often spreads widely among populations, most of whom do not have immunity.

It is clear that the issue of promoting and maintaining public health within a country or worldwide requires the combined efforts of different organisations, both health services and those with wider responsibilities. Education and leadership within the different areas of activity are also essential and the qualities of fortitude and resilience are crucial.

Apply

Pandemic spread

The speed of spread of pandemics is strongly related to the transport methods used. Thus, the spread of plague was largely through ship travel and then horse-drawn travel on land and was thus fairly slow. Nowadays, air travel can facilitate rapid transport of infection around the world. It is crucial to understand the diseases which may become pandemic, e.g. Ebola, and how to screen travelers and implement quarantine methods as necessary.

Go Deeper

Non-communicable disorders

WHO estimates that this group of diseases make up 59% of the 56.5 million deaths on this planet per year (Marshall, 2004). It is creating a growing problem for middle and lower income countries to deal with while

(Continued)
still trying to meet the problems associated with infectious diseases. These non-communicable disorders include conditions related to obesity such as cardiovascular diseases, diabetes and cancers. Most of the cardiovascular diseases are related to major risk factors such as high cholesterol, high blood pressure, low fruit and vegetable intake, inactive lifestyle and tobacco use, and obesity is becoming a major problem in many developing countries. WHO’s strategy for tackling this worldwide problem recognises the importance of all sectors contributing, including governments, NGOs (non-governmental organisations), the private sector and stakeholders (including the food industry) (WHO, 2018b).

Communicable Disorder (Pandemic)

Probably the most well-known pandemic in human history is the plague, known as the Black Death, of the mid-14th century (1346–1353). It was caused by a bacterium (carried by rats), *Yersinia pestis*, named after Alexandre Yersin, who isolated the bacterium. It is transmitted by the carrier biting someone and regurgitating the gut’s contents into the bloodstream of the human. There have been a number of outbreaks of plague over the years and this bacterium is still found in the American Southwest and parts of Asia.

There are three variants of this disease:

- **Bubonic**: large swollen areas (buboes) appear around lymph nodes. 18% survival rate for individuals. Pus released is unpleasant to see and smell.
- **Pneumonic**: affected respiratory system. Easily transmitted between people. Patient drowns in own blood. Survival rate 1%, dies within 2 days.
- **Septicaemic**: least common form. Disseminated intravascular coagulation (DIC) occurs: blood clots and causes necrosis, then loses ability to clot properly.

This pandemic is said to have reduced the world population by about 50%. The social organisation of the population changed radically, with the size of the labouring population shrinking. Thus, the workers were able to be much more selective in the work they would undertake. The merchant class grew and became wealthier. The nobles became less wealthy and the noble and merchant classes began to intermarry (Armstrong, 2016).

It is thought that the disease originated in China and was transmitted around the world by sailing ships to sea ports. The bacteria were carried in fleas on rats and by jumping from rats to humans, transmitted to humans and carried inland. However, recently, it appears that the plague bacteria were carried by lice and human fleas (although further research is needed to confirm this) (Benedictow, 2004; Dean et al., 2018).

CHAPTER SUMMARY

In this chapter we have looked at two major concepts that influence person-centred care: epidemiology and public health. Epidemiology uses research methodology to provide the data to facilitate understanding of the context of health and disease, and to identify factors that influence the presentation of disease in populations. These data are used to enable public health services and approaches to promote health of populations through health protection, health improvement and service improvement. All these contribute to health promotion by preventing the key causes of ill-health, rather than just focusing on treatment and cure.
• Epidemiology plays an important role in identifying factors that influence health and disease and provides the understanding necessary to plan and implement conditions for optimum health for individuals and communities.

• There are three major areas of study in epidemiology: descriptive, analytical and intervention, with a number of sub-groups within them.

• Public health is underpinned by the science of epidemiology and it is defined by WHO as the science and art of preventing disease, prolonging life and promoting health through the organised efforts of society.

• Areas of public health practice are health protection, health improvement and service improvement, and health promotion is a key element in all three of these.

• The three key elements of health promotion have been identified by WHO as: good governance for health, literacy and healthy cities which together enable people to increase control over their own health.

• Worldwide public health requires the application of public health principles worldwide and is known as Global Health which aims to improve health and achieve health equity worldwide.

• Global health is achieved by acting within three main areas: inequalities in relation to socioeconomic status and poverty; challenges related to infrastructure, population growth and agriculture and livestock; and health disorders falling into two main groups – infectious diseases and non-communicable disorders.

**Key Points**

The content of this chapter will help you understand the principles of epidemiology underpinning public health, and approaches to enhancing the health of the public and individual communities. Revise the different sections in turn then try to answer the questions.

Answers are available online. If you are using the eBook just click on the answers icon below. Alternatively go to [https://study.sagepub.com/essentialpatho/answers](https://study.sagepub.com/essentialpatho/answers)

1. What is epidemiology and how does it contribute to public health?
2. Identify the main types of epidemiological studies and their contribution to an understanding of the distribution of disease.
3. Discuss what is meant by a double-blind controlled trial and their contribution to planning health care interventions.
4. Define and explain public health and the importance of the three areas of public health practice.
5. Explain why public health is valuable and evaluate five examples of public health achievements.
6. Analyse the importance of health promotion in influencing the health of the population.
7. Identify and evaluate the main elements of public health.
8. Discuss the importance of global health and the major groups of issues in promoting worldwide health.
REVISE
• Further revision and learning opportunities are available online
• Test yourself away from the book with Extra multiple choice questions
• Learn and revise terminology with Interactive flashcards

ACE YOUR ASSESSMENT

If you are using the eBook access each resource by clicking on the respective icon. Alternatively go to https://study.sagepub.com/essentialpatho/chapter2

REFERENCES


THE HUMAN CELL

UNDERSTAND: KEY CONCEPTS

Before working through this chapter, you might find it useful to watch these external video clips on the human cell.

The URLs for these videos can be accessed via the companion website https://edge.sagepub.com/essentialaandp.

LEARNING OUTCOMES

When you have finished studying this chapter you will be able to:

1. Describe the structure and functions of the different components of human body cells
2. Describe the two types of cell division: meiosis for the formation of gametes for reproduction, and mitosis involved in growth and development
3. Identify how different types of body cells are adapted for their different functions and how they interact synergistically
INTRODUCTION

In the previous chapter you were introduced to the systems of the multicellular body which supply the necessary conditions for life. It will make understanding the human body easier if you know rather more about how each cell works and this chapter focuses on the structure and function of the different types of cell in the human body.

The two types of cell division will also be examined:

- **mitosis** for growth and repair of tissues,
- **meiosis** for creation of sperm or ova (gametes) containing half the normal genetic material for formation of the next generation.

Context

During the life story of every individual the balance in the cells of which they are composed goes through various stages. Cell multiplication increases the number of body cells while apoptosis (programmed cell death) reduces this number. Both processes occur throughout life but the balance between them varies and permits normal growth and development.

The Bodie family members are at different stages of the life-cycle. Danielle, the baby, is growing fast with cell multiplication occurring rapidly but apoptosis (programmed cell death) contributing to shaping and reshaping of the body as she moves through infancy and childhood, adolescence and adulthood.

In the younger adults (Thomas (30), Derek (29), Michelle and Margaret (27), Kwame (28)), cell multiplication and cell death are roughly in balance, with a good state of health and physical strength. The older adults (Edward (57), Sarah (55), Hannah and Richard (both 54)), are likely to have moved towards a preponderance of cell death although they are still in general good health. Physical strength and health tends to be maintained in those who undertake regular exercise as Matthew does (45).

The grandparents (George (84) and Maude (77)) are very likely to have more cell death than cell multiplication occurring and their general health and tolerance for activity are probably deteriorating.

While this brief consideration of the Bodies provides some guidance, it is important to recognise the considerable variation between individuals in their physical status through the life span. Person-centred practice requires consideration of individual variation in all aspects of a person.

THE CELL

Introduction

This chapter focuses on the human cell and how it works. However, although not essential, it is interesting to understand where this fits in the wider biological context.

GO DEEPER

Types of living cells

There are three types of living cell, two prokaryotes and the eukaryotes:

**Prokaryotes** (before nucleus): there are two of these - bacteria and archaea, both of which are single-celled organisms without membrane-bound organelles (small organs) with specialist functions such as a nucleus, mitochondria, etc.:
**Bacteria (bacterium) – singular** very similar to archaea in size and shape;

**Archaea (archaeon) – singular** much of its metabolism is more similar to eukaryotes than bacteria.

**Eukaryotes:** have well-defined membrane-bound nuclei and organelles and form all the multicellular organisms on earth. Lane (2015) has proposed that these developed through endosymbiosis in which these complex cells arose from a unique merger of a bacterium into an archaeon resulting in the first eukaryote from which all others evolved. The bacterium replicated, transferred much of the surplus DNA (genetic material – see later in this chapter) to the host archaeal chromosomes, and developed into the mitochondria. The major benefit is the large amount of Adenosine Triphosphate (ATP) available from the mitochondria thus increasing the energy available and quantity of protein formed.

The human (mammalian) cell is a complex structure able to carry out all the functions required to maintain cell life and also makes its contribution to homeostasis through the activities of the different organelles (small organs) within the cell. Figure 2.1 illustrates a generic cell filled with the liquid cytoplasm and the range of organelles, and surrounded by the cell (plasma) membrane.

**Figure 2.1** A human cell

Table 2.1 identifies the different organelles and their functions within the cells of the body. The nucleus of the cell is unique to eukaryotes and contains the genetic material of the individual which determines the characteristics of each person. We are going to consider the nucleus first, including how it carries genetic information, and how the cells divide.

**THE NUCLEUS**

The nucleus is surrounded by a double membrane similar in structure to the cell membrane. The nucleus contains the genetic information which determines the constitution of the body in the 46
chromosomes (23 pairs). The nucleus also contains the nucleolus which is involved in the formation of ribosomes in the nucleus which are then moved out into the cytoplasm of the cell through the nuclear pores.

### Table 2.1 The functions of cell organelles

<table>
<thead>
<tr>
<th>Organelle</th>
<th>Functions</th>
</tr>
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</table>
| Cell (plasma) membrane (double membrane) | Controls movement of substances, ions and nutrients into, and waste products out of, the cell thus determining composition of cytoplasm (cell contents excluding organelles)  
Response to external stimuli                                               |
| Nucleus (double membrane)              | Contains genetic information within DNA of chromosomes of the cell; provides the template (RNA) for protein formation. Controls activities of the cell |
| Nucleolus                              | Made of protein and RNA and is in the nucleus. Synthesises and assembles ribosomes which leave the nucleus and enter the cytoplasm            |
| Mitochondria (double membrane)         | The ‘power houses’ of the cell. Glucose used as fuel to create ATP for storage of energy which is released when required               |
| Ribosomes                              | The protein factories of the cell, translate RNA into protein. When loose in cytoplasm, forms proteins for use within the cell             |
| Endoplasmic reticulum                  | Directs movements of lipids and proteins through the cell  
Synthesises lipids and steroid hormones  
Combined with some ribosomes – synthesises proteins for ‘export’ from the cell |
| Smooth                                 |                                                                                                                                         |
| Rough                                  |                                                                                                                                         |
| Golgi body (apparatus)                 | ‘Packages’ proteins within membrane as vesicles, stored, then exported through cell (plasma) membrane                                  |
| Lysosomes                              | Contain enzymes which break down unneeded large molecules which are recycled or excreted from cell  
One type digests foreign bodies such as microbes                               |
| Cytoskeleton                           | Microfilaments and microtubules form an internal framework of the cell allowing movement  
Centrosome and centrioles: play important role in cell division               |

### Chromosomes

The nucleus contains 44 autosomes (i.e. not sex chromosomes) and two sex chromosomes. Normally each body cell (except ova and sperm – the gametes involved in reproduction) contains the diploid number of 46 chromosomes composed of a haploid set of 23 from each parent.

**Understand**

- **Diploid**: two complete sets of chromosomes.
- **Haploid**: one complete set of chromosomes present in gametes.
The gametes are the specialist cells formed in the reproductive organs which combine at fertilisation to form the zygote. These differ in the two genders:

**Female:** Ovum (pl. ova);
**Male:** Spermatozoon (or sperm) (pl. spermatozoa).

Chromosomes differ in size and, when being examined, a cell is prepared so that the chromosomes are spread out in a smear (Figure 2.2a). The individual chromosomes are then cut out, paired up and laid out in order of size as a karyotype (Figure 2.2b) with the 22 pairs of autosomes followed by the two sex chromosomes at the end. The female karyotype has two X chromosomes, while the male has one X and one Y chromosome in each cell. Each pair of autosomes are called homologous chromosomes and are the same size and carry comparable genes for the same characteristics. The genes may not be identical and allele is the term used for an alternative form of the same gene located at the corresponding position on homologous chromosomes.

![Diagram of human chromosome](image)

**Figure 2.2** The human chromosome (a) Chromosome smear (b) Human karyotype

**Deoxyribonucleic acid (DNA)**

Chromosomes are composed of Deoxyribonucleic Acid (DNA). DNA acts as the genetic code which provides instructions for the formation of RNA (Ribonucleic Acid) which is transported out of the nucleus and provides a code for the formation of proteins (in collaboration with ribosomes). The proteins may form part of the structure of the cell or the matrix surrounding it, or are enzymes which act as catalysts for the range of chemical reactions in the cell.

Chromosomes are formed of two DNA strands with backbones of alternating sugar (deoxyribose) and phosphate molecules connected by pairs of nitrogen-containing bases (Figure 2.3a), two purine (adenine and guanine) and two pyrimidine (thymine and cytosine); a purine always connects with a pyrimidine. Adenine joins with thymine by two chemical bonds, and guanine connects with cytosine by three chemical bonds. The two chains form a double helix (Figure 2.3b) described by Watson and Crick in 1953.
Figure 2.3 (a) DNA structure – The sugar-phosphate backbone and pairing of the nucleotides

Figure 2.3 (b) DNA structure – DNA helix
In forming the chromosomes shown in Figure 2.3, the DNA helices are further coiled around histones (proteins), and coiled and folded yet further to create the demonstrable thickness of the chromosomes (Figure 2.4).

Condensed chromatin: transcriptionally inert

Open chromatin: transcriptionally active

Figure 2.4 Histone modification of DNA activity

---

**GO DEEPER**

**Cell division and ageing**

At the end of each DNA chain is a section called a telomere consisting of a repeated chain of nucleotides (in vertebrates this sequence is TTAGGG). These protect the ends from damage but shorten at each cell division placing a limit on the number of cell divisions that can occur and resulting in ageing (Allsopp et al., 1995). Human foetal cells divide between 40 and 60 times before the telomeres become too short for further division and the cells become quiescent and eventually die. This ageing of the cells correlates with overall ageing of the body. There are certain conditions of premature ageing in which this takes place more rapidly than normal (e.g. progeria).

The enzyme telomerase can lengthen the telomere and result in continued division. This is linked with development of cancer; metastases (i.e. secondary growths) often show telomerase activity (Turnpenny and Ellard, 2007).

Although rare, errors in DNA during cell division can occur and, without repair, it is suggested that 16,000 nucleotide errors could occur in a single cell (Westman, 2006). A number of different repair mechanisms exist to minimise the deleterious effects of such errors.

The chromosomes in the nucleus of the cell contain about 99.9% of the total DNA in the cell and hold information, half from each parent, determining the individual characteristics of the person. The remaining 0.1% of DNA is in the mitochondria (see below) and is concerned with energy metabolism (Chapter 9). Under all normal circumstances this is handed down only from the mother as the spermatozoon...
(male gamete) loses its tail and mid-piece (in which the mitochondria are arranged) in the process of entering and fertilising the ovum. Thus all mitochondria in the body develop from those in the ovum.

**Protein formation**

We have already indicated that the DNA in the chromosomes carries the genetic material that determines the individual’s characteristics. It does this by acting as a template for the formation of the proteins of the body from the amino acids, the nutrients absorbed into the body after the breakdown of proteins in the diet (Chapter 8). This determines both structure and function of the components of the body. The process occurs in the two main stages illustrated in Figure 2.5.

**Figure 2.5** Transcription (to RNA) and translation (to proteins)

**Transcription of DNA to Ribonucleic Acid (RNA)**

The bases within DNA are linked by weak bonds which allow separation of the DNA strands to permit formation of messenger RNA (mRNA) by transcription (Figure 2.6). The RNA is a single-strand molecule similar in structure to DNA except that the deoxyribose is replaced by ribose, and thymine is replaced by uracil. Each base in the RNA is complementary to the base in the DNA.

**Translation (to proteins)**

The bases in the DNA transcribed into RNA act as a code in which each group of three bases is code for a specific amino acid. The strand of messenger RNA (mRNA) is the template for translation.
of this code into a chain of amino acids forming a polypeptide chain which becomes a protein. The mRNA is carried out of the nucleus and joins with a ribosome (see below) in the cytoplasm where the translation takes place (Figure 2.7). Each group of three bases (called a codon) is translated into one amino acid picked up by transfer RNA (tRNA). Figure 2.8 shows the genetic code and illustrates how a number of codons can code for a single amino acid, or can code to stop or start the formation of the chain of amino acids.

Figure 2.6  Formation of mRNA from DNA

Figure 2.7  Translation of mRNA into polypeptide chain

The nucleolus

The nucleolus is the largest structure in the nucleus of eukaryotic cells. It is composed of proteins and RNA and its main function is to synthesise ribosomes and assemble them for export into the cytoplasm of the cell. It is also involved in how the cell responds to stress.
OTHER ORGANELLES

The cell or plasma membrane is double-layered and the nucleus and mitochondria are covered with similar membranes. Most of the other organelles are surrounded or formed from single-layered membranes.

Cell or plasma membrane

The boundary of each cell is a double-layered lipid membrane, the cell or plasma membrane, composed of phospholipids (fatty molecules with a phosphate group), proteins and carbohydrates arranged in a mosaic structure (Figure 2.9) (Chapter 8 includes the structure of different nutrients). The phosphate ends of phospholipids are attracted to water (hydrophilic) and they face outwards from the cell membrane while the fatty acid tails are water repellent (hydrophobic) and face each other in the centre of the membrane, preventing passage of all but very small molecules.

Some protein molecules are incorporated into one layer of the membrane while some pass through both layers and facilitate transport across the membrane. Proteins compose about 50% of the structure of these membranes, and are particularly important in transport of substances across the cell membrane. Proteins in combination with carbohydrates on the outside of the cell membranes can act as receptors by having a specific binding site where hormones or other substances can link. This initiates other actions in the cell.
The composition of the cytoplasm (intracellular fluid (ICF)) is very different from that of the Extracellular Fluid (ECF) surrounding it (Table 2.2). The concentration gradient of a substance sets the initial parameter, with substances moving from high to low concentration (Chapter 11). However, the plasma membrane plays a vital role in maintaining the differences between the interior and exterior of the cell through regulating the movement of substances in and out of the cell.

**Table 2.2 Ionic composition of cytoplasm (ICF) compared to extracellular fluid (ECF) (these values can vary depending on which type of cell is being looked at and results can vary somewhat in different laboratories)**

<table>
<thead>
<tr>
<th>Element</th>
<th>Ion</th>
<th>ICF</th>
<th>ECF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>Na⁺</td>
<td>15</td>
<td>141</td>
</tr>
<tr>
<td>Potassium</td>
<td>K⁺</td>
<td>140</td>
<td>4</td>
</tr>
<tr>
<td>Calcium</td>
<td>Ca²⁺</td>
<td>0.0001</td>
<td>2.5</td>
</tr>
<tr>
<td>Chloride</td>
<td>Cl⁻</td>
<td>8</td>
<td>103</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>HCO₃⁻</td>
<td>15</td>
<td>25</td>
</tr>
</tbody>
</table>

**Transport across the cell membrane**

The cell membrane controls how substances can move in and out of the cell and these are discussed in detail in Chapter 11. They include passive movement, in which molecules pass down a concentration gradient and no energy is required, and active movement, in which energy is required to move molecules against resistance.
- **Carrier proteins** facilitate the movement of specific molecules. These are proteins which pass through the cell membrane, and provide a site recognised by specific molecules which link to it. The protein then changes conformation and releases the molecule on the other side of the membrane (Figure 2.10). The proteins assist movement of substances by what is known as carrier-mediated transport requiring energy.

![Figure 2.10 Carrier proteins](image)

- **Ion channels** (Figure 2.11) determine the concentration of fluid and inorganic ions (which have a positive or negative charge) within the cytoplasm, including sodium (Na\(^+\)), potassium (K\(^+\)), chloride (Cl\(^-\)), bicarbonate (HCO\(_3^-\)) and calcium (Ca\(^{2+}\)). The distribution of ions within the Intracellular Fluid (ICF) and outside the cell in the ECF results in an electrical difference across the cell membrane (the electrical potential) with the inside of the cell being more negative than the outside.

![Figure 2.11 Ion channels](image)

- **Exocytosis and endocytosis:** these processes enable large molecules that cannot pass though the cell membrane to move between the ICF and ECF. Endocytosis enables molecules to enter
the cell by engulfing it (Figure 2.12a). Exocytosis enables the contents of a vesicle formed from the Golgi apparatus to fuse with the cell membrane and the contents are released from the cell (Figure 2.12b).

![Image](https://edge.sagepub.com/essentialaandp)

**Figure 2.12** Endocytosis and exocytosis

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**Activity 2.1: Understand**

Watch these two short video clips to see endocytosis and exocytosis in action. The URLs for these online videos can be accessed via the companion website https://edge.sagepub.com/essentialaandp.

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### Receptors and the cell membrane

The cell membrane also plays an important role in signalling between cells through the action of receptors. These are proteins embedded in the plasma membrane surrounding the cell, within the cell or in the nucleus which bind to chemicals from outside the cell called ligands. Receptors detect the specific molecules (ligands) to which they are sensitive, for instance a hormone (Chapter 7), neurotransmitter (Chapter 5), small protein, a drug or part of an infectious agent (bacterium or virus), and modify the activity of the target cell.

The receptors modify the activity of the target cells in one of four ways:

1. By opening an ion channel and thus modifying the action potential (i.e. the electrical difference across the membrane) which in nerve cells cause nerve impulses to pass along the cell (Chapter 5). In other types of cell they activate particular cellular processes.
2. By activating a membrane-bound receptor and initiating a particular metabolic pathway.
3. By activating a receptor that activates another protein in the membrane (a G protein). The G protein can have effects on the cell in one of two ways: either through influencing activity of the ion channels or by influencing the concentration of second messengers.
4. By activating an intracellular receptor to adjust the transcription of specific genes.

Receptors are discussed again in Chapters 5 and 7.
Major Histocompatibility Complex (MHC)/Human Leukocyte Antigen (HLA)

The Major Histocompatibility Complex (MHC) proteins (known as Human Leukocyte Antigens (HLAs) in humans) are based in the cell membrane and play an important role in protecting against external agents entering the body (e.g. bacteria or viruses) or abnormalities developing in cells (e.g. cancer). The defence of the body depends on the immune system (Chapter 13) being able to recognise the difference between ‘self’ and ‘non-self’ cells. The specific body cell proteins involved in this recognition are known as antigens.

During development the foetus develops antigens on its cells which act as markers for the ‘self’. However, each cell modifies itself in response to infection or by changes in the cell, becoming, for example, malignant, and binds fragments derived from the breakdown of these pathogens (infective agents). The cells of the body’s immune system recognise these not-self antigens and destroy these cells (Chapter 13).

Mitochondria

The mitochondria (singular mitochondrion) are surrounded by a bilipid layer similar to the plasma membrane and they act as the power houses of the cell. They metabolise nutrients to produce ATP (Adenosine Triphosphate) – the energy store of the cell – which is used to power the various cell activities (Chapter 9).

Mitochondria are positioned in cells according to the particular cell function and where energy is required. For example, in a cardiac muscle cell where energy is required all the time, the mitochondria are clustered near the muscle fibres.

Ribosomes

These granules are formed by the nucleolus within the nucleus of the cell and pass through the nuclear pores into the cytoplasm. They are composed of RNA and protein and those that are either loose or in groups within the cytoplasm form proteins for use within the cell. Other ribosomes combine with Endoplasmic Reticulum (ER).

Endoplasmic reticulum (ER)

ER is a cell-wide network of membrane which provides a surface on which lipids and proteins can be formed and transported round the cell.

Go Deeper

Cellular transport

ER forms transport vesicles to carry the substances formed between the ER, plasma membrane, Golgi apparatus and lysosomes. Special transfer proteins carry the same substances to mitochondria and lysosomes.

- Smooth ER is involved in the synthesis of steroid molecules (a type of lipid) in cells where these are produced, and in calcium storage. This is particularly important in muscle cells in which smooth ER is known as sarcoplasmic reticulum.
• **Rough ER** is combined with ribosomes and thus appears rough under the microscope. The ribosomes with the ER form proteins such as enzymes or hormones which are exported from the cell for use elsewhere in the body.

**Golgi apparatus or body**

The Golgi apparatus is more important and larger in cells which have a secretory role. It is composed of stacks of flattened sacs of membrane which receive proteins and lipids from the ER and package them into secretory vesicles. These are stored and moved to the plasma membrane when needed, and exported by exocytosis.

**Lysosomes**

These are one of the types of secretory vesicles created by the Golgi apparatus and essentially are bags of enzymes. These break down large molecules that are no longer required into smaller fragments which may be reused or eliminated from the cell. Those in white blood cells contain enzymes which break down microbes.

**Cytoskeleton**

As indicated in Table 2.1 this forms a framework for the cell and enables movement. The centrosome is the core from which single filaments radiate and enable movement of vesicles and organelles. The centrosome is near the nucleus and has two cylinders, centrioles, at right angles to each other. During cell division these separate and move to opposite poles of the nucleus (see Cell Division below).

**CELL DIVISION**

From the formation of the zygote, cell division continues to occur throughout life. There are two types of cell division: one which forms the gametes, called meiosis, and the other is mitosis, the cell division involved in growth and development.

**Meiosis**

Meiosis occurs in relation to reproduction and is the division that occurs to form the gametes – sperm or ova – in preparation for fertilisation and formation of the zygote which develops into the foetus. As already stated, the gamete from each parent normally contains half the full number of chromosomes (i.e. 23:22 autosomes and one sex hormone).

Figure 2.13 shows the stages involved in meiosis. Initially the DNA replicates so that each chromosome in a pair of homologous chromosomes consists of two chromatids. Some of the DNA often swaps over (chromosomal crossover) between the two chromosomes as shown in the different colours in the chromatids in meiosis I. The pairs of chromosomes separate into two daughter cells each with half the original number of chromosomes, but with the sister chromatids remaining together. During meiosis II the chromatids separate into two separate cells so that from the original single cell, there are now four daughter cells. These can all be somewhat different in their DNA structure. These cells then mature into the male sperm or a female ovum with three polar bodies (Figure 2.14) which usually die.
Homologous chromosomes  Sister chromatids  Homologues separate, sisters remain attached  Sisters separate

DNA Replication  Chromosome recombination  Chromosome segregation (meiosis I)  Chromosome segregation (meiosis II)  Gametes (ova or sperm)

Figure 2.13  Meiosis in formation of gametes

Figure 2.14  Formation of polar bodies

**Activity 2.2: Understand**

Go online and watch the following video clip to see meiosis in action.

To save time, this external video link can be accessed via the companion website https://edge.sagepub.com/essentialaandp.
Mitosis

After the merger of the ovum and the sperm in fertilisation, mitosis, the alternative type of cell division, occurs as the embryo grows and differentiates into the different cells and tissues making up the body. Mitosis is when a cell divides into two genetically identical daughter cells. Continued cell division occurs as the body develops through the stages of development discussed in Chapter 17. It also enables tissue repair.

Mitosis consists of five main stages shown in Figure 2.15 and described below.

- **Interphase:** occurs between cell divisions and for most of this time the cells continue to function. During part of this phase, each chromosome duplicates to form two chromatids tightly coiled around each other.
- **Prophase:** the two chromatids become visible. The centrioles separate and go to each end of the cell with the mitotic spindle of microtubules between. The nuclear membrane disappears.
- **Metaphase:** the chromatids line up along the equator of the spindle attached by their centromeres (where the DNA is constricted and the two chromatids join).
- **Anaphase:** the microtubules forming the spindle begin to contract and draw the two chromatids of a chromosome apart to the ends of the cell.
- **Telophase:** The mitotic spindle disappears, the chromosomes reform and the nuclear membrane reforms.

![Figure 2.15 Mitosis](image)

The original cell then divides into two daughter cells by the division of the cytoplasm and cell membrane and may re-enter the cell cycle. However, many specialised cells remain in interphase and undergo no further cell division.
Introduction

Following fertilisation, the cells initially created have the potential to differentiate into any type of body cell – they are known as stem cells and, while most become fixed as a particular type of cell, some retain their flexibility and continue in certain tissues as stem cells. Cells with the ability to develop into any of the cells which make up the body are known as pluripotent stem cells (e.g. embryonic stem cells); those which are more limited but able to form more than one cell type are known as multipotent (e.g. adult stem cells and cord blood cells).

All body cells contain the same set of instructions, the DNA, but the expression of those genes is altered to produce the different types of cell. Differentiation is not thought to involve loss of DNA but occurs by modifying the expression of genes either through epigenetic changes to the DNA (Chapter 3) or by environmental factors extrinsic to the cell such as specific small molecules, secreted proteins from other cells, temperature and oxygen. The changes in gene expression can turn genes on or off or adjust the level of transcription of RNA and formation of proteins (Ralston and Shaw, 2008).

Cell differentiation produces five main types of body cell within the different tissues of the body:

- Blood and lymph,
- Connective tissue,
- Nervous tissue,
- Muscle tissue,
- Epithelial tissue.

Nervous and muscle tissue are both excitable tissues.

Blood and lymph

These differ from the other tissues in that the cells are not combined to form solid structures but are dispersed and transported in liquid. Table 2.3 outlines the major information about formation and functions of these tissues, which are sometimes included as connective tissues. The cells within these systems are formed from pluripotent stem cells, originally present in the embryo and capable of forming any type of body cell, which create the two multipotent stem cells from which the range of blood cells are formed (Figure 2.16).
**Table 2.3 Components of blood and lymph**

<table>
<thead>
<tr>
<th>Constituent</th>
<th>Formation</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood</td>
<td>Carried in blood vessels: arteries, capillaries and veins</td>
<td>Transport medium</td>
</tr>
<tr>
<td>Plasma</td>
<td>Water containing ions, plasma proteins, nutrients and waste products</td>
<td>Supplies all organs and tissues. Carries nutrients, waste products, blood gases, blood cells</td>
</tr>
<tr>
<td>Erythrocytes (red blood cells)</td>
<td>Formed in bone marrow, require iron and vitamin B_{12}. Have no nucleus. Life span 120 days</td>
<td>Transport O_{2} to cells of the body, and some CO_{2} to the lungs in haemoglobin of red blood cells (RBC)</td>
</tr>
<tr>
<td>Platelets (thrombocytes)</td>
<td>Cell fragments, formed in bone marrow</td>
<td>Initiate haemostasis (blood clotting)</td>
</tr>
<tr>
<td>Leucocytes (white blood cells)</td>
<td>Formed in bone marrow or lymphoid tissue</td>
<td>See below</td>
</tr>
<tr>
<td>Lymph</td>
<td>From tissue fluid from plasma, carried in lymphatic vessels. Returns fluid to blood circulation</td>
<td></td>
</tr>
<tr>
<td>Lymphatic fluid</td>
<td>Clear fluid formed in the tissues</td>
<td>Transports substances between blood and body cells. Carries leucocytes</td>
</tr>
<tr>
<td>Leucocytes</td>
<td></td>
<td>Combat infection, foreign bodies, malignant cells</td>
</tr>
<tr>
<td><strong>Agranulocytes:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Monocytes</td>
<td>Formed in bone marrow</td>
<td>Phagocytic – engulf bacteria</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>Formed in bone marrow, lymphoid tissue, thymus, spleen</td>
<td>Produce antibodies (B Lymphocytes)</td>
</tr>
<tr>
<td>Granulocytes:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basophils</td>
<td>Formed in bone marrow</td>
<td>Produce histamine and heparin</td>
</tr>
<tr>
<td>Eosinophils</td>
<td></td>
<td>Congregate at inflammation, antihistamine properties</td>
</tr>
<tr>
<td>Neutrophils</td>
<td></td>
<td>Phagocytic, become macrophages when migrate to tissues</td>
</tr>
</tbody>
</table>

All organs of the body are supplied with blood through the circulation (Chapter 12). Fluid from the blood enters the tissue spaces to provide the cells with the necessary nutrients and remove waste products. Some of the tissue fluid re-enters the circulation via the capillaries. Excess tissue fluid enters the lymph channels, passes through lymph nodes where it acquires additional lymphocytes, and returns to the circulation at the subclavian vein (Chapters 12 and 13).
Connective tissue

These tissues are also known as structural tissues as they provide structural support for the organs of the body. They vary considerably in appearance but all consist of cells, inter-cellular substance (matrix) and fibres, the last two being manufactured by the cells. The cells in connective tissue are much more spread out than in other tissues and there is considerably more matrix than in other tissues. The matrix and fibres vary according to the type of tissue and its function, major functions being structural support, protection, transport and insulation. The types of cells in connective tissue are given in Table 2.4.

The different tissues which these cells can form are specified in Table 2.5.
### Table 2.4 Cells in connective tissue

<table>
<thead>
<tr>
<th>Cells</th>
<th>Description</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibroblasts: large flat cells</td>
<td>Produce extracellular matrix and fibres:</td>
<td>Major role in tissue repair</td>
</tr>
<tr>
<td></td>
<td>Collagen - minimal stretch coarse fibres in wavy bundles</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Elastin - fine branching elastic fibres</td>
<td></td>
</tr>
<tr>
<td>Adipocytes: vary in size and shape</td>
<td>Singly or grouped in most connective tissues abundant in adipose tissue</td>
<td>Major energy store as fat in adipose cells</td>
</tr>
<tr>
<td>Macrophages: irregular shape</td>
<td>Some fixed, some mobile. Phagocytic - engulf and digest cell debris, bacteria, foreign bodies</td>
<td>Part of immune system</td>
</tr>
<tr>
<td>Leucocytes: white blood cells</td>
<td>Small numbers in healthy connective tissue</td>
<td>Raised numbers in infection</td>
</tr>
<tr>
<td>Mast cells: like basophils</td>
<td>In loose connective tissues and fibrous capsule of some organs, around blood vessels</td>
<td>Histamine, heparin, etc., released after damage</td>
</tr>
</tbody>
</table>

### Table 2.5 Connective tissues

<table>
<thead>
<tr>
<th>Type of tissue</th>
<th>Subtypes</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loose (areolar) connective tissue</td>
<td>Most generalised type</td>
<td>Connects and supports other tissues through elasticity (elastin fibres) and tensile strength (collagen fibres)</td>
</tr>
<tr>
<td>Adipose tissue</td>
<td>• White adipose tissue</td>
<td>Is 20-25% of (non-obese) body under skin, stores fat around kidneys and other organs, acts as insulation and energy store</td>
</tr>
<tr>
<td></td>
<td>• Brown adipose tissue</td>
<td>Has substantial blood supply. Maintains body temperature by producing considerable heat</td>
</tr>
<tr>
<td>Dense connective tissue</td>
<td>• Fibrous tissue</td>
<td>Bundles of collagen fibres with little matrix. Forms ligaments to bind bones together. Protective covering for: bones, some organs (e.g. kidney, brain) Muscle sheaths (or fascia) become tendons (Chapter 15)</td>
</tr>
<tr>
<td></td>
<td>• Elastic tissue</td>
<td>Can stretch and recoil, few cells, much elastic tissue secreted by fibroblasts in organs which need to stretch or change shape (e.g. blood vessels, lungs, trachea)</td>
</tr>
<tr>
<td>Lymphoid tissue</td>
<td>Or reticular tissue</td>
<td>See Chapter 13. Internal protection: immune system</td>
</tr>
<tr>
<td>Cartilage</td>
<td>• Hyaline cartilage</td>
<td>Solid and smooth bluish-white matrix, cells in small groups Flexible, supportive and smooth for movement at joints: ends of long bones, costal cartilages join ribs to sternum, parts of airway</td>
</tr>
<tr>
<td></td>
<td>• Fibrocartilage</td>
<td>Contains dense collagen fibres (see above) within matrix like hyaline cartilage. Tough and flexible: intervertebral discs, in knee, hip and shoulder joints, forms ligaments joining bones</td>
</tr>
<tr>
<td></td>
<td>• Elastic fibrocartilage</td>
<td>Consists of yellow elastic fibres in solid matrix. Supports and maintains shape (e.g. lobe of ear)</td>
</tr>
<tr>
<td>Bone</td>
<td>• Compact bone</td>
<td>A very dense hard tissue (Chapter 15)</td>
</tr>
<tr>
<td></td>
<td>• Cancellous (spongy) bone</td>
<td>A spongy bone tissue (Chapter 15)</td>
</tr>
</tbody>
</table>
Adipose tissue

Adipose tissue has long been known as an energy source for the body and as having an important role in maintaining body temperature. Adipose tissue is composed of adipocytes, specialist cells that store fat. There are two types of adipose tissue, white and brown. White fat contributes to temperature regulation by reducing heat loss, and brown fat, with a considerably greater blood supply, produces heat through metabolism. The recognisable deposits of brown fat are particularly important in maintaining body temperature in babies as they generate heat and until fairly recently were not thought to exist in adults.

**GO DEEPER**

**Brown adipose tissue**

It has now been confirmed that precursor cells for brown adipose tissue are present in the supraclavicular regions (and possibly in other areas) of adults and that, under cold conditions, these can convert into active brown adipose tissue. Lean men or those working in cold conditions have more brown fat than those not working in such conditions, or those who are obese or overweight (Lee et al., 2011, 2014).

White adipose tissue is more prevalent in adults and also acts as an endocrine organ secreting the hormone leptin which influences food intake by reducing appetite and fat storage (Chapters 7 and 9). Interestingly, we never lose adipocytes, we only regulate the amount of fat stored in them. If there is too much fat for the number of adipocytes, we replicate adipocytes to increase available storage.

**Cartilage**

Cartilage is firmer than other connective tissues with fewer cells (chondrocytes) within a substantial matrix. Details of the three types are given in Table 2.5 and they vary due to the presence or absence of particular fibres (Figure 2.17).

![Figure 2.17 Cartilage: (a) hyaline, (b) fibrocartilage and (c) elastic fibrocartilage](image-url)
Bone

Bone is the main component of the skeleton and the detailed structure of bone and its repair following injury are discussed in Chapter 15. The main functions are to provide the basic framework of the body, protection of organs within spaces in the skeleton, and formation of blood cells.

Nervous tissues

Nerve and muscle are both excitable tissues meaning that the cells respond to chemical, electrical or mechanical stimulation. Nerve cells respond by transmitting a nerve impulse to carry instructions to or from the central nervous system (Chapter 5). Muscle cells respond by contracting (Chapter 15).

Muscle tissue

There are three types of muscle tissue – details are given in Table 2.6. They are discussed in relation to the systems with which they are concerned in the following chapters:

- **Striated**: Chapter 15 – Musculoskeletal System;
- **Non-striated**: Chapter 8 – Gastrointestinal Tract;
- **Cardiac**: Chapter 12 – Cardiovascular and Lymphatic Systems.

<table>
<thead>
<tr>
<th>Table 2.6 Types of muscle tissue</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Skeletal muscle</strong></td>
</tr>
<tr>
<td>Location</td>
</tr>
<tr>
<td>Appearance</td>
</tr>
<tr>
<td>Nucleus</td>
</tr>
<tr>
<td>Striations</td>
</tr>
<tr>
<td>Neural control</td>
</tr>
<tr>
<td>Function</td>
</tr>
</tbody>
</table>

Epithelial tissue

These tissues cover the body and line cavities, organs and hollow organs, and line glands. Their main functions are:
There are numerous types of epithelium (Table 2.7) but they share certain characteristics:

- Cells are tightly joined together by specialist cell-to-cell junctions to form a continuous sheet called epithelium (pl. epithelia);
- These cells lie on a basement membrane of connective tissue fibres which supports and separates the epithelium from the underlying tissue;
- Continual cell division takes place to replace any dead and damaged cells, which occur as skin and gut lining are continually abraded;
- The epithelial sheets enable transport of substances in a particular direction either into or out of the compartment as functionally necessary.

**Table 2.7 Epithelial tissues of the body**

<table>
<thead>
<tr>
<th>Type of Tissue</th>
<th>Position in Body/Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simple epithelia</td>
<td>walls of lung alveoli&lt;br&gt;Endothelium of blood vessels</td>
</tr>
<tr>
<td>Squamous (pavement): exchange of small molecules between compartments</td>
<td>walls of lung alveoli&lt;br&gt;Endothelium of blood vessels</td>
</tr>
<tr>
<td>Cuboidal (cubical)</td>
<td>Small collecting ducts in kidney</td>
</tr>
<tr>
<td>Columnar: secretion or absorption</td>
<td>Large collecting ducts of kidney&lt;br&gt;Lining of small intestine</td>
</tr>
<tr>
<td>Ciliated: waft materials along surface</td>
<td>Lining of Fallopian tubes and respiratory tract</td>
</tr>
<tr>
<td>Stratified epithelium</td>
<td>Lines upper airways</td>
</tr>
<tr>
<td>Pseudostratified columnar ciliated</td>
<td>Lines upper airways</td>
</tr>
<tr>
<td>Stratified squamous: waterproof and keratin barrier to bacteria</td>
<td>Epidermis of skin</td>
</tr>
<tr>
<td>Transitional</td>
<td>Bladder lining</td>
</tr>
<tr>
<td>Glandular epithelium</td>
<td>Epithelial surface of airways and gut</td>
</tr>
<tr>
<td>Goblet cells: secrete mucus</td>
<td>Epithelial surface of airways and gut</td>
</tr>
<tr>
<td>Endocrine: secretions enter bloodstream</td>
<td>Hormones (e.g. thyroxine, insulin)</td>
</tr>
<tr>
<td>Exocrine: secretions via duct onto surface</td>
<td>e.g. salivary and sweat glands and intestinal lining</td>
</tr>
<tr>
<td>Serous membranes</td>
<td>Pleura, pericardium, peritoneum</td>
</tr>
<tr>
<td>Double layer loose areolar connective tissue lined with simple squamous epithelium</td>
<td>Pleura, pericardium, peritoneum</td>
</tr>
<tr>
<td>Serous fluid between two layers</td>
<td>Pleura, pericardium, peritoneum</td>
</tr>
</tbody>
</table>

A range of epithelial tissues and glands are illustrated in Figure 2.18. These include single layer and multiple layer tissues including transitional epithelium which lines the bladder and can stretch to allow increased storage of urine. The figure of the stratified epithelium of the skin shows how the outer layers become thinned and then fall away. The glandular epithelium consists of unicellular or multicellular glands. Goblet cells secrete mucus, a slippery substance which lubricates the tissues of mucous membranes. The multicellular glands can secrete a number of different substances including sebaceous or intestinal secreretions.
Figure 2.18 Epithelial tissues and glands
Mucus (noun): viscous, slippery substance secreted as a protective lubricant coating cells and glands of the mucous membranes.

Mucous (adjective): means containing, producing, or secreting mucus.

CONCLUSION

This chapter has provided the foundation about human cells and how they work, multiply and differentiate into different types of cell which you can refer back to while you learn about the systems in which these cells function in later chapters. In relation to person-centred practice, this chapter provides some of the basic knowledge for you to understand how the human body works, and how that relates to psychological well-being.

Key points

- Understanding the structure of the human cell and how it works will help you to understand the human body as a whole.
- There are two types of cell division:
  - Meiosis occurs to prepare the gametes with the haploid number of chromosomes. When a sperm fertilises the egg the zygote then has the diploid number of chromosomes and further cell division is mitosis.
  - Mitosis forms two identical daughter cells and is the key to growth and development and is linked with cell differentiation to form the different types of cells and tissues forming the body: blood and lymph, connective tissue, nervous and muscle cells, epithelial cells.
- Understanding the function of the different cells of the body and how they interact synergistically will enable you to understand their contribution to homeostasis.

REVISE

This chapter and the previous one provide an underpinning for the remaining chapters in this book, so it is important to ensure that you have a good understanding of the content. In revising this chapter, it is useful to work through from the beginning. The areas to revise are as follows:

1. All the cell organelles and their functions, including transport across the cell membrane, DNA and RNA.
2. Meiosis - cell division for gamete formation; Mitosis - cell division for growth and repair.
3. The different types of cells and tissues: blood and lymph, connective, nervous and muscle (excitable) tissues, epithelial tissues.
4. The role of apoptosis in human development and functioning.
5. How this knowledge can be used in achieving person-centred practice.

In order to help you revise, consider the following questions, answers for which can be found by visiting https://edge.sagepub.com/essentialaandp. Test yourself by revising the chapter first, and then answering
these questions without looking at the book. Afterwards compare your answers with the text and with the notes you made. Did you miss anything in your notes? Here are the questions:

1. Identify the different organelles in the human body and state their functions.
2. Outline the stages of mitosis and meiosis.
3. Identify the five types of human cells, outline the different variants within each group and clarify their functions.

For additional revision resources visit the companion website at: https://edge.sagepub.com/essentialaandp.

- Revise key terms with interactive flashcards
- Test yourself with multiple-choice questions
- Access the glossary with audio to hear how complex terms are pronounced
- Print out or download the key points from the chapter for quick revision
- Explore recommended websites suitable for revision.

REFERENCES

Chapter 3  Infection

Chapter aims

After reading this chapter you will be able to:

• describe the main types of microorganism important for human health;
• explain the difference between pathogenic and commensal microorganisms;
• explain the chain of infection and how it may be broken;
• relate the signs and symptoms of infection to the underlying pathophysiology of infection;
• explain how the main anti-microbial drugs work and their main side-effects;
• demonstrate awareness of antibiotic resistance and the steps that can be taken to reduce the risk of antibiotic resistance developing.

Introduction

Imagine a world in which it is too dangerous to go into hospital for surgery and just cutting your finger could result in death. It seems ridiculous yet according to the World Health Organization (WHO) the threat from bacterial infection due to increasing resistance to antibiotics is making this a real possibility (WHO, 2015).

In this chapter we examine four of the main types of microorganisms important to human health and give examples of the infectious diseases they cause. The microorganisms we have included are: viruses, fungi, protozoa and bacteria. We will examine how microorganisms are passed on in ‘the chain of infection’ and how this chain may be broken. We then look at the general signs and symptoms of infection and how these are caused. We will consider the drugs used to treat the different types of infections and the side-effects of these. Finally, we will examine how antibiotic resistance occurs and what steps you can take to reduce antibiotic resistance developing.
Infection

Statistics from the World Health Organization for 2000–2016 place infectious diseases among the top ten causes of death worldwide (WHO, 2018). These infections include lower respiratory tract infections, diarrhoeal diseases and tuberculosis. In low income countries lower respiratory tract infections and diarrhoeal diseases are the top two causes of death. In high income countries, such as the United Kingdom, people predominantly die of chronic diseases, such as cardiovascular disease, cancer and dementia. However, lower respiratory tract infections remain a leading cause of death.

Infectious diseases are caused by microorganisms including bacteria, viruses and fungi. Disease-causing microorganisms are said to be pathogenic. A microorganism that causes disease is called a pathogen. Infection arises when a microorganism enters the body, resists the innate defences, and invades the tissues. The most common sites of infection are the respiratory and gastrointestinal tracts. The skin can be a site of entry – usually when it is disrupted, for example by a wound. Another important site of entry is the genitourinary tract. Pathogens can spread from person to person, directly or indirectly, or from animals to humans in what is called a zoonotic disease. Salmonella food poisoning is a classic example of a zoonotic disease, originating from contaminated food, often poultry.

Not all infections with pathogenic microorganisms cause symptoms. An infection is said to be subclinical if it produces no symptoms. Infection will result in disease and clinical symptoms if the infection causes tissue injury. Some microorganisms such as viruses cause direct damage to cells because they multiply inside our cells. Many viruses then spread by rupturing or ‘bursting’ the cell and spreading through the tissues or bloodstream (see below under ‘Viruses’). Some bacteria produce toxins which kill or damage cells and tissues. An example is Vibrio cholera. This microorganism produces a toxin that damages the gastrointestinal tract leading to severe diarrhoea. However, our immune response and inflammatory responses to invading pathogens can cause additional tissue damage.

The microbiome

In a healthy person, the internal tissues are normally free of microorganisms. The skin and parts of the body connected to the external environment (for example, the mouth, nose, intestinal and genitourinary tracts) become colonised by microbial species soon after birth. These organisms make up what is called the microbiome. The microorganisms making up the microbiome are commensal, which means they live harmlessly in or on the body. The microbiome prevents more pathogenic microorganisms colonising and infecting us. The microbiome includes some fungi, viruses and protozoa but is mainly made up from bacteria (Goering et al., 2013). The microbiome can contain pathogenic species. For example, E. coli bacteria usually live harmlessly in
the gastrointestinal tract but can cause urinary tract infections (UTIs) if they enter the urinary system. Wounds may become infected by microorganisms from the skin microbiome. **Immunocompromised** people (those with a weakened immune response) are particularly susceptible to infections and these infections often originate from the microbiome.

**Activity 3.1 Reflection**

Identify five infectious diseases. For each disease write down whether it is caused by a virus, bacteria, fungi or protozoa. Name the microorganism that causes the disease.

*Examples are given at the end of the chapter. As you proceed through the chapter, compare your choices with the infectious diseases discussed.*

Activity 3.1 will have enabled you to connect five infectious diseases with the type and name of the pathogen responsible. We now examine four main types of microorganisms that can cause disease in humans.

**Bacteria**

Bacteria are **prokaryotic** organisms. Prokaryote means ‘lacking a nucleus’ (Figure 3.1). Bacteria are all single-celled organisms, and although they do not have a nucleus, they do have genetic material in the form of DNA (deoxyribonucleic acid). Bacteria have their own cellular ‘machinery’ to grow and reproduce. This cellular machinery includes enzymes, and **ribosomes** needed to manufacture proteins. By contrast, **eukaryotic** cells, which includes all human cells, have a more complex structure. Human cells have a cell **nucleus** and other **organelles**, such as lysosomes (Chapter 2). The cellular machinery of human cells is different from that of bacteria. The difference in cellular machinery of human and bacterial cells enables the effective use of antibacterials. When we take antibacterial drugs, orally for example, the drugs circulate throughout the body. However, antibacterial drugs target the bacteria’s unique cellular components and machinery without affecting the cellular components of our own cells. This helps reduce the number of side-effects. Antibacterial drugs are explained further later in the chapter.

Bacteria are free-living organisms. ‘Free-living’ means that bacteria can live independently of the human body (or any other host organism). Most bacteria do not cause human disease but approximately 50 pathogenic species of bacteria exist. As mentioned under ‘The microbiome’, pathogenic species of bacteria may live harmlessly as part of the microbiome of the skin, mouth or intestines. Disease may occur if they invade our body tissues to cause damage. Some clinically common bacteria are identified in Table 3.1.
**Figure 3.1** A bacterium

<table>
<thead>
<tr>
<th>Name of bacterium</th>
<th>Disease expression</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td>Skin infections, pneumonia, sepsis, endocarditis</td>
</tr>
<tr>
<td><em>Clostridium difficile</em></td>
<td>Diarrhoea, pseudomembranous colitis</td>
</tr>
<tr>
<td><em>Klebsiella pneumoniae</em></td>
<td>Pneumonia</td>
</tr>
<tr>
<td><em>Escherichia coli</em></td>
<td>Diarrhoea, urinary tract infections, respiratory disease, sepsis</td>
</tr>
<tr>
<td><em>Pseudomonas aeruginosa</em></td>
<td>Sepsis, wound and burn infections</td>
</tr>
<tr>
<td><em>Bacteroides fragilis</em></td>
<td>Gastrointestinal upset</td>
</tr>
<tr>
<td><em>Mycobacterium tuberculosis</em></td>
<td>Tuberculosis</td>
</tr>
</tbody>
</table>

**Table 3.1** Selected human bacterial diseases

Table 3.1 shows that the same bacterial species may be responsible for more than one type of ‘disease expression’. The disease expression refers to the nature of the disease caused (for example, skin infection or respiratory disease), and depends on the part of the body that is infected.

**Viruses**

Viruses are one of the simplest types of microorganism. Viruses are smaller and simpler than cells. Viruses are not free-living and need to infect our cells to multiply. Viruses are described as ‘intracellular parasites’.

Figure 3.2 shows the structure of the human immunodeficiency virus. The simplest viruses are particles made of a protein ‘coat’ or capsid, containing the genetic material. The genetic material can be RNA (ribonucleic acid), or DNA (deoxyribonucleic acid), depending on the type of virus. Some virus particles are more complex and have an envelope surrounding the capsid. For example, HIV and influenza virus are more complex viruses with envelopes. The envelope comes from the host cell membrane as the virus particles are released. The envelope contains viral envelope proteins which are needed for cell infection, and are also important targets of the host immune response (Chapter 4). Virus particles are called virions.
A single virus particle is shown to attach to the host cell membrane. This will result in the virus particle being brought into the cell by a process called endocytosis. Once inside the cell cytoplasm, the virus’s genetic material, which in this case is made of a single molecule of RNA (ribonucleic acid), is copied many times. At the same time this genetic material is used to make the proteins making up the virus particle. The proteins are manufactured using the host cell machinery. The virus proteins and genetic material are then brought together to make many thousands of new viruses. These are released from the cell.

Figure 3.3  Virus replication within a host cell
Viruses lack the cellular ‘machinery’ (such as ribosomes) needed to convert their genetic material into new virus particles. Viruses are therefore reliant on a host cell which they infect in order to make more copies of themselves (‘replicate’). This makes antiviral therapy difficult to develop as any antiviral agent that stops viral replication will also affect the host cell function. Antiviral therapy is considered later in the chapter. The virus life cycle is shown in Figure 3.3.

Viruses show ‘host cell specificity’. This means that they can only usually infect one cell type. Virus infection involves attachment of the virus particle to a cell membrane receptor on the host cell. This interaction needs a specific protein on the surface of the virus particle and a target receptor on the host cell. Only those host cells with the ‘matching’ receptor for the virus surface protein can be infected. For example, HIV uses the virus membrane protein gp120 (Figure 3.2) to infect white blood cells which have a target receptor called CD4. This restricts HIV to infecting only cells with the CD4 receptor on the surface. These cells include T helper lymphocytes and macrophages (Chapter 4). Influenza virus uses the sialic acid receptor which is found on lung epithelial cells and the upper respiratory tract.

Some examples of clinically important viruses are given in Table 3.2.

<table>
<thead>
<tr>
<th>Viral pathogen</th>
<th>Disease expression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Herpes simplex type 1</td>
<td>‘Cold sores’</td>
</tr>
<tr>
<td>Herpes simplex virus type 2</td>
<td>Genital herpes</td>
</tr>
<tr>
<td>Human papilloma virus 6, 11, 16, 18</td>
<td>Warty growths, cervical carcinoma</td>
</tr>
<tr>
<td>Hepatitis B virus</td>
<td>Hepatitis</td>
</tr>
<tr>
<td>Rhinovirus</td>
<td>Upper respiratory tract infections</td>
</tr>
<tr>
<td>Varicella-zoster virus</td>
<td>Chickenpox, shingles</td>
</tr>
<tr>
<td>Norovirus</td>
<td>Acute gastroenteritis</td>
</tr>
<tr>
<td>Human immunodeficiency virus</td>
<td>Acquired immunodeficiency disease (AIDS)</td>
</tr>
</tbody>
</table>

*Table 3.2 Selected human viral diseases*

**Fungi**

Most fungi are multicellular organisms. Not all fungi are considered to be microorganisms. Many fungi grow as thread-like filaments. Other well-known types include single-celled yeast and mushrooms. Fungi are free-living organisms and are important ecologically for breaking down dead plant material. Pathogenic fungi invade tissues and cause damage by releasing digestive enzymes.

Examples of clinically important fungi that cause disease are given in Table 3.3.

Diseases associated with fungal infections are mostly seen in those with compromised immune systems. For example, patients on oral steroids (Chapter 2) or people with
a pre-existing disease may have weakened immunity. Many fungi cause opportunistic infections. These are infections that occur more frequently in those with weakened immune systems. People with healthy immune systems do not usually develop disease when exposed to these pathogens. Oral thrush (candidiasis) is a common side-effect of chemotherapy. Opportunistic infections are the common cause of death for those people with AIDS (Chapter 4).

<table>
<thead>
<tr>
<th>Fungal pathogen</th>
<th>Disease expression</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Candida albicans</em></td>
<td>Thrush</td>
</tr>
<tr>
<td><em>Aspergillus fumigatus</em></td>
<td>Lung infection in immunocompromised patients</td>
</tr>
<tr>
<td><em>Trichophyton interdigitale</em></td>
<td>Athlete’s foot</td>
</tr>
<tr>
<td><em>Pneumocystis jirovecii</em></td>
<td>Pneumonia in immunocompromised patients</td>
</tr>
</tbody>
</table>

*Table 3.3  Selected human fungal diseases*

**Protozoa**

Protozoa are single-celled animals. Many are free-living but some are important parasites of humans. Some free-living species infect humans as opportunistic infections if the person’s immune system is weakened.

Examples of clinically important protozoa are shown in Table 3.4.

<table>
<thead>
<tr>
<th>Protozoan pathogen</th>
<th>Disease expression</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Cryptosporidia parvum</em></td>
<td>Cryptosporidiosis</td>
</tr>
<tr>
<td><em>Giardia lamblia</em></td>
<td>Giardiasis</td>
</tr>
<tr>
<td><em>Toxoplasma gondii</em></td>
<td>Toxoplasmosis</td>
</tr>
<tr>
<td><em>Entamoeba histolytica</em></td>
<td>Amoebic dysentery</td>
</tr>
<tr>
<td><em>Plasmodium vivax</em></td>
<td>Malaria</td>
</tr>
</tbody>
</table>

*Table 3.4  Selected human protozoan diseases*

**The chain of infection**

The chain of infection (Figure 3.4) illustrates the stages needed for microorganisms to be passed on from one individual to another and cause disease. Prevention of infection is one of the most important roles of nurses and other healthcare professionals. By being aware of each potential link in the chain, infection may be reduced or eliminated within the clinical setting.

Terms used in the chain of infection are given in Table 3.5.
For example: methicillin-resistant *Staphylococcus aureus* (MRSA) is an example of an organism. A reservoir might be a person who is either colonised or infected with MRSA. The portal of exit might be contaminated body fluids such as wound exudate from an infected wound. Mode of transmission might be hands or contaminated equipment. The portal of entry in this case would be by a wound that another person has. That person is the susceptible host. From this scenario we can see that handwashing could break this chain at more than one point.

For further information on the chain of infection, please see the further reading section at the end of this chapter.

**Figure 3.4** Chain of infection

<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Organism</td>
<td>The pathogenic microorganism</td>
<td><em>E. coli</em>, <em>C. difficile</em>, MRSA (meticillin-resistant <em>Staphylococcus aureus</em>)</td>
</tr>
<tr>
<td>Reservoir</td>
<td>Source of the infection</td>
<td>A patient/health professional, animal, equipment, food, water</td>
</tr>
<tr>
<td>Portal of exit</td>
<td>How the microorganism leaves the body of the host</td>
<td>Faeces, urine, aerosols, droplets from respiratory tract (coughing/sneezing), vomit, blood, wound drainage</td>
</tr>
<tr>
<td>Transmission</td>
<td>How the microorganism is passed on</td>
<td>Direct contact, airborne</td>
</tr>
<tr>
<td>Portal of entry</td>
<td>How the microorganism enters the body</td>
<td>Break to the skin (wound), injection, needlestick injury, scalp, animal bite, catheter, mucous membranes</td>
</tr>
<tr>
<td>Vulnerable hosts</td>
<td>The person who becomes infected</td>
<td>Any susceptible person, elderly, pre-existing disease, immunocompromised</td>
</tr>
</tbody>
</table>

**Table 3.5** Terms used in the chain of infection

In the next section we will look more closely at bacterial infection.
Bacterial infection

Scenario

You are the staff nurse on duty when George is admitted to the ward. He is a 72-year-old man who lives alone at home. He is confused and disorientated on admission and has a respiratory rate of 35 breaths per minute. He has a temperature of 38°C and has been expectorating (‘coughing up’) yellow sputum. He is diagnosed with probable community acquired pneumonia. Sputum samples are taken and empirical antibiotic treatment with amoxicillin is started.

In this scenario, George’s symptoms and history have led to a diagnosis of community acquired pneumonia. A ‘best-guess’ approach is used to consider which microorganism may have caused the infection. Then an antibiotic is selected which is most likely to be effective. This is known as empirical antibiotic therapy. In clinical practice, it is not possible to wait for the results from the sputum sample to confirm the bacteria as this can take some time. However, a sputum sample has been sent for microbiology to confirm the causative bacteria. We will consider how microbiologists use the sputum sample to identify the bacteria.

Microbiologists have a range of tests they can use to identify which bacteria are causing the disease. They might prepare a sample of the sputum and look at this under the microscope to identify the bacteria. Many bacteria can, however, appear very similar. Special stains can help with the identification. A common stain which is used is the Gram stain. Bacteria are described as either Gram-positive or Gram-negative. Gram-positive bacteria take up the stain and appear purple under the microscope. Gram-negative organisms are not stained. In general, Gram-positive bacteria have a simpler cell wall structure than Gram-negative bacteria. This may mean that antibiotics are more able to penetrate and destroy Gram-positive bacteria. The shape and colour of the bacteria can also give vital clues. *Staphylococcus aureus*, for example, is round (coccus = round) and golden in colour (aureus = golden). The microbiologist may also identify the conditions the bacteria need in order to grow. Some grow well with no oxygen (called anaerobic bacteria) while others require oxygen (aerobic bacteria). Some are described as facultative anaerobes. This means that they prefer oxygen but can grow without it. Identifying the bacteria which are causing the disease is very important as different bacteria will be sensitive to different antibacterial drugs. Microbiologists can also test this directly by testing whether the bacteria grow in the presence of certain antibiotics.

The bacteria which could be causing George’s community acquired pneumonia are shown in Table 3.6.

In George’s case, the microbiology report identified *Streptococcus pneumoniae* and that the bacterium is sensitive to amoxicillin, confirming that the antibiotic choice was appropriate.
Table 3.6 Properties of bacteria associated with community acquired pneumonia

<table>
<thead>
<tr>
<th>Bacterium</th>
<th>Gram staining</th>
<th>Oxygen needs</th>
<th>Shape</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Streptococcus pneumoniae</em></td>
<td>G +ve</td>
<td>Anaerobic but tolerates oxygen</td>
<td>Cocci (round shape)</td>
</tr>
<tr>
<td><em>Haemophylis influenza</em></td>
<td>G –ve</td>
<td>Facultative anaerobe</td>
<td>Rod shaped</td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td>G +ve</td>
<td>Facultative anaerobe</td>
<td>Cocci (round shape)</td>
</tr>
<tr>
<td><em>Klebsiella pneumoniae</em></td>
<td>G –ve</td>
<td>Facultative anaerobe</td>
<td>Rod shaped</td>
</tr>
</tbody>
</table>

G +ve: Gram-positive; G –ve: Gram-negative.

General signs and symptoms of microbial infection

In the scenario, we can see that George was exhibiting some signs of a bacterial lung infection, for example rapid respiration, cough, yellow sputum and a high temperature (pyrexia).

Many of the signs and symptoms of infection are due to the body’s immune response to the infection. Pyrexia is caused when our temperature control centre, the hypothalamus in the midbrain, is reset to a higher than normal temperature, which in humans is 37°C. This occurs when chemicals called prostaglandins and pro-inflammatory cytokines are released from immune cells as part of the inflammatory response (Chapter 2). Metabolism is increased and tissue oxygen requirements increase resulting in increased respiratory rate. The advantages of pyrexia are unclear as it consumes energy at a time when a person is often eating little. It is thought that higher temperatures might stop some microorganisms multiplying as quickly. Higher temperatures may also stimulate the immune system and tissue repair. In most cases the pyrexia does not need treatment and the temperature will return to normal. Some microorganisms, however, may cause a rise in temperature above 41.5°C. This is considered a medical emergency. The pro-inflammatory cytokines which cause fever, also cause general malaise, weakness and loss of appetite associated with infection.

The course of an illness can be divided into different stages (Table 3.7) which help to explain the pattern of symptoms shown by a patient.

Invading microorganisms can cause tissue injury through a variety of mechanisms. For example, HIV causes the destruction of CD4-positive T lymphocytes during its active replication phase (Chapter 4). CD4-positive lymphocytes are vital for our immune system to work properly. HIV-infection may eventually lead to immunodeficiency.
Chapter 3

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incubation period</td>
<td>Pathogen begins to replicate but symptoms not noticeable.</td>
</tr>
<tr>
<td>Prodromal phase</td>
<td>Initial appearance of symptoms but these may be vague and non-specific, for example malaise, fatigue.</td>
</tr>
<tr>
<td>Acute stage</td>
<td>Maximum impact of infection. Symptoms are obvious and usually specific for sites of infection.</td>
</tr>
<tr>
<td>Convalescent period</td>
<td>Infection is contained and is progressively eliminated.</td>
</tr>
<tr>
<td>Resolution</td>
<td>Pathogen is eliminated from the body. No further symptoms.</td>
</tr>
</tbody>
</table>

Table 3.7 Stages of illness

Some microorganisms damage our cells by producing toxins. An example of this is *Vibrio cholera*, the bacterium which results in cholera. *V. cholera* infects the gut and releases a toxin causing severe watery diarrhoea. The toxin is classified as an exotoxin. This means a toxin that is released from the bacterial cells. By contrast, an endotoxin is part of the cell wall of Gram-negative bacteria. It is only released if the bacterial cell disintegrates. At low levels endotoxins are useful and activate our innate immune system. However, at high levels, the immune response to the endotoxin can lead to sepsis (see below, under ‘sepsis’). Many microorganisms cause damage indirectly by activating the body’s immune system. The immune system responds by killing infected cells. For example, the immune response to hepatitis B virus results in destruction of liver cells through a type IV hypersensitivity response (Chapter 4). The long-term complication of hepatitis B infection can be cirrhosis of the liver or liver cancer.

Pharmacological management of infections

Antimicrobial drugs

An antimicrobial is a substance which kills or inhibits the growth of microorganisms. The British National Formulary (BNF) groups these according to which type of microorganism is killed and lists: antibacterials, antifungals, antivirals and antiprotozoal drugs. In general, antibacterials work by exploiting differences between human cells and those of the microorganism. If the antimicrobial was not selective in this way it would kill human cells as well as killing the microorganism.

Antibacterial drugs

Antibacterial drugs are used to combat infections caused by bacteria. They will not kill viruses. Antibacterial drugs are sometimes referred to as antibiotics. Antibacterial drugs can be classified in different ways. A common way to group them is by chemical structure. Penicillins, for example, all have a similar chemical structure called a beta lactam ring. Groups of antibiotics may share certain features including the way they...
work, side-effects and any contraindications they may have. They may also kill similar bacteria, although this is not always the case. Activity 3.2 will give you an overview of some of the important groups of antibiotics and the names of drugs that belong to each group.

**Activity 3.2 Research**

Using the table format below, list one or two drugs for each group of antibacterial drugs. Use the British National Formulary to help you. You should be able to access paper copies from the ward or library. An electronic version is also available for NHS staff.

<table>
<thead>
<tr>
<th>Antibacterial group</th>
<th>Name of individual antibacterials</th>
</tr>
</thead>
<tbody>
<tr>
<td>Example: Cephalosporin</td>
<td>Example: Cefaclor, Cefalexin</td>
</tr>
<tr>
<td>Penicillins</td>
<td></td>
</tr>
<tr>
<td>Macrolides</td>
<td></td>
</tr>
<tr>
<td>Quinolones</td>
<td></td>
</tr>
<tr>
<td>Tetracyclines</td>
<td></td>
</tr>
</tbody>
</table>

*Suggested answers are found at the end of the chapter.*

Now that you are more familiar with the different antibacterial groups, we will look more closely at how these drugs act to treat infections.

Some antibacterial drugs kill bacteria. These are known as *bactericidal* antibiotics. Examples include penicillins and aminoglycosides. Other antibacterial drugs do not kill the bacteria outright but prevent bacteria replicating. These are known as *bacteriostatic* antibiotics. Examples include tetracyclines. The bacteriostatic action helps the body’s immune system kill the bacteria.

*Narrow spectrum* antibiotic drugs kill only very specific bacteria. By contrast, *broad spectrum* antibiotics kill a wide variety of bacteria. Examples of broad spectrum antibiotics include erythromycin, ciprofloxacin and doxycycline. A broad spectrum antibiotic is useful when the cause of infection is unknown. Unfortunately, they tend also to kill bacteria comprising the gut microbiome. The possible consequences of this are illustrated in the scenario below.

*Clostridium difficile* is a Gram-positive bacterium. It can persist in the environment for a long time as it produces tough spores which are resistant to alcohol based cleaning agents. It is found in the gastrointestinal tract of about 5% of the population where it usually causes minimal harm. However, in Edward’s case in the scenario below, treatment with the broad spectrum antibiotic ciprofloxacin
Scenario: *Clostridium difficile*

Edward is an elderly man currently an inpatient on a medical ward. He has recently been taking a course of ciprofloxacin for acute pyelonephritis (a type of kidney infection; in Edward's case due to *E. coli*).

A week later his infection seems to have improved. However, he is now experiencing profuse watery diarrhoea. A diagnosis of *Clostridium difficile* infection is made. You transfer Edward into a single-bedded side room. He is treated with the antibiotic metronidazole in accordance with guidance. You ensure that strict handwashing procedures are in place on the ward.

disrupted his normal gastrointestinal bacteria (microbiome). As a result, *C. difficile* was able to grow without competition from other bacteria. *C. difficile* can lead to **pseudomembranous colitis** as it produces toxins which inflame the bowel. Although still a problem in hospitals, infection control procedures and more careful use of antibiotics have reduced the incidence of *C. difficile* infections.

**Mechanism of action of antibacterial drugs**

Antibacterial drugs work in different ways and we can divide them up according to their mechanism of action.

1. **Antibacterials that disrupt cell wall synthesis:** Examples of antibacterial drugs that disrupt cell wall synthesis are penicillins, cephalosporins, carbapenems and monobactams. The bacterial cell wall is made of a substance called peptidoglycan which forms a protective mesh. The antibacterial drugs interfere with the synthesis of peptidoglycans and, as a result, the cell wall is weakened. In the absence of a rigid cell wall, water enters the bacterial cell by osmosis, causing it to swell, burst and die.

2. **Antibiotics that affect bacterial protein synthesis:** Antibacterial drugs that affect bacterial protein synthesis are tetracyclines, chloramphenicol, macrolides and aminoglycosides. Bacterial proteins are manufactured within the bacterial cell using cellular ‘machinery’ called ribosomes (Figure 3.1). Antibacterial drugs that affect bacterial protein synthesis bind to the bacterial ribosomes and prevent bacteria from making proteins. This stops the bacteria growing or multiplying.

3. **Antibacterial drugs that inhibit bacterial DNA synthesis:** Antibacterial drugs that inhibit bacterial DNA synthesis include quinolones. DNA synthesis is vital for bacterial cell replication.

4. **Antibacterial drugs that affect folic acid synthesis:** Antibacterial drugs that affect folic acid synthesis include Trimethoprim. Bacteria need to manufacture their own folic acid to survive. Some antibacterial drugs interfere with this folic acid synthesis and kill bacteria.
Side-effects and clinical implications of antibacterial drugs

Many antibacterial drugs cause gastrointestinal upset as a side-effect leading to symptoms of nausea, vomiting and diarrhoea. This is often because the antibacterial has damaged the natural gut bacteria (microbiome). This was seen in the scenario with Edward, above.

Drug allergies

Many people report that they are allergic to antibiotics. It is important to understand the symptoms that lead to this belief. Some people may have experienced feeling sick which would not be a true allergic reaction. Symptoms such as rashes, difficulty breathing, and swelling, are more indicative of allergy. If a person is truly allergic to an antibacterial it may make their infection much more difficult to treat. Penicillins are especially prone to causing allergies. If patients are allergic to one type of penicillin, for example amoxicillin, they are likely to be allergic to all of them, for example flucloxacillin and penicillin V. This is because they all have the same beta lactam ring which causes the allergy. They may also be allergic to cephalosporins but these can be used as alternatives if people are monitored. A patient with a penicillin allergy would have to be given an alternative from a different group. For example, patients allergic to penicillins are often offered the macrolide erythromycin instead.

Activity 3.3 Decision-making

A patient on your ward has a drug chart which states that they are allergic to penicillin. The following antibiotic is written up for the patient: Co-amoxiclav 500 mg three times a day. Would this be safe to give?

An outline answer is available at the end of the chapter.

Some antibacterial drugs are more toxic and cause more side-effects than others. Aminoglycosides, such as gentamicin for example, are toxic to the kidney and ear. The dose and blood levels need to be closely monitored to prevent toxicity. The full range of side-effects caused by antibiotics can be found in the BNF or the summary of product characteristics for each antibiotic.

Choosing antibiotics

Scenario

Josie is a 30-year-old woman who attends your nurse-led clinic appointment. She describes increasing urinary frequency over the last couple of days and a burning
sensation on passing urine. She has also felt tired and a bit flu-like since yesterday. She has no drug allergies and is on no other medication. You carry out a mid-stream urinalysis test. This tests positive for nitrite and leucocyte esterase. You suspect a lower urinary tract infection. Josie is prescribed a three-day supply of Trimethoprim tablets 200 mg twice a day.

In order to treat infection an antibiotic has to be chosen which is likely to kill the infective organism. As we can see from the scenario, a diagnosis has been made according to symptoms and the results of the urinalysis test. As the infection is mild, no attempt has been made to identify the microorganism causing the disease and a best-guess has been made as to what the likely microorganism is. You may remember that the same ‘best-guess’ occurred with Edward. Activity 3.4 should give you an insight into how empirical antibiotic therapy is chosen.

### Activity 3.4 Critical thinking

Do you think that Trimethoprim, at this dose and for this length of time, was an appropriate choice of antibacterial therapy for this urinary tract infection? The BNF contains a summary of antibacterial therapy which you might find useful.

A suggested answer is given at the end of the chapter.

Although the BNF gives a useful quick reference guide it is important to be aware of where this information comes from and to be able to compare it to other sources of information. Public Health England have issued a guideline ‘Management of infection guidance for primary care for consultation and local adaptation’. It can be accessed from Public Health England’s website. A link is provided at the end of the chapter.

You may want to research the evidence for treatment of urinary tract infections and compare the guidance with that in the BNF. NICE also has guidance relating to many different types of infection. Local hospitals often have their own antibacterial policies which take into account local patterns of resistance. We will look at resistance later in the chapter.

**Antifungals**

Fungi are eukaryotic and therefore have more similarity to human cells than bacteria. Ensuring antifungals are selective (which means they kill only fungi and not human
Infection

cells as well) can be a challenge as many of the targets used by antibacterial drugs do not exist in fungi. Penicillins, for example, would not kill fungi as fungi do not contain peptidoglycans in their cell walls. Luckily, although both human cells and fungi have cell membranes they are slightly different. Antifungal agents act by damaging the cell membrane of fungi. The two most commonly used groups of antifungals are ‘Triazole’ antifungals and ‘Imidazole’ antifungals. The name relates to their chemical structure. Some antifungals are available as oral or parenteral formulations but many are also used topically to treat localised infections, for example, clotrimazole cream for vaginal thrush.

Side-effects and implications for practice

Like antibacterial drugs, common side-effects include gastrointestinal disturbance. Many antifungal agents are toxic to the liver and should be used with care or avoided in patients with liver problems.

Antifungals often interact with other drugs. This is because they inhibit specific enzymes in the liver which are involved in the metabolism of other drugs (Chapter 1). So, for example, a patient on simvastatin should not be given ketoconazole at the same time because the levels of simvastatin could rise leading to toxic side-effects.

Antivirals

Viruses can be very difficult to kill as they are such simple structures and offer few targets for killing agents. Immunisation with a vaccine is often our best defence against viruses (Chapter 4). However there are some very useful antiviral agents. Aciclovir was one of the first effective antivirals to be developed. It is effective against herpes simplex, which causes cold sores and genital infections. It is also effective against varicella-zoster, which causes shingles and chickenpox. Aciclovir is actually a pro-drug (Chapter 1) and is activated by an enzyme in the virus. This is what makes the drug selective. The active form of aciclovir prevents DNA replication and inhibits this process for viral DNA more effectively than for human DNA. Viruses are therefore prevented from reproducing. Other useful antiviral agents include those used for the treatment of HIV but these are outside the scope of this book.

Antimicrobial resistance

Resistance is the ability of a microorganism to resist the effects of an antimicrobial drug. It is becoming a serious global health concern. In 2013 the Chief Medical Officer said that ‘Antibiotic resistance poses a catastrophic threat’ and went on to explain that if we do not act now we could be at risk of dying from ordinary infections we now think of as treatable. Some microorganisms are naturally resistant to some antimicrobials; this is known as innate resistance. We cannot kill a virus with an antibacterial agent such as penicillin, for example. Knowing which microorganism is causing the infection helps us choose the right
antimicrobial and should mean this kind of resistance is not generally a problem. In other cases microorganisms evolve and become resistant to antimicrobials that previously treated the infection they caused. It is this second type of resistance which is the most troubling. Although resistance can occur to many antimicrobials, we will concentrate here on resistance to antibacterials, the drugs that treat bacterial infections, as this is causing the greatest concern. The more that antibacterials are used, the more likely resistant organisms are to emerge. The resistant organisms go on to infect other people and may become widespread in the population.

Scenario

You are a nurse working on a surgical ward. Your patient, Nisa, is going for hip replacement surgery. She had a screen for meticillin resistant *Staphylococcus aureus* (MRSA) earlier and was found to be positive for this bacterium. As a result, you have been asked to ensure that she receives mupirocin nasal ointment and chlorhexidine body wash and shampoo. This will help to reduce the amounts of MRSA present and thereby reduce the risk of infection by MRSA during surgery. If Nisa was infected during surgery, the treatment would be much more complicated than for non-resistant strains of *Staphylococcus aureus*. This is because MRSA is resistant to the antibacterial agent flucloxacillin. This antibacterial would therefore not cure the infection. More toxic antibacterials such as vancomycin might be needed instead.

Mechanisms of resistance

Bacteria reproduce by binary fission – that is, dividing into two. This is an example of asexual reproduction which, in biology, means without the mixing of genes. One parent therefore gives rise to two identical daughter cells. However, in order to survive in a world with a changing environment, which may include one with the new challenge of antibacterial therapy, bacteria need to mutate, which means they need to change genetically. Spontaneous mutations may occur in a bacteria’s genetic make-up as it reproduces. *E. coli*, for example, can replicate itself every 20 minutes. This means that in 7 hours one bacterium can generate over two million bacteria. If a mutation means the bacteria is more able to survive the antibacterial than other bacteria this will quickly be passed on. However, in reality, if an appropriate antibacterial is taken properly the body’s immune system will usually mop these resistant bacteria up. The most important way that resistance is passed on is when bacteria pass genes to each other via a process called conjugation (see Figure 3.5). Bacteria have one main chromosome but also contain separate pieces of DNA called plasmids. These plasmids often contain genes for antibacterial resistance. Two bacteria join together using sex pili and pass plasmids from one bacterium to another. The genes that are passed on in the plasmid now enable the bacteria to counteract the antibacterial. Bacterial conjugation enables bacterial antibacterial resistance to spread very rapidly through bacterial populations. The mechanisms bacteria use to counteract antibacterials are outlined below:
• Bacteria may produce enzymes that inactivate the antibacterial.
• Bacteria may actively remove the antibacterial from themselves.
• Bacteria may alter the binding site that the antibacterial usually adheres to so that it no longer 'sticks'.
• Bacteria may begin using different metabolic pathways to those that are being inhibited by the antibacterial.

Antibiotic resistance genes are often carried on circular pieces of DNA called plasmids. A donor bacterial cell joins with a recipient bacterial cell through the pilus. The plasmid is copied and passed on to the recipient cell. Both the donor and recipient cell now have a copy of the plasmid. This process can continue with other bacterial cells and antibiotic resistance genes can be passed on very rapidly through the bacterial population.

Overcoming antibacterial resistance

Resistance is a global issue and needs to be tackled with different approaches. New antimicrobial agents need to be developed which microorganisms are not yet resistant to. However, there has been a decline in the development of new antimicrobials as they are expensive to research and are held in reserve for serious infections which means relatively little money is spent buying them. Countries also need to tighten
control of antibacterial supplies so they are not used for inappropriate infections. Healthcare professionals also have a role to play. The more that antimicrobials are used the faster resistance develops. If infection prevention and control is improved, the need for antimicrobials in the healthcare setting can be reduced. Correct use of antimicrobials is also important. Advice from experts such as microbiologists is important for choosing the correct drug. Narrow spectrum antibacterials are preferable to broad spectrum which was illustrated by the *Clostridium difficile* case study. Patient education is important. Patients should be encouraged not to seek antibacterials for viral infections. It has been shown that people are satisfied to receive reassurance and accurate explanations about their condition compared to antibacterials they do not need (Britten, 1995). The TARGET antibacterials toolkit produced by the Royal College of General Practitioners and others has some useful resources and leaflets for patients. The website can be found at the end of the chapter.

**Activity 3.5 Reflection**

List interventions that you have carried out or observed others carrying out which help to prevent the spread of antimicrobial resistance. For each think about how this helps to combat resistance.

*A suggested answer is given at the end of the chapter.*

Having reviewed the general signs and symptoms of infection and the pharmacological management of infections, we turn to examine sepsis which is one of the most important concerns in healthcare today (NICE, 2016).

**Sepsis**

Sepsis is defined as ‘life-threatening organ dysfunction caused by a dysregulated host response to infection’ (Singer et al., 2016). This rather technical definition tells us that sepsis is a life-threatening response to infection. Sepsis is estimated to affect 31.5 million people worldwide every year and cause an estimated 5.3 million deaths each year.

Our understanding of sepsis has changed over the years, and its definition has been updated a number of times. The current definition is The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3; Singer et al., 2016). As sepsis is such an important concern in healthcare today, it is worth examining this definition. How sepsis is understood in the healthcare setting affects its recognition and prompt treatment. The box below examines the Sepsis-3 definition.
Our understanding of sepsis has evolved over the years. The 1991 and 2001 consensus definitions were based upon the ‘systemic inflammatory response syndrome’ (Balk, 2014). The systemic inflammatory response syndrome (SIRS) is a widespread (systemic) inflammatory response that can follow a diverse group of injuries. The injuries include not only infection, but trauma, burns, and pancreatitis and other injuries. Within the SIRS framework, sepsis was defined as ‘SIRS with documented or suspected infection’. There were two further categories within a sepsis continuum: severe sepsis and septic shock – of increasing severity.

The Sepsis-3 definition was developed to reflect advances in our understanding of the pathogenesis of sepsis (Singer et al., 2016). In particular, the Sepsis-3 definition focuses on the central role of infection in sepsis. Singer et al. (2016) argue that this is an important development of the previous central focus on the systemic inflammatory response, SIRS. The stronger emphasis on infection, it is argued, helps focus treatment on prompt antimicrobial therapy (see below under ‘Treatment of sepsis’). Sepsis is clearly more complex than the original ‘systemic inflammatory response with infection’ suggests. There is often an anti-inflammatory response, especially in the late stages of sepsis that can make patients susceptible to further infection. There are metabolic dysfunctions which affect cellular respiration and cell function. These advances in the understanding of sepsis pathogenesis have important implications for sepsis management and the development of new treatments.

The Sepsis-3 definition has produced different criteria for recognition of sepsis than the older SIRS criteria. There is ongoing debate about the most appropriate criteria for recognition of sepsis in clinical practice. For example, the British Medical Journal Best Practice: Sepsis in Adults (2018) notes that ‘these changes have prompted much debate and the 1991 definitions remain in widespread clinical use while the controversies are resolved’.

Having examined the Sepsis-3 definition of sepsis in the box above, we will now look at the most common causes of sepsis, the risk factors for sepsis and the pathophysiology of sepsis.

The most common causative agents of sepsis are bacteria – either Gram-negative or Gram-positive. Fungal infections can also cause sepsis. However, the causative agent of sepsis is only isolated and identified in around half of all cases. Of those cases in which the agent has been identified, common agents are Gram-positive *Staphylococcus aureus*, Gram-negative *Escherichia coli* and *Pseudomonas* spp. Common fungal agents include *Candida* spp. and *Aspergillus* spp. In the UK, sepsis is the most common direct cause of maternal death. In the 6-week postnatal period, group A *Streptococci* are the most common causative agent. Many causes of sepsis in the community setting are likely to be from the patient’s microbiome and are described as endogenous (or ‘from within’).
NICE (2016d) provides guidelines for early recognition, diagnosis and treatment of sepsis. The guidelines state: ‘Think “could this be sepsis?” if a person presents with signs or symptoms that indicate possible infection’. Assessment includes a person’s risk of infection, and in a face-to-face assessment includes assessing temperature, heart rate, respiratory rate, blood pressure, level of consciousness and oxygen saturation. The details of risk stratification tools can be found within NICE (2016d).

Knowledge of risk factors for sepsis are pivotal in early recognition (NICE, 2016d). Table 3.8 lists some of the main risk factors for sepsis.

<table>
<thead>
<tr>
<th>Risk factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underlying malignancy</td>
</tr>
<tr>
<td>Age &gt; 65 years</td>
</tr>
<tr>
<td>Haemodialysis</td>
</tr>
<tr>
<td>Alcoholism</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Recent surgery or other invasive procedures</td>
</tr>
<tr>
<td>Breached skin integrity</td>
</tr>
<tr>
<td>Indwelling lines or catheters</td>
</tr>
<tr>
<td>Intravenous drug use</td>
</tr>
<tr>
<td>Pregnancy or recent pregnancy</td>
</tr>
</tbody>
</table>

Table 3.8 Key risk factors for sepsis

As can be seen from Table 3.8, many risk factors involve a pre-existing medical condition, such as a malignancy, alcoholism or diabetes. These may be associated with a weakened immune system and a greater susceptibility to infection. Other risk factors include breached skin integrity and indwelling lines or catheters. These again increase the risk of infection.

The common sites of infection in sepsis include the respiratory tract, the bloodstream, the abdomen, the skin and urinary tract. However, infection at any site in the body can potentially lead to sepsis. To help with the recognition and management of sepsis, an understanding of the pathophysiology of sepsis is essential. We turn to describing this below, building from the Sepsis-3 consensus definition.

**Pathophysiology of sepsis**

Sepsis-3 defines sepsis as a dysregulated host response to infection which can lead to life-threatening organ dysfunction. ‘Dysregulation’ refers to impairment of normal regulatory responses. In sepsis, the dysregulated host response to infection has components which will be explained shortly. These responses are often described as the ‘sepsis cascade’ and can occur very rapidly in some patients, in some cases over a period of hours. Multi-organ failure can result and possible death.
Figure 3.6 shows the main components of the ‘dysregulated host response’ to infection that are found in sepsis. Many of these responses have been described in Chapter 2 within the sections on acute inflammation. In an acute inflammatory response, the responses were noted as being localised, well-regulated and offering a protective effect. By following the description below, you will see the contrast between a well-located and well-regulated response and the ‘dysregulated’ response which constitutes part of the host response in sepsis. This, in turn, can cause widespread life-threatening organ dysfunction.

An infection and/or the presence of microbial products from disrupted bacterial cells causes a widespread host response. Part of this host response includes activation of:

- innate immune cells – especially white blood cells called neutrophils and monocytes;
- the complement system (a set of plasma proteins which help fight infection);
- the coagulation system (another set of plasma proteins which lead to blood clotting);
- the endothelial cells (these cells make up the ‘endothelium’ or inner lining of the blood vessels).

These responses result in the widespread release of ‘pro-inflammatory cytokines’ and other mediators of inflammation (Chapter 2). Pro-inflammatory mediators and complement components cause further endothelial activation and further activation of immune cells. The activated immune cells and the endothelium continue to produce mediators of
inflammation. These include IL-6, IL-8, nitric oxide, platelet activating factor, and reactive oxygen species. There is a counter-balancing production of anti-inflammatory mediators, which paradoxically, may lead to immunosuppression.

The activation of the endothelium is key to understanding the progression to low blood pressure (hypotension) and organ failure in sepsis. Activation of the endothelium results in widespread vasodilation, and an increased permeability of blood vessel walls. Increased vascular permeability causes excessive fluid loss from the vascular compartment (from the circulation) and a reduced blood volume. Vasodilation and reduced blood volume together reduce blood pressure (hypotension) and may lead to reduced tissue perfusion, potentially leading to organ failure.

Activation of the endothelium also activates the coagulation system in the blood. A ‘procoagulant state’ may be reached in which microthrombi (small ‘blood clots’) form in small blood vessels. This is known as disseminated intravascular coagulation (DIC) and can lead to tissue ischaemia.

The inflammatory mediators also cause systemic effects including fever. There is evidence that mediators and reactive oxygen species directly affect cell metabolism. This includes a reduction in contractility of the myocardial cells of the heart. Sepsis is also characterised by insulin resistance and hyperglycaemia. This has been attributed to the production of stress hormones glucagon, adrenaline and cortisol.

Multi-organ failure may result. This is due to the multiple impacts of hypotension, oedema, and disseminated intravascular coagulation. There are direct metabolic effects from inflammatory mediators and cytokines, leading to anaerobic metabolism and cell dysfunction. In many cases the lungs are the first organs affected. This manifests as acute respiratory distress syndrome and acute lung injury. There is often cardiovascular instability and deteriorating renal function. Acute kidney injury can result (Chapter 14).

The magnitude of a patient’s response and hence the outcome and severity of sepsis depends upon a range of factors: the virulence of the infecting microorganism, the host’s immune status and other co-morbidities (Mitchell et al., 2016). The nature and level of mediators and cytokines released has a significant effect.

Treatment of sepsis

As we have seen sepsis is a complicated condition. Early recognition and treatment is vital for the patient. Treatment guidelines suggest that a range of interventions are needed to stop a patient deteriorating. One bundle of therapies is known as the ‘sepsis six bundle’.

These interventions are:
1. **Administer oxygen:** Oxygen should be given to all patients with sepsis. In sepsis oxygen saturation of the tissues falls. This is because blood pressure is reduced leading to hypoperfusion of tissues. In addition, leaky capillaries lead to oedema which means oxygen must diffuse further to reach tissues. Small blood clots can occur in the capillaries which reduces oxygen delivery.

2. **Take blood cultures:** This should be done before antibiotic therapy is started where possible. Blood cultures help to identify the causative pathogen and antibiotics can be adjusted accordingly.

3. **Give IV antibiotics:** Sepsis is usually triggered by a bacterial infection and it is important that the infection is treated as soon as possible. Intravenous antibacterial drugs should be given within one hour of a sepsis diagnosis (NICE, 2016d). At the beginning it may not be clear which bacteria is causing the infection. Broad-spectrum antibacterial drugs are therefore often prescribed as they kill a wide variety of bacteria. The exact antibacterial prescribed will vary depending on local resistance patterns and the likely causative pathogen. It is also important that any potential sources of infection, such as urinary catheters, are removed.

4. **Give intravenous (IV) fluids:** During sepsis a patient’s blood pressure may fall leading to reduced tissue perfusion. Intravenous fluid replacement such as Hartmann’s solution would be given. Fluid replacement increases the circulating volume and restores blood pressure.

5. **Check serial lactate levels:** High lactate levels indicate anaerobic metabolism due to reduced tissue perfusion; this means the patient is in shock. Monitoring lactate helps to show how effective oxygen therapy and IV fluids are.

6. **Monitor hourly urine output:** Urine output falls when renal perfusion is reduced. This is a useful way of monitoring cardiac output which again helps to monitor response to treatment.

It is now time to review what you have learned within this chapter by undertaking some multiple choice questions.

### Activity 3.6 Multiple choice questions

1. What is the definition of a commensal microorganism?
   a) A microorganism that causes disease
   b) A microorganism that is transmitted from animal to human
   c) A microorganism that usually lives harmlessly on our bodies
   d) A microorganism that needs other microorganisms to survive

(Continued)
2. For each of the following state whether the answer is TRUE or FALSE.
   a) *Candida albicans* is a bacterium
   b) Hepatitis B is a virus
   c) *Toxoplasma gondii* is a protozoan
   d) *Escherichia coli* is a fungus

3. Bacteria are prokaryotic cells and differ from human, eukaryotic cells because:
   a) They have no nucleus
   b) They have no DNA
   c) They have no cell wall
   d) They have no ribosomes

4. Symptoms of infection can be caused by
   a) Our immune system
   b) Toxins produced by microorganisms
   c) The death of invaded human cells
   d) All of the above

5. Which of the following statements about *Clostridium difficile* is TRUE?
   a) It is a type of virus which infects the gastrointestinal tract
   b) It is one of the leading causes of pneumonia
   c) It can occur after the use of broad spectrum antibiotics
   d) It is spread by droplets which are inhaled by patients

6. If a patient is allergic to penicillin which of the following antimicrobial drugs can they take? Answer TRUE or FALSE for each drug.
   a) Flucloxacillin
   b) Trimethoprim
   c) Co-amoxiclav
   d) Fluconazole

7. Which of the following is NOT an example of how an antibacterial agent works?
   a) Inhibition of cell wall synthesis
   b) Inhibition of mitochondrial respiration
   c) Disruption of protein synthesis
   d) Inhibition of DNA synthesis

8. For each of the following statements about antimicrobials state whether the answer is TRUE or FALSE.
   a) Aciclovir is an antiviral agent
   b) Clotrimazole is an antibacterial agent
c) Oxytetracycline is an antifungal agent
d) Metronidazole is an antibacterial agent

9. As part of a strategy to combat antibiotic resistance, patients may be given a leaflet explaining why they are not being given an antibiotic and explaining how long symptoms of their illness might last. A cough usually lasts:
   a) 4 days
   b) 7 days
   c) 14 days
   d) 21 days

10. Which of the following are useful strategies for preventing antibiotic resistance? Mark each answer as TRUE or FALSE.
   a) Using broad spectrum antibiotics where possible
   b) Preventing infections by good hygiene
   c) Ensuring doses of antibiotics are not missed
   d) Ensuring courses of antibiotics continue for at least two weeks

Chapter summary

There are many different types of microorganisms but many live harmlessly in the environment and on our body. Microorganisms include bacteria, viruses, protozoa and fungi. However sometimes microorganisms are pathogenic and cause diseases. The body has many defences against disease, but infectious diseases can still be lethal. Different medicines have been developed to fight infections. These usually work by exploiting differences between human cells and the cells of the invading microorganism. Resistance to antimicrobial agents is an increasing problem. Microorganisms are constantly evolving different ways to survive the antimicrobials that we design to kill them. If we do not use the antibacterials we have very carefully and invent new ones, then we could one day be faced with dying from simple infections. Understanding the importance of correct antibacterial choice and administration can help nurses prevent resistance spreading. The cycle of infection can be used to identify infection control measures nurses can undertake to prevent infections in the first place.

Sepsis is a life-threatening response to infection and is an important concern in healthcare today. Early recognition and management of sepsis are essential. Sepsis results from complex poorly regulated host responses to infections. It can lead to septic shock, multi-organ failure and death.
Activities: Brief outline answers

Activity 3.1 Reflection (p60)

List five infectious diseases. For each disease write down whether it is caused by a virus, bacteria, fungi or protozoa. If you can, name the microorganism that causes the disease.

There are many examples you could have used – here are a few:

The common cold is caused by a virus. The name of the virus is *rhinovirus*.

Meningitis can be caused by bacteria, viruses or fungi. Bacteria causing meningitis include *Neisseria meningitidis* and *Streptococcus pneumoniae*. Viruses causing meningitis include enteroviruses and mumps virus. *Cryptococcus neoformans* is a fungus that can cause meningitis.

Activity 3.2 Research (p69)

For each of the groups of antibacterials list one or two medicines that belong to this group. Use the BNF to help you.

<table>
<thead>
<tr>
<th>Antibacterial group</th>
<th>Name of individual antibacterials</th>
</tr>
</thead>
<tbody>
<tr>
<td>Example: Cephalosporin</td>
<td>Cefaclor, cefalexin</td>
</tr>
<tr>
<td>Penicillins</td>
<td>Amoxicillin, flucloxacillin</td>
</tr>
<tr>
<td>Macrolides</td>
<td>Erythromycin, clarithromycin</td>
</tr>
<tr>
<td>Quinolones</td>
<td>Ciprofloxacin, moxifloxacin</td>
</tr>
<tr>
<td>Tetracyclines</td>
<td>Doxycycline, oxytetracycline</td>
</tr>
</tbody>
</table>

Activity 3.3 Decision-making (p71)

A patient on the ward has a drug chart which states they are allergic to penicillin. The following antibiotic is written up for the patient. Co-amoxiclav 500 mg three times a day. Would this be safe to give?

No. Co-amoxiclav consists of clavulanic acid and amoxicillin. Amoxicillin is a penicillin and could cause an allergic reaction in this patient.

Activity 3.4 Critical thinking (p72)

Trimethoprim is an appropriate choice. A three-day course may be enough for women with uncomplicated infections.

Activity 3.5 Reflection (p76)

List interventions that you have carried out or observed others carrying out which help to prevent the spread of antimicrobial resistance.

There are many examples you could give. These might include ensuring correct dose, route frequency, not missing doses, keeping to administration times for antibiotics on drug charts. These all help to kill microorganisms most effectively and prevent resistant organisms occurring. You may have seen pharmacists checking charts and querying doses, times, frequencies. You may have seen prescribers referring to guidelines, local policies or microbiologists. You may have had to ensure samples were sent to labs quickly to allow early identification of microorganisms. Washing your hands after seeing each patient prevents the spread of infections and hence the need for antimicrobials. Explaining to patients how to take their antimicrobials or educating
them about what antibiotics can and cannot treat is also useful. Involvement in antimicrobial audits is another example.

**Activity 3.6 Multiple choice questions (pp81–3)**

1. What is the definition of a commensal microorganism?
   - c) A microorganism that usually lives harmlessly on our bodies

2. For each of the following state whether the answer is TRUE or FALSE.
   a) *Candida albicans* is a bacterium
   b) Hepatitis B is a virus
   c) *Toxoplasma gondii* is a protozoan
   d) *Escherichia coli* is a fungus

3. Bacteria are prokaryotic cells and differ from human, eukaryotic cells because:
   - a) They have no nucleus

4. Symptoms of infection can be caused by:
   - d) All of the above

5. Which of the following statements about *Clostridium difficile* is TRUE?
   - c) It can occur after the use of broad spectrum antibiotics

6. If a patient is allergic to penicillin which of the following antimicrobial drugs can they take? Answer TRUE or FALSE for each drug.
   a) Flucloxacillin
   b) Trimethoprim
   c) Co-amoxiclav
   d) Fluconazole

7. Which of the following is NOT an example of how an antibacterial agent works:
   - b) Inhibition of mitochondrial respiration

8. For each of the following statements about antimicrobials state whether the answer is TRUE or FALSE.
   a) Aciclovir is an antiviral agent
   b) Clotrimazole is an antibacterial agent
   c) Oxytetracycline is an antifungal agent
   d) Metronidazole is an antibacterial agent

9. As part of a strategy to combat antibiotic resistance, patients may be given a leaflet explaining why they are not being given an antibiotic and explaining how long symptoms of their illness might last. A cough usually lasts:
   - d) 21 days

10. Which of the following are useful strategies for preventing antibiotic resistance? Mark each answer as TRUE or FALSE.
    a) Using broad spectrum antibiotics where possible
    b) Preventing infections by good hygiene
    c) Ensuring doses of antibiotics are not missed
    d) Ensuring courses of antibiotics continue for at least two weeks
Further reading


A very accessible textbook on medical microbiology.


This is a comprehensive review of sepsis.


Royal College of General Practitioners, Public Health England and The Antimicrobial Stewardship in Primary Care. TARGET antibiotic toolkit. Available at: www.rcgp.org.uk/TARGETantibiotics/

Useful websites

For further information on drugs, their uses, side-effects and patient information leaflets:

www.medicines.org.uk/

Summary of product characteristics.

www.earthlife.net/prokaryotes/welcome.html

Website for more information on microorganisms.

https://sepsistrust.org/professional-resources/education-resources/

Educational resources from the UK Sepsis Trust.
Disorders of Oxygenation and Carbon Dioxide Elimination

Watch the following videos to ease you into this chapter. If you are using the eBook just click on the play buttons. Alternatively go to https://study.sagepub.com/essentialpatho/videos

- Asthma (10:29)
- Chronic Obstructive Pulmonary Disease (5:02)
- Pneumothorax (10:52)
- Cystic Fibrosis (2:49)
- Cystic Fibrosis A-Z (6:36)

Learning Outcomes

When you have finished studying this chapter you will be able to:

1. Identify terms associated with disorders of the respiratory system.
2. Describe the pathophysiology of upper and lower respiratory tract infections.
3. Explain what happens when there is damage to the pleura.
4. Explain the pathophysiological changes that occur in pulmonary disorders.
5. Explain the pathophysiology of asthma and chronic obstructive pulmonary disease.
6. Describe the changes that occur with cystic fibrosis.
7. Identify the changes that occur when there are interruptions to the pulmonary circulation.
8. Explain the changes that occur in the respiratory system during acute lung disorders.
INTRODUCTION

In this chapter, you will consider disorders of pulmonary function; various types of disease can disrupt the normal processes associated with gaseous exchange. Pulmonary disease may be classified as infectious or non-infectious, acute or chronic, obstructive or restrictive. We look at these various diseases and the impact that they have on the person. Additionally, you will also consider the emotional/psychological and social implications of such pathophysiological changes, a central element of person-centred nursing.

Every cell in the body requires a constant supply of oxygen to undertake their metabolic function. In doing so, they produce carbon dioxide (a waste product) that must be removed. The respiratory system is therefore vital to how we function physically and is essential in maintaining homeostasis.

PERSON-CENTRED CONTEXT: THE BODIE FAMILY

As a person-centred nurse, it is imperative that you understand that disorders of the respiratory system can have a significant impact on health-related quality of life; many conditions are long-term, progressive and can be life-limiting. The mature members of the family may start to notice changes in their respiratory systems. For example, Maud (age 77) and George (age 84) will have structural changes and may have reduced lung capacity that could impact on their activity levels as they may become short of breath more readily. They may also be more susceptible to respiratory tract infections such as pneumonia or bronchitis due to the decrease in the number of alveolar macrophages; thus they receive the flu vaccine each year.

Derek Jones has asthma and needs to monitor his condition closely as exacerbations of his condition may occur; also due to his condition he needs to be careful that he does not develop chest infections. Danielle, as the youngest member of the family, may be susceptible to many respiratory tract infections as she does not yet have a fully developed immune system.

REVISE: A&P RECAP

Before reading this chapter, you may want to revise Chapter 10 in Essentials of Anatomy and Physiology for Nursing Practice (Boore et al., 2016). These videos may also help with revision. If you are using the eBook just click on the play button. Alternatively go to https://study.sagepub.com/essentialpatho/videos

The principal function of the respiratory system is to ensure that the body extracts enough oxygen ($O_2$) from the atmosphere and that excess carbon dioxide ($CO_2$) is expelled (Boore et al., 2016). The process by which this happens consists of three distinct phases: pulmonary ventilation, external respiration and internal respiration (Boore et al., 2016). If for whatever reason these processes are interrupted, pulmonary function may become dysfunctional. There are a number of terms associated with pulmonary dysfunction presented in Table 14.1; it is essential that you understand these terms as they will be referred to frequently as we move through the rest of the chapter.
Table 14.1  Terms associated with pulmonary dysfunction

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnoea</td>
<td>Difficulty in breathing</td>
</tr>
<tr>
<td>Hyperventilation</td>
<td>Ventilation in excess of what is needed for normal elimination of CO₂</td>
</tr>
<tr>
<td>Hypoventilation</td>
<td>Decreased ventilation, unable to eliminate adequate amounts of CO₂</td>
</tr>
<tr>
<td>Hyperpnoea</td>
<td>Increase in rate and depth of breathing (normal during exercise)</td>
</tr>
<tr>
<td>Tachypnoea</td>
<td>Increased respiratory rate</td>
</tr>
<tr>
<td>Bradypnoea</td>
<td>Decreased respiratory rate</td>
</tr>
<tr>
<td>Orthopnoea</td>
<td>Difficulty in breathing when lying flat</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>Reduction in tissue oxygenation</td>
</tr>
<tr>
<td>Hypoxaemia</td>
<td>Decreased levels of oxygen in the blood</td>
</tr>
<tr>
<td>Hypercapnia</td>
<td>Increased levels of carbon dioxide content of the blood</td>
</tr>
<tr>
<td>Acidosis (can be either respiratory or metabolic)</td>
<td>Clinical condition as a result of a low blood pH (&lt;7.35)</td>
</tr>
<tr>
<td>Alkalosis (can be either respiratory or metabolic)</td>
<td>Clinical condition as a result of a high blood pH (&gt;7.45)</td>
</tr>
<tr>
<td>Atelectasis</td>
<td>Lack of gas exchange within alveoli, due to alveolar collapse or fluid consolidation</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>Bluish discolouration of skin and/or mucous membrane due to increased levels of deoxygenated blood in the small vessels</td>
</tr>
<tr>
<td>Ventilation</td>
<td>The amount of air that enters the alveoli</td>
</tr>
<tr>
<td>Perfusion</td>
<td>The amount of blood perfusing the capillaries around the alveoli. Also refers to the delivery of blood to capillary bed in systemic circulation</td>
</tr>
<tr>
<td>Ventilation/Perfusion (V/Q) mismatch</td>
<td>Abnormal ventilation/perfusion ratio, e.g. when areas of the lungs are better perfused by blood than they are ventilated (e.g. lack of alveoli), or better ventilated than perfused with blood (e.g. lack of blood supply to a well-ventilated lung with sufficient alveoli)</td>
</tr>
<tr>
<td>Cough</td>
<td>Protective reflex that helps clear the airways</td>
</tr>
<tr>
<td>Acute cough</td>
<td>Resolves within 2-3 weeks of onset of illness or with treatment</td>
</tr>
<tr>
<td>Chronic cough</td>
<td>Persistent, does not resolve with treatment</td>
</tr>
<tr>
<td>Haemoptysis</td>
<td>Coughing up of blood or bloody secretions</td>
</tr>
<tr>
<td>Minute volume</td>
<td>Volume of air/gas inhaled (inhaled minute volume) or exhaled (exhaled minute volume) from a person’s lungs in one minute.</td>
</tr>
</tbody>
</table>

Let’s have a look at a few of these terms in more detail and how/why they occur.

**HYPOVENTILATION AND HYPERVENTILATION**

Hypoventilation refers to inadequate alveolar ventilation compared to the metabolic demands of the body. It occurs when the minute volume is reduced and is caused by changes in the mechanics of breathing or due to changes in the neurological control of breathing. Hypoventilation results in the accumulation of CO₂ (rate of production exceeds rate of excretion), leading to hypercapnia that may result in respiratory acidosis and affect the normal functioning of many tissues throughout the body.

Hyperventilation refers to increased alveolar ventilation that exceeds the metabolic demands of the body. It occurs when the excretion rate of CO₂ exceeds that which is produced by cellular metabolism, leading to hypocapnia that may result in respiratory alkalosis that can also affect the normal functioning of body tissues.
HYPOXIA AND HYPOXAEemia

Hypoxia refers to a reduction in tissue oxygenation and can result from pulmonary alterations, or other abnormalities that are not related to pulmonary function, e.g. decreased cardiac output. Hypoxaemia refers to a reduction of oxygen in arterial blood and is caused by pulmonary dysfunction and may lead to hypoxia. There are four types of hypoxia:

1. Ischaemic hypoxia – when blood supply to the tissue is inadequate
2. Anaemic hypoxia – when blood is unable to carry enough oxygen to the tissues
3. Hypoxic hypoxia – when inadequate amounts of oxygen enter the lungs
4. Histotoxic hypoxia – when cells are unable to effectively use the oxygen reaching them.

Hypoxaemia may result from a number of factors. Hypoventilation, caused by neurological or muscular disorders restricting chest expansion, can lead to retention of CO₂ and a decrease in O₂. Ventilation/perfusion mismatch; either low V/Q, i.e. inadequate ventilation of well perfused areas of the lungs causing shunting (e.g. atelectasis, asthma, pulmonary oedema or pneumonia) or high V/Q, i.e. poor perfusion of lung areas that are well ventilated (e.g. pulmonary embolus). Signs and symptoms of hypoxaemia include cyanosis, altered mental state and confusion, tachycardia and decreased urinary output (Table 14.2).

HYPERCAPNIA

Hypercapnia refers to an increase in the CO₂ content of arterial blood and is usually caused by disorders that lead to hypoventilation or V/Q mismatching. Increased CO₂ production may result from increased metabolic activity, e.g. exercise, a rise in body temperature, disorders of respiratory muscle function or disorders of neural control of respiration. Hypercapnia can have profound effects on the body and its ability to function normally (Table 14.2).

### Table 14.2  Effects of hypoxia and hypercapnia on the body

<table>
<thead>
<tr>
<th>Hypoxia</th>
<th>Hypercapnia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy production reduced – anaerobic metabolism produces less ATP</td>
<td>Respiratory system – chemoreceptors stimulated – increased respiratory rate</td>
</tr>
<tr>
<td>pH – Increased lactic acid production (↓ pH)</td>
<td>Cardiovascular system – vasodilation of blood vessels, tachycardia, diaphoresis</td>
</tr>
<tr>
<td>Cell – oedema due to ↑ Na⁺ and ↓ H₂O inside cell, ↑ membrane permeability, ↓ mitochondrial activity</td>
<td>Nervous system – vasodilation of cerebral vessels – headache, disorientation, coma</td>
</tr>
<tr>
<td>Central nervous system – restlessness, agitation, uncoordinated movements, impaired judgement, delirium, coma</td>
<td></td>
</tr>
<tr>
<td>Cardiovascular system – tachycardia, peripheral vasconstriction (cool moist skin), increased BP. Later leads to bradycardia and hypotension</td>
<td></td>
</tr>
<tr>
<td>GI system – reduced gut function – constipation, anorexia. Reduced liver function – reduced plasma protein production</td>
<td></td>
</tr>
<tr>
<td>Muscles – reduced function – fatigue</td>
<td></td>
</tr>
<tr>
<td>Pulmonary circulation – pulmonary hypertension (vessels constrict in response to hypoxia), pulmonary oedema</td>
<td></td>
</tr>
<tr>
<td>Cyanosis – excessive concentration of deoxygenated haemoglobin (5g/100 ml blood)</td>
<td></td>
</tr>
</tbody>
</table>
PHASES IN RESPIRATORY DISEASE

Respiratory disease can be categorised into three phases: respiratory impairment, respiratory insufficiency and respiratory failure.

• Phase I: Respiratory impairment
  o Normal healthy adult has a very large functional reserve
  o Although having respiratory impairment, they will have no symptoms
  o Reduced respiratory function only identified through respiratory function tests

• Phase II: Respiratory insufficiency
  o Person becomes aware of respiratory discomfort during exertion
  o Exercise tolerance becomes progressively impaired
  o Blood gases remain within normal limits

• Phase III: Respiratory failure
  o Person loses the ability to maintain normal arterial blood gases at rest
  o $\text{PO}_2$ is low at rest
  o $\text{PCO}_2$ is raised at rest

In your practice as a nurse, you will be caring for people across this range of phases and so it is important to consider the physiological impact and what phase of respiratory disease this person is in. This will influence how much care they may require as a result of the impact on their independence and it may also require you to consider if they and their family need end of life care.

INFECTIONS OF THE RESPIRATORY TRACT

Respiratory tract infections (RTIs) refer to an infectious disease that can affect any part of the respiratory tract; they tend to be discussed in terms of upper respiratory tract infections (URTIs), i.e. infection of nose, oropharynx or larynx, or lower respiratory tract infections (LRTIs), i.e. lower airways and lungs. Diao et al. (2018) state that RTIs comprise as many as 34 kinds of infections. Whilst any pathogen can cause infection of the respiratory tract, viruses are the most frequent cause. RTIs are the most common diseases in humans; Hull et al. (2013) found that adults usually experience one to three episodes of URTIs per year. Signs and symptoms of RTIs depend on the structure infected, the severity of the infection and the person’s age and health status. They are usually relatively limited, consisting mainly of sore throat, fever, cough, productive cough, rhinorrhea with or without pus, shortness of breath, headache and/or general discomfort, earache and/or tinnitus (Diao et al., 2018).

Upper respiratory tract infections

The upper respiratory tract consists of the nasal cavity, sinuses, pharynx and larynx. The upper respiratory tract is continuously exposed to potential pathogens which are usually dealt with by the mucociliary escalator and coughing. Despite these defence mechanisms, infections are fairly common and include the common cold, influenza, rhinitis, sinusitis and otitis media.
Common cold

The common cold is the most frequent cause of upper respiratory tract infection and is usually caused by one of a number of viruses, including rhinovirus, coronavirus, adenovirus, parainfluenza, influenza and respiratory syncytial types, which together have around 200 serotypes (Hemilä and Chalker, 2017). Due to the large number of causative agents, it would be impossible for a person to develop immunity to all, hence they can occur frequently throughout the year. The common cold is spread by respiratory droplets that can be directly inhaled or acquired through contamination of objects by infected secretions. It is highly contagious, especially during the first three days following onset of signs and symptoms; incubation may last up to five days. Signs and symptoms include nasal congestion, rhinorrhea, sneezing, increased secretions and watery eyes. As the nose is congested, mouth breathing may be common and there may be a change in the person’s voice. The person may also complain of a sore throat, headache, mild fever and general malaise.

Influenza

Influenza is a viral infection that can affect both the upper and lower respiratory tract. Influenza is caused by viruses that belong to the Orthomyxoviridae family; there are three different groups of influenza virus that cause disease in humans: type A (most prevalent), type B and type C. Regardless of the type, they mutate constantly, meaning that initiating an effective immune response for a long time is prevented. Influenza type A and type B can cause seasonal epidemics (World Health Organisation [WHO], 2018a) (see Go Deeper box). Influenza C is responsible for causing mild URTIs in adults and children. Influenza may cause three types of infection: uncomplicated URTI, viral pneumonia (discussed later) and respiratory viral infection that is then followed by a bacterial infection. Once a virus establishes an URTI, it then targets and destroys mucus secreting cells, ciliated epithelium and other epithelial cells, thereby leaving large spaces between the underlying basal cells, allowing extracellular fluid to escape, i.e. rhinorrhea. If the infection spreads to the lower respiratory tract, it can cause diffuse shedding of bronchial and alveolar cells.

Signs and symptoms of flu are usually similar to those of other viral infections. Onset is abrupt with fever, chills, malaise, myalgia, headache, rhinorrhea, non-productive cough and a sore throat.

**GO DEEPER**

**Influenza subtypes/lineages**

- *Influenza A* viruses can be categorised into subtypes based on two glycoproteins: haemagglutinin (H) that allows the virus to anchor to the epithelium of the respiratory tract and neuraminidase (N) that facilitates digestion of host secretions and release of viral particles from host cells (Labella and Merel, 2013). There are 16 variants of haemagglutinin (H1–H16) and nine variants of neuraminidase (N1–N9). For example, in the winter of 2017/2018 the main strains circulating were Influenza A(H3N2) and A(H1N1).
- *Influenza B* viruses are not classified into subtypes but can be broken down into lineages, e.g. B-Victoria lineage, B-Yamagata lineage.
- *Influenza C* virus does not present public health importance as it is detected less frequently and usually only causes mild infections (WHO, 2018a).

Epidemics (type A and type B) and pandemics (only caused by type A) result from the ability of viruses to mutate and develop new subtypes against which the population has no protection. Worldwide, these annual
epidemics are estimated to result in about 3–5 million cases of severe illness, and about 290,000 to 650,000 deaths (WHO, 2018a).

In winter 2017/2018, influenza A H3N2 caused widespread disease as it is a particularly virulent strain of influenza type A, mutating at a faster rate and being more dominant. This particular strain spreads with much more severity, is harder to vaccinate against and increases health complications.

Rhinitis

Rhinitis refers to inflammation of the nasal passages. B cells produce immunoglobulin (Ig) IgE against allergens. IgE binds to mast cells and causes degranulation with the release of histamine, proteases, prostaglandins, cysteinyl leukotrienes and cytokines. These inflammatory mediators cause the acute symptoms including sneezing, itch, rhinorrhea and nasal congestion. Allergens presented to T cells cause the release of interleukins (IL), IL-4 and IL-13, that further stimulate B cells to release IL-5, IL-9 and granulocyte macrophage colony-stimulating factor. This results in a switch from a T-helper cell 1 (T\text{h}1) response to a T\text{h}2 response to activate eosinophils, basophils, neutrophils and T lymphocytes, leading to nasal obstruction, hyper-reactivity and anosmia.

Sinusitis

Sinusitis refers to inflammation/infection of the paranasal sinuses, caused mainly by Streptococcus pneumoniae and Haemophilus influenzae; occasionally it may be caused by a fungal infection. The infection leads to obstruction and prevents drainage of the paranasal sinuses into the nasal cavity. Pressure builds up inside the sinus cavity due to the accumulation of exudate, causing severe facial pain that can be confused with toothache (due to blockage of the maxillary sinus) or headache (blockage of the ethmoid sinus).

Otitis media

Otitis media, whilst technically not an infection of the respiratory tract, refers to infection of the middle ear seeded from an upper respiratory tract infection through the eustachian tube. Streptococcus pneumoniae and Haemophilus influenzae are the most common bacteria that cause otitis media. Infection causes inflammation of the middle ear mucosa and inflammatory exudate in the middle ear space. It usually presents with otalgia (ear pain) and hearing disturbances. If it does not resolve it can lead to tympanic membrane perforation and discharge.

Croup

Croup is a condition that affects the airways of babies and young children aged between 3 months and 3 years (NICE, 2017c). It is usually caused by viruses, and the parainfluenza virus accounts for 75% of cases (Johnson, 2014) with the remaining 25% caused by adenoviruses, respiratory syntical virus and influenza types A and B (Zoorob et al., 2011). Whilst the disease is well defined in children, it remains an uncommon cause of respiratory distress in adults (Patel et al., 2018). However, it is believed that adult croup syndrome takes a more severe course than in children and may require definitive airway management and intensive care monitoring (Patel et al., 2018). Croup is characterised by abrupt onset
although it is usually preceded by upper respiratory tract infections. Symptoms are most often worse at night and can fluctuate rapidly depending on whether the child is calm or agitated (Bjornson and Johnson, 2013). As the larynx and subglottic area become inflamed, oedema and exudate can cause obstruction, leading to the characteristic barking cough, hoarse voice, inspiratory stridor and respiratory distress. Symptoms are usually short-lived, with about 60% of children having resolution of the barking cough within 48 hours; less than 2% of children have symptoms persisting for more than 5 nights (Bjornson and Johnson, 2013).

LOWER RESPIRATORY TRACT INFECTIONS

Pneumonia

Pneumonia refers to infection of the pulmonary parenchyma, i.e. bronchioles and alveoli. It may develop as an acute primary infection or may occur as a secondary infection due to another respiratory or systemic condition. In the majority of cases, organisms enter the lungs by inhalation, aspiration or translocation of resident bacteria that spread along the mucosa. Pneumonia can be classified in a number of ways based on the pathogen (typical or atypical) or the area of infection (lobar pneumonia or bronchopneumonia). Pneumonia may also be classified according to the setting in which they occur and referred to as community-acquired pneumonia (CAP), hospital-acquired pneumonia (HAP), health care-associated pneumonia (HCAP) or ventilator-associated pneumonia (VAP) (Table 14.3).

<table>
<thead>
<tr>
<th>Table 14.3</th>
<th>Classification of pneumonia</th>
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</table>
| **Pathogen** | Typical – bacterial  
Atypical – variety of organisms including: *Mycoplasma pneumoniae*, viruses and **fungi** |
| **Area of infection** | Lobar pneumonia – confluent consolidation involving one or more lung lobes. Most often due to *Streptococcus pneumoniae* (the pneumococcus)  
Bronchopneumonia – widespread small patches usually affecting both lungs, especially lower lobes. Causative organisms more varied including: *Streptococcus pneumoniae*, *Haemophilus influenza*, *Staphylococcus*, anaerobes, *coliforms* |
| **Setting** | Community-acquired – describes infection caused by organisms (bacterial or viral) found in the community, begins outside the hospital or develops within 48 hours after admission to hospital  
Health care-associated – occurs in someone who has had a recent hospitalisation or resides in a care home, receiving chronic dialysis or home infusion therapy  
Hospital-acquired – nosocomial infection, LRTI not present on admission or develops 48 hours or more following admission  
Ventilator-associated – nosocomial infection that occurs in a person requiring mechanical ventilation 48 hours or more following intubation. May occur in 9–27% of ventilated patients (Kalanuria et al., 2014) |

The areas of lung below the bronchi are usually sterile despite the entry of pathogens into the airways by inhalation or aspiration of nasopharyngeal secretions. A number of defence mechanisms exist to protect the lower respiratory tract and prevent infections; these include: cough reflex, mucociliary escalator, alveolar macrophages, immunoglobulins (Ig) (IgA and IgG) and cell-mediated and humoral immunity. Loss of one or more of these defence mechanisms predisposes the lower respiratory tract to colonisation and subsequent infection. Those that are critically ill or have a long-term condition are more susceptible to pneumonia as their epithelial cells are much more receptive to binding...
pathogens that cause pneumonia. Colonisation of the tracheobronchial tree is also enhanced in those that smoke, have diabetes or chronic bronchitis.

If a pathogen gains entry to the lower respiratory tract, especially the alveolar region, local host defences (antibodies, complement and cytokines) prepare the bacteria for ingestion by alveolar macrophages. Macrophages present the antigens to the adaptive immune system, thereby activating both cell-mediated and humoral immunity with the release of T and B cells. Macrophages also release inflammatory cytokines, e.g. tumour necrosis factor-alpha, interleukin (IL)-1, while mast cells and fibroblasts release chemokines and chemotactic signals that result in neutrophil recruitment from the lungs into the alveoli. Intense cytokine-mediated inflammation ensues, resulting in the destruction of bronchial mucous membranes and alveolocapillary membranes which leads to vascular engorgement, oedema and the production of fibrinopurulent exudate which infiltrates the acini and terminal bronchioles. Dyspnoea, V/Q mismatching and hypoxaemia result. Consolidation of lung tissue may occur if certain bacteria release toxins that further damage the lung tissue.

**Typical pneumonia (acute bacterial)**

Typical pneumonia is caused by bacteria, with the most common causative agent being *Streptococcus pneumoniae* (the pneumococcus). This type of pneumonia is often referred to as pneumococcal pneumonia and is a frequent cause of lobar pneumonia. Bronchopneumonia may be caused by one or more species of microorganism; infection usually begins in the bronchial mucosa and spreads to adjacent alveoli. Legionnaires’ disease is a type of pneumonia caused by *Legionella pneumophila* (gram-negative bacteria), which thrive in warm, moist environments, e.g. air conditioning units, spas. The infection can cause severe congestion and consolidation and necrosis of lung tissue that potentially may have fatal consequences. Signs and symptoms associated with typical pneumonia include: sudden onset with pyrexia and chills, fatigue, dyspnoea, tachypnoea, tachycardia, productive cough and pleuritic pain.

**Atypical pneumonia**

Atypical pneumonia, or primary atypical pneumonia, can be caused by either viral or mycoplasmic pathogens and is associated with a patchy involvement of the lung that is predominantly confined to the alveolar septum or interstitium (support tissue). The term atypical refers to the lack of consolidation, absence of alveolar exudate, production of moderate amounts of white sputum and a slightly elevated white blood cell count. The most common causative agents of atypical pneumonia are *Chlamydia pneumoniae*, *Mycoplasma pneumoniae* and *Legionella pneumophila* (Arnold et al., 2016) and it is common in older children and young adults. Viruses responsible for causing atypical pneumonia include influenza types A and B, respiratory syncytial virus, adenovirus, rhinovirus, rubella and varicella viruses (Gu et al., 2017). Pathogens responsible for atypical pneumonia cause initial destruction of ciliated epithelium of the distal airway cellular material and impair respiratory defences. In doing so they predispose the respiratory tract to secondary bacterial infections. Signs and symptoms associated with atypical pneumonia are usually vague but can include a non-productive cough (absence of exudate in alveoli), mild fever, malaise, myalgia, headache, hoarseness and a sore throat.

**Tuberculosis**

*Tuberculosis* (TB) is an infection caused by *Mycobacterium tuberculosis* (MTB) (an acid-fast bacilli), usually affecting the lungs but it may invade other body systems, e.g. skin (cutaneous TB), kidneys
(Fogel, 2015). It is highly contagious and spread is airborne by means of droplet nuclei that are harboured in the respiratory secretions of persons with active tuberculosis (Zuma et al., 2013). TB is the ninth leading cause of death worldwide and it is the leading cause of death from a single infectious agent (WHO, 2017). Cruz-Knight and Blake-Gumbs (2013) state that approximately one-third of the world’s population is latently infected with MTB. Whilst the incidence of TB is dropping globally at a rate of approximately 2% annually, the burden remains high with over 6 million new cases being diagnosed annually (WHO, 2017). Drug-resistant TB remains a threat; of the new cases reported in 2016, 600,000 were resistant to rifampicin (the most effective first-line drug) and, alarmingly, 490,000 of these cases were multidrug resistant (MDR-TB) (WHO, 2017).

As previously stated, MTB is spread from person-to-person via airborne droplets. Salgame et al. (2015) identify that a specific, complex interplay between host and pathogen with the environment determines the outcome of MTB infection, resulting in one of three possible outcomes: cure, latency or active disease. The virulence of the strain, intensity of exposure, size of bacterial inoculum and host factors (e.g. age, comorbidities) can contribute to the possible outcomes (Salgame et al., 2015). The majority of people mount an effective response with successful inhibition of the growth of MTB and the bacteria becomes dormant and inactive, i.e. latent (Thillai et al., 2014). Immunocompetent latent individuals infected with MTB do not present symptoms and do not transmit the disease to others (Fogel, 2015). Risk groups and factors for the development of the disease are presented in Box 14.1.

**Box 14.1 Risk groups and factors for the development of tuberculosis**

- Young adults – male > female
- People living in developing countries
- Poverty (higher level = higher risk)
- Health care workers who are exposed to disease on a frequent basis
- Those with compromised/weakened immune systems
- Cancer
- Human immunodeficiency virus (HIV)
- Smoking
- Host deficiency in IL-2-promoting T-helper 1 response (de Martino et al., 2014)
- Foreign-born individuals living in impoverished areas
- Undernutrition

_Sources_: Fogel, 2015; WHO, 2017

Once inhaled, MTB droplets pass down the bronchial tree and deposit in peripheral alveoli and cause non-specific pneumonitis (localised inflammation). Some MTB may migrate through the lymphatic system and lodge in the lymph nodes where they encounter lymphocytes and initiate an immune response. Activation of alveolar macrophages leads to bacilli being engulfed by phagocytes. Macrophages initiate a cell-mediated immune response and the infection is contained. Whilst the bacilli are phagocytosed and contained, they resist lysosomal killing and continue to multiply inside the alveolar macrophage. Macrophages degrade the bacilli and present the antigens to helper CD4+ T lymphocytes. The sensitised T cells further stimulate the macrophages to increase the concentration of lytic enzymes, increasing the ability to destroy the bacilli. However, they also damage the lung tissue. In the immunocompetent person, this cell-mediated immune response leads to the development of a granulomatous lesion known as a **Ghon focus**. The Ghon focus contains tubercle bacilli, modified macrophages and other immune cells.
Infected tissue inside the Ghon focus dies and undergoes necrosis that subsequently produces a cheese-like material of dead cells known as caseous necrosis. Tubercle bacilli (free or inside macrophages) drain along lymph channels to the tracheobronchial lymph nodes of the affected lung and stimulate the formation of caseous granulomas. The primary lung lesion and lymph node granulomas are collectively known as the Ghon complex (Figure 14.1). As the Ghon complex heals it undergoes shrinkage, fibrous scarring and calcification. Tuberculosis may remain dormant for life; however, reactivation and progressive disease may occur when the immune system is compromised or impaired.

Signs and symptoms are not always initially apparent but may include: mild fever, cough with purulent sputum, fatigue and night sweats. As the disease progresses, dyspnoea, wheezing, haemoptysis, chest pain, weight loss and anorexia may become apparent.

Figure 14.1  Ghon complex

Primary TB

Primary TB develops in previously unexposed, unsensitised people (Zuma et al., 2013). Most people with primary TB are asymptomatic and go on to develop latent TB whereby the cell-mediated response (T lymphocytes and macrophages) limits the spread as the organisms are surrounded by granulomas. In approximately 5% of people newly infected with MTB there will be an inadequate response that leads to progressive primary tuberculosis with the destruction of lung tissue and spread to multiple sites within the lung (Zuma et al., 2013). Most commonly affected are young children with immature immune systems, those with HIV infection or other immunodeficiency disorders, or those receiving immunosuppressive therapy.

Secondary TB

Secondary TB refers to the reinfection or reactivation of the disease in a person with some immunity. The tubercle ruptures and re-establishes an active infection, and bacteria can spread through the lungs via the
bronchioles. The disease tends initially to remain localised, often in apices of the lung. It tends to recur when there are impaired body defence mechanisms.

**BRONCHIOLITIS**

*Bronchiolitis* is the most common lower respiratory tract infection in the first year of life, affecting one in five children with 2–3% of these requiring hospitalisation (Ricci et al., 2015). It is caused by a range of viruses, including the respiratory syncytial virus, parainfluenza virus and adenoviruses (Meissner, 2016). However, *mycoplasmas* may also cause the disease. Children usually present with a *coryzal illness* (head cold, inflammation of nasal cavities) that progresses over 3–5 days to a troublesome cough, dyspnoea, tachypnoea, wheeze, crackles and difficulty feeding (Ricci et al., 2015). It is associated with an increased risk of chronic respiratory conditions such as asthma. However, it is unknown if it actually causes these conditions (NICE, 2017b).

The infection produces an inflammation and necrosis of the small bronchi and bronchioles accompanied by oedema, increased secretions and reflex *bronchospasm* that lead to obstruction (partial or complete). Partial obstruction of the lungs may cause hyperinflation and air trapping or alveolar collapse. In complete obstruction, air becomes trapped distal to the obstruction and may cause atelectasis or non-aeration, hypoxaemia and in severe cases hypercapnia.

**DISORDERS OF THE PLEURA**

The pleura is a thin double-layered serous membrane that surrounds the lungs consisting of the parietal pleura (attached to the inside of the thoracic cavity) and the visceral pleura (attached to the surface of the lungs). Between the two layers is the pleural cavity, filled with pleural fluid that allows the layers to move freely over each other, preventing friction when breathing (Boore et al., 2016). The pleura and pleural fluid create a pressure gradient that assists with lung inflation. Anything that interferes with the pleura can have a negative impact on the ability to breathe properly and maintain effective gaseous exchange. The two disorders we are going to discuss in this chapter are *pneumothorax* and *pleural effusion*.

**Pneumothorax**

A pneumothorax refers to the presence of air in the pleural cavity caused by rupture of either the parietal or visceral pleura. Separation of the parietal and visceral pleural disrupts the negative pressure and changes the balance between elastic recoil forces of the lung and chest wall. A pneumothorax can cause either partial or complete collapse of the affected lung, which recoils towards the hilum (Figure 14.2). There are various types of pneumothorax: open/traumatic, closed/spontaneous and tension.

- *Spontaneous*: occurs when an air-filled bleb or blister on the surface of the lung ruptures (Weldon and Williams, 2012). Blebs are usually situated at the apices of the lungs. Following rupture of the bleb, atmospheric air from the airways enters the pleural cavity and changes the pressure gradient. Alveolar pressure is greater than pleural pressure so air flows from the alveoli to the pleural space; air takes up space, thereby restricting lung expansion and causing the lung to collapse. Spontaneous pneumothoraces can be further subdivided into primary and secondary (Weldon and Williams, 2012).
Primary spontaneous – spontaneous rupture of blebs that occur unexpectedly in healthy individuals (predominantly males) between the ages of 20 and 40 years. Tall, thin people are at higher risk as it is suggested that pleural pressure from top to bottom of the lung is greater and thus may contribute to the development of blebs (Weldon and Williams, 2012). Smoking is also a contributory factor as it is thought to cause inflammation of the small airways.

Secondary spontaneous – occurs as a complication associated with underlying lung disease and tends to be more serious. Various types of lung disease may be associated with secondary spontaneous pneumothorax, including asthma, tuberculosis, cystic fibrosis and sarcoidosis.

- **Traumatic**: may be caused by either penetrating or non-penetrating chest injuries. The most common cause is fractured or dislocated ribs that subsequently puncture the pleura.
- **Tension**: occurs when intrapleural pressure exceeds atmospheric pressure (Walden and Williams, 2012). Air enters the pleural cavity on inhalation but cannot leave on exhalation, resulting in a rapid increase in pressure within the chest, making it a life-threatening condition. Due to the increased pressure, compression of the unaffected lung and mediastinal shift to the opposite side occurs, leading to compression of the vena cava. This reduces venous return to the heart and results in reduced cardiac output.

Signs and symptoms associated with a pneumothorax include **dyspnoea**, cough and chest pain. **Atelectasis** also manifests and there will be reduced air entry and breath sounds on the affected lung(s). There may also be unequal, asymmetrical chest rise and fall and mediastinal shift depending on the severity of pneumothorax; this may cause tracheal deviation. Over time, **hypoxia** results and initiates a sympathetic nervous system response, leading to tachycardia, pallor and anxiety; decreased venous return will lead to hypotension.
Pleural effusion

Pleural effusion refers to an abnormal collection of fluid in the pleural cavity (Saguil et al., 2014). Whilst a small amount of fluid is usually present to allow for lubrication of the pleural membrane, a pleural effusion occurs when there are large amounts of fluid in the pleural cavity. This excess fluid firstly increases the pressure within the pleural cavity and then causes separation of the pleural membranes. This prevents cohesion during inspiration, thereby preventing expansion of the lung, leading to atelectasis. Atelectasis on the affected side and shift of the mediastinal contents towards the unaffected lung limit expansion and gaseous exchange. Venous return in the inferior vena cava and cardiac filling become impaired due to increased pressure in the mediastinum when the pleural effusion is large. There are a number of types of pleural effusions that are characterised by the presence of substances in them: hydrothorax (serous fluid, transudative or exudative), empyema (pus), chylothorax (chyle) and haemothorax (blood) (Weldon and Williams, 2012):

- **Hydrothorax**: refers to the collection of serous fluid in the pleural cavity caused by increased **hydrostatic pressure** or decreased **osmotic pressure** in blood vessels, leading to a shift of fluid out of blood vessels into the potential space in the pleural cavity. May occur secondary to cardiac failure, liver or kidney disease.
- **Empyema**: refers to infection in the pleural cavity, resulting in pus (purulent fluid containing glucose, proteins, leukocytes and debris from dead cells and tissue) accumulation. It usually occurs as a result of infection, usually pneumonia.
- **Chylothorax**: refers to the presence of lymphatic fluid in the pleural space secondary to leakage from the thoracic duct or one of its main tributaries due to trauma, **malignant infiltration** that prevents the transport of chyle from thoracic duct to central circulation or inflammation.
- **Haemothorax**: refers to the presence of blood in the pleural cavity due to chest trauma, tumours, aortic **aneurysm** rupture or chest surgery.

Signs and symptoms associated with pleural effusion will vary according to the cause (Weldon and Williams, 2012). However, as previously noted, lung expansion on the affected side will be decreased proportionally to the amount of fluid collected in the pleural cavity. Dyspnoea is the most common symptom associated with it; the person may also complain of chest discomfort/pleural pain. There will also be decreased breath sounds on auscultation and dullness on percussion. If the person has an empyema, they may also present with fever, tachycardia and cough.

**PULMONARY DISORDERS**

**Atelectasis**

The term atelectasis is derived from the Greek words *ateles* and *ektasis*, which mean incomplete expansion or collapse resulting in reduced or absent gas exchange. Atelectasis may affect all or part of a lung. There are three different types of atelectasis: compression atelectasis, absorption atelectasis and surfactant impairment (Table 14.4). It may be present at birth (primary atelectasis) or develop later in life (acquired atelectasis).

Signs and symptoms associated with atelectasis include dyspnoea, **tachypnoea**, cyanosis, signs of hypoxaemia (e.g. altered **consciousness** levels), tachycardia, reduced chest expansion and absence of breath sounds on the affected side.

**Obstructive lung disorders**

This is a group of disorders caused by an obstruction or limitation to airflow characterised by a reduction in expiratory airflow and respiratory symptoms. The main disorders of obstructive lung disease are **asthma**, chronic bronchitis and **emphysema**.
### Table 14.4  Different types of atelectasis

<table>
<thead>
<tr>
<th>Type</th>
<th>Cause</th>
</tr>
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<tbody>
<tr>
<td>Compression atelectasis</td>
<td>External pressure exerted by tumour, fluid or air in pleural space, or by abdominal distension, causing alveolar collapse</td>
</tr>
<tr>
<td>Absorption atelectasis</td>
<td>Removal of air from obstructed/hypoventilated alveoli, inhalation of: anaesthetic agents, concentrated $O_2$</td>
</tr>
<tr>
<td>Surfactant impairment</td>
<td>Decreased production or inactivation of surfactant due to premature birth, acute respiratory distress syndrome (ARDS), anaesthetics, mechanical ventilation</td>
</tr>
</tbody>
</table>

### Bronchial asthma

**Bronchial asthma** is a chronic inflammatory disease and the most prevalent chronic respiratory disease. The World Health Organisation (WHO, 2018a) states that between 100 million and 150 million people worldwide suffer from asthma and this number continues to grow. Asthma is linked to genetic and environmental factors and it is classified as atopic/allergic (triggered by allergic sensitisation) or non-atopic/non-allergic. The Global Initiative for Asthma defines asthma as a disease characterised by chronic airway inflammation with a history of wheeze, shortness of breath, chest tightness and cough (GINA, 2018). This can be due to inflammation, oedema and mucus production (Papi et al., 2018).

Asthma is associated with the release of inflammatory mediators from mast cells in the airways and this leads to a response clinically manifested as expiratory wheeze, experience of chest tightness, dyspnoea, tachypnoea and cough. In atopic (allergic) asthma the inflammatory response is triggered by allergens and is often associated with other allergic conditions such as eczema, rhinitis or food allergy and a positive family history of the disease (Papi et al., 2018). Non-atopic (non-allergic) asthma occurs in people with no history of allergy and often develops in middle age. In both types, bronchospasm is triggered by exposure to a stimulant such as viral respiratory infection, smoke, exercise and cold air.

#### Activity 14.1: Apply

**Living with asthma**

As a person-centred practitioner, it is important to understand the lived experience of asthma in order that you can support people in their illness. Watch the following video to help you gain some insight into that lived experience and reflect on how it may help you in your practice. If you are using the eBook just click on the play button. Alternatively go to [https://study.sagepub.com/essentialpatho/videos](https://study.sagepub.com/essentialpatho/videos)

#### Allergic (atopic) asthma

The allergic (atopic) form of asthma is associated with exposure to a stimulant, usually an allergen, that induces a type 1 hypersensitivity response (see Chapter 7). The mechanisms of response can be described in two distinct phases: the early phase and the late or delayed phase. During the early phase
response following antigen exposure to the bronchial mucosa, dendritic cells activate T-helper-2 cells to produce interleukins (ILs) IL-5, IL-4, IL-13.

IL-5 activates eosinophils and IL-4 and IL-13 stimulate B lymphocytes to produce immunoglobulin E (IgE) (Russell and Brightling, 2017). IL-13 also stimulates mucus production and eosinophil recruitment to the lung mucosa. IgE antibodies attach to the receptors on the mast cell surface, causing them to degranulate their contents. The inflammatory mediators histamine, bradykinins, interleukins, tumour necrosis factor, prostaglandins and leukotrienes are released, causing vasodilation and altered capillary permeability and resulting in mucosal oedema and smooth muscle contraction with subsequent bronchospasm and mucus secretion (Figure 14.3), narrowing the airways and obstructing airflow. Eosinophil products cause damage directly to the lung epithelial tissue and may also cause bronchoconstriction through the release of leukotrienes (Diver et al., 2018).

The late phase response occurs 4–8 hours after the early phase and involves the recruitment of more eosinophils, neutrophils and lymphocytes to the lung tissue, which increases and sustains the inflammatory response. Recurrent episodes of inflammation lead to injury to the epithelium and the formation of scar tissue (Kudo et al., 2013). Ciliated epithelial cells are damaged and mucus accumulates in the lumen of the airways. Long-term airway damage leads to airway remodelling that results in goblet cell hyperplasia, epithelial damage and cilia dysfunction with increased smooth muscle mass and increased vascularity. This leads to a thickening of the airway wall and narrowing of the airway lumen (Brightling et al., 2012). This, along with an increased production of mucus, can lead to smaller airways becoming completely blocked (Russell and Brightling, 2017).

Non-allergic (non-atopic) asthma

Asthma can be triggered by non-allergic stimulants, for example exercise, drug-induced or respiratory infection. Exercise can induce bronchoconstriction and is thought to be due to heat loss, dehydration and increased osmolarity of the respiratory mucosa, triggering bronchospasm (Smoliga et al., 2016). Drug-induced asthma is mainly associated with aspirin ingestion and can lead to bronchospasm, rhinorrhea (when the nasal cavity is filled with large amounts of mucus), rash and itching. It is thought to involve abnormal pathways in prostaglandin metabolism and release of leukotrienes, causing bronchoconstriction (Rajan et al., 2015). Respiratory tract infection (RTI), particularly viral infection, causes epithelial damage and stimulates IgE antibodies against the virus (Wos et al., 2008). It has been suggested that there is an increased risk for asthma development associated with recurrent RTIs in childhood (Del Giacco et al., 2017). RTIs also increase hyper-responsiveness of the airways to other triggers. Airway hyper-responsiveness is a feature of asthma where airway smooth muscle is hypercontractile, resulting in airway narrowing. This can result from direct or indirect stimuli; the degree of hyper-responsiveness is determined by the number of mast cells affected (Diver et al., 2018).

Bronchoconstriction, inflammation and secretions lead to airway obstruction. Constriction of the smooth muscle in the walls of the airways narrows the lumen and increases resistance to airflow, particularly on expiration (Figure 14.4). Normally during expiration, the elastic recoil of the lungs decreases the diameter of the airways but air can still flow out. Where there is high airway resistance the airways collapse just before expiration, trapping air in the alveoli and causing hyperinflation; this leads to non-uniform ventilation. Alveolar gas pressure increases and perfusion decreases, leading to ventilation–perfusion (V/Q) mismatching. Hypoxaemia results. However, due to hyperventilation, CO₂ levels initially remain normal or may fall with associated respiratory alkalosis. Continued hyperinflation reduces the effectiveness of the respiratory muscles, leading to hypoventilation and reduced tidal volumes that subsequently result in systemic hypoxaemia, hypercapnia and respiratory acidosis.
Allergen/antigen exposure

- Dendritic cells activate T-helper 2 cells
  - IL5 production
    - Eosinophils activated
      - Epithelial tissue damage
      - Leukotrienes release
  - IL4 production
    - Stimulate B lymphocytes to produce IgE
      - IgE antibodies attach to mast cell receptors
      - Degranulation of contents
      - Inflammatory mediators released
        - Vasodilation
        - Mucosal oedema + smooth muscle contraction
          - Bronchospasm
            - ↓ Lumen of airways
            - Obstructed airflow
        - ↑ Capillary membrane permeability
          - Mucus secretion

Figure 14.3 Pathophysiology of asthma
A person with asthma is often asymptomatic in between attacks and the severity of an attack will determine the physical effects. A mild attack shows signs of expiratory wheezing, dyspnoea, tachypnoea and the person may complain of chest tightness and fatigue. A more severe attack is associated with dyspnoea, use of the sternocleidomastoid and scalene accessory muscles, and inspiratory as well as expiratory wheeze. Oxygen saturations may fall below 90% and arterial blood gases indicate hypoxaemia and later hypercapnia.

**Status asthmaticus** occurs where bronchospasm has not responded to bronchodilators and or anti-inflammatory drugs. Hypoxaemia worsens, and hypercapnia leads to respiratory acidosis. The person may appear cyanotic and there may be very little airflow and ventilation, requiring emergency medical intervention and mechanical ventilation.

**Diagnosis**

Diagnosis is based on history, including family history of asthma or allergens, and history of recurrent episodes of cough and wheezing or shortness of breath. Pulmonary function tests are an important part of diagnosis and management, using spirometry. A forced expiratory volume test measures the amount of air that can be forcibly expired in one second (FEV₁) and peak expiratory flow rate (PEFR), measured in litres per second, is the fastest rate air can flow out on expiration. These tests provide an indication of expiration ability and airway obstruction. Forced vital capacity (FVC) is the amount of air exhaled forcefully and quickly after inhaling as much as you can. A FEV₁/FVC ratio of less than 70% is a positive test for obstructive airway disease.

*Source: NICE, 2017a*
Treatment
Self-management and education play a key role in the approach to asthma management. Avoidance of known triggers and risk factors should reduce the frequency and risk of asthmatic attacks. Pharmacological therapy needs to be monitored and adjusted to find the minimum effective dose for the individual. A short-acting inhaled beta-2 agonist is used to relax bronchial smooth muscle and is used in people with low risk for exacerbations and who have symptoms less than twice per month (GINA, 2018). A regular low-dose inhaled corticosteroid to reduce inflammation and improve symptom control is used in combination with the short-acting beta-2 agonist. A long-acting beta-2 agonist can be used where symptoms are not well controlled (GINA, 2018).

Severe or uncontrolled asthma is where there is poor symptom control and frequent severe exacerbations (Chung et al., 2014). IgE monoclonal antibodies have been used to treat moderate to severe allergic asthma (Olin and Wechsler, 2014). Omalizumab was the first monoclonal antibody used for severe asthma and it works by binding to IgE to prevent it from attaching to the receptor on the surface of the mast cell, basophils and dendritic cells (Diver et al., 2018).

Chronic obstructive pulmonary disease (COPD)

Chronic obstructive pulmonary disease (COPD) refers to a group of respiratory disorders that are characterised by persistent respiratory symptoms and airflow limitation due to abnormalities caused by exposure to noxious particles or gases (GOLD, 2016). It is a progressive disease and is associated with risk factors, in particular tobacco smoking, and exposure to respiratory irritants such as dust, chemicals and environmental pollution (Kim and Criner, 2013). COPD is the fourth leading cause of death in the world, accounting for 6% of all deaths globally. It is a major cause of morbidity and mortality and is in most cases a preventable disease (GBD [2015 Chronic Respiratory Disease Collaborators], 2017). There are two types of obstructive airway disease associated with COPD: emphysema, and chronic bronchitis. Whilst the two conditions have many similarities, they also display distinct differences. Table 14.5 identifies the characteristics of emphysema and chronic bronchitis.
Table 14.5  Characteristics of emphysema and chronic bronchitis

<table>
<thead>
<tr>
<th>Feature</th>
<th>Emphysema</th>
<th>Chronic bronchitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Health history</td>
<td>Generally healthy, but smoker</td>
<td>Recurrent chest infections</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Exacerbation of symptoms by irritants and</td>
</tr>
<tr>
<td></td>
<td></td>
<td>cold air, smoker</td>
</tr>
<tr>
<td>Cough/sputum</td>
<td>Minor</td>
<td>Significant/copious, purulent</td>
</tr>
<tr>
<td>Physical examination and</td>
<td>Cachetic, history of weight loss and protein</td>
<td>Tendency towards obesity, cyanotic,</td>
</tr>
<tr>
<td>general appearance</td>
<td>calorie malnutrition</td>
<td>polycythaemia, oedematous, distended neck</td>
</tr>
<tr>
<td></td>
<td></td>
<td>veins</td>
</tr>
<tr>
<td>Dyspnoea</td>
<td>Slowly progressive</td>
<td>Variable, often late in illness</td>
</tr>
<tr>
<td>Breath sounds</td>
<td>Quiet or diminished</td>
<td>Scattered wheezing, ronchi, rales</td>
</tr>
<tr>
<td>Chest appearance</td>
<td>Increase in anteroposterior diameter, barrel</td>
<td>Slight to marked increase in</td>
</tr>
<tr>
<td></td>
<td>chest, prominent accessory muscles of</td>
<td>anteroposterior diameter, pulmonary</td>
</tr>
<tr>
<td></td>
<td>respiration, limited diaphragmatic excursion</td>
<td>hypertension</td>
</tr>
<tr>
<td>ABGs</td>
<td>Near normal, ↓PaO₂, normal or</td>
<td>↓PaO₂, ↑PaCO₂</td>
</tr>
<tr>
<td></td>
<td>↓PaCO₂, hypercapnia (late stages)</td>
<td></td>
</tr>
<tr>
<td>Chest X-ray</td>
<td>Hyperinflation, flat diaphragm, widened</td>
<td>Congested lung fields, cardiac</td>
</tr>
<tr>
<td></td>
<td>intercostal margins</td>
<td>enlargement</td>
</tr>
</tbody>
</table>

Chronic bronchitis

The Global Initiative for Chronic Obstructive Lung Disease defines chronic bronchitis as a persistent cough and sputum production for at least 3 months per year for two consecutive years (GOLD, 2016). Exposure to respiratory irritants such as tobacco smoke or acute and chronic respiratory infections causes airway inflammation and the overproduction of mucus by goblet cells and hypersecretion from increased degranulation by neutrophil-mediated elastase (Kim and Criner, 2013). There is difficulty in clearing mucus due to poor ciliary function and ineffective cough. This causes narrowing of the lumen of the airway and obstruction to airflow. Initially, the larger airways are affected but with disease progression all airways are involved.

Inflammatory mechanisms responsible for mucus overproduction are attributed to Th1 cells (T-helper cells type 1) and specifically the subset Th17 cells (Kim and Criner, 2013). Interleukin 6 (IL-6) and interleukin 17 (IL-17) induce the production of proteins called mucins by lung epithelial cells which contribute to thick mucus production and the formation of mucus plugs, blocking the airway lumen. Impaired mucociliary clearance due to dysfunction and loss of cilia leads to the consolidation of mucus in the lungs.

Inflammatory infiltration of neutrophils, lymphocytes and macrophages in the bronchial wall leads to oedema of the bronchial mucosa and eventually fibrosis. These changes extend to the alveoli and further contribute to airway obstruction. Bacterial and viral infections are common and lead to exacerbations of COPD and amplification of alveolar injury (Tuder and Petrache, 2012). The narrowed airways increase resistance to airflow and cause obstruction, resulting in ventilation–perfusion mismatch with hypoxaemia and hypercapnia. Pulmonary blood vessels constrict in response to hypoxaemia, leading to pulmonary hypertension (Portillo et al., 2015) which can eventually lead to right-sided heart failure.

The classic signs of chronic bronchitis are a productive cough and prolonged expiration. Dyspnœa occurs late in the disease progression and cyanosis is often present as a result of hypoxaemia. The person usually has a history of smoking.
Emphysema

Emphysema is an obstructive airway disease characterised by destructive changes of the alveolar walls and irreversible enlargement of the alveolar sacs with loss of surface area for gas exchange (Tuder and Petrache, 2012) (Figure 14.5). The main characteristic of emphysema is the destruction of lung tissue rather than mucus production and inflammation as in chronic bronchitis. Abnormal, permanent enlargement of gas-exchange airways accompanied by destruction of alveolar walls without obvious fibrosis of the small airways contributes to airflow obstruction.

![Image of Emphysema and Healthy Alveoli]

**Figure 14.5  Structural alveolar changes in emphysema**

The distribution of damage to terminal respiratory units (acini) within the respiratory lobule and the extent of alveolar wall damage are used as a basis for the classification of emphysema. **Centriacinar** and **panacinar** emphysema are the most common forms of emphysema:

- **Centriacinar emphysema**: refers to the central or proximal parts of the acini and is associated with smokers. It is often seen in COPD.
- **Panacinar emphysema**: involves the acini at the terminal alveoli and is associated with alpha(α)-1-antitrypsin deficiency, an **autosomal recessive** inherited disorder. This enzyme inhibits the action of **proteolytic enzymes** released by neutrophils during inflammation. Absence of alpha-1-antitrypsin increases the risk of developing emphysema (Baraldo et al., 2015), particularly if the person smokes, as proteolytic enzymes are not inhibited and contribute to the damage of acini.
Prolonged exposure to a respiratory irritant such as tobacco smoke stimulates the inflammatory response and infiltration of inflammatory cells such as neutrophils and inflammatory mediators (i.e. leukotrienes, IL-8 and tumour necrosis factor [TNF]) in the lung tissue (Goldklang and Stockley, 2016). Proteases (enzymes that break down proteins) are released and there is inadequate production of protective anti-proteases to counteract the action of the proteases, resulting in the breakdown of elastin and destruction of alveolar walls.

The destruction of alveoli results in large air spaces known as bullae; these are not effective in gaseous exchange, leading to ventilation–perfusion mismatch and hypoxia. The loss of the elastic recoil makes expiration difficult and leads to air-trapping in the alveoli (Gelb et al., 2015). This causes hyperexpansion of the chest and increases the work of breathing. Destruction of pulmonary capillaries leads to pulmonary hypertension and cor pulmonale (enlargement of the right side of the heart secondary to lungs or pulmonary blood vessel disease) with right-sided heart failure.

People with emphysema have a history of increasing dyspnoea, particularly on exertion. Use of accessory muscles is evident and expiration is prolonged through pursed lips in an effort to exhale as much air as possible before the alveoli and small airways collapse. They may or may not have a cough and a wheeze. Hyperinflation of the lungs produces an increase in the anteroposterior dimensions of the chest, resulting in a barrel-shaped chest typical of a person with emphysema which further contributes to dyspnoea and activity limitation (Langer et al., 2014). The person with emphysema will often have a history of smoking.

**Bronchiectasis**

*Bronchiectasis* is characterised by the permanent dilation of the bronchi caused by destruction of the bronchial wall and elastic supporting tissue. There may be a genetic predisposition, or it may occur in conjunction with another respiratory disease such as cystic fibrosis and tuberculosis and a compromised host defence to infection (King, 2018). Bronchiectasis is associated with recurrent infection of the lower respiratory tract, which leads to a persistent inflammatory response and permanent dilation of the medium-sized bronchi and bronchioles (Chen et al., 2018). Chronic inflammation causes destruction of the central bronchial wall with collapse of the peripheral bronchi and bronchioles. Loss of ciliated columnar epithelium and production of copious secretions lead to obstruction of airflow (Pastue et al., 2010). The microorganisms that cause infection in bronchiectasis are present in the microbiome of the upper respiratory tract and include *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Pseudomonas aeruginosa* (King, 2018).

A chronic productive cough with copious foul-smelling sputum is characteristic of bronchiectasis as a result of these pathophysiological changes. The person may have a fever, dyspnoea, wheezing and haemoptysis. Systemic manifestations include night sweats, anaemia and weight loss (due to the increased work of breathing and associated energy expenditure). In severe cases, clubbing may be present in the fingertips. Clubbing is enlargement of the distal segment of the digit (fingers or toes). It is thought that vascular endothelial growth factor (VEGF) (a platelet-derived factor) is an important component in its development (Rajagopalan and Schwartz, 2018). Hypoxia stimulates the release of VEGF that induces vascular hyperplasia, oedema and proliferation of fibroblasts/osteoblasts at a peripheral level in the nail (Rajagopalan and Schwartz, 2018). Large megakaryocyte fragments enter the systemic circulation and affect distal sites, releasing growth factors including VEGF. Platelets in the vasculature of the fingertips release platelet-derived growth factor that subsequently stimulates growth, increased vascular permeability and monocyte and neutrophil chemotaxis, leading to the proliferation of vascular smooth muscle cells and fibroblasts (Figure 14.6). It is associated with disorders such as bronchiectasis and cystic fibrosis and the severity of clubbing reflects the severity of the respiratory disease.
Cystic Fibrosis (CF)

Cystic fibrosis is an inherited autosomal recessive disorder of the exocrine glands and is a major cause of severe respiratory disease in children and young adults. It affects 70,000 people worldwide and improved survival has led to increasing numbers reaching adulthood, with average survival to 40 years (Elborn et al., 2016).

Cystic fibrosis is a genetic disorder arising from a mutation on the cystic fibrosis transmembrane conductance regulator (CFTCR) gene on chromosome 7, resulting in a defective CFTCR protein (Stolz et al., 2015). This protein normally functions as a chloride channel in the epithelial cells lining the airways, the bile duct, the pancreas and the vas deferens. The defective protein means that chloride transport is impaired and both secretion and reabsorption can be affected. In the lung, the transport of chloride into the airway lumen is impaired which leads to increased absorption of sodium and water into the circulation, resulting in the secretion of thick tenacious mucus and dehydration of the mucociliary layer with a subsequent reduction in ciliary mobility and an ineffective clearing of mucus. The build-up of mucus obstructs the airways and provides a medium for recurrent pulmonary infection. Neutrophils release tissue-damaging proteases and oxidants that induce airway cells to destroy immunoglobulin G (IgG) and produce interleukin 8 (IL-8), which attracts more neutrophils and stimulates mucus secretion. Airway obstruction from mucus plugs and chronic inflammation and infection results in respiratory signs and symptoms.

Respiratory symptoms of CF include a persistent cough and the production of thick sputum and recurrent respiratory infections. Dyspnoea, tachypnoea and wheeze may be present. Over time, structural changes in the bronchial wall lead to bronchiectasis, and later signs of barrel chest and digital clubbing may be present. Chronic recurrent infection is common and microorganisms such as *Staphylococcus aureus* and *Haemophilus influenzae* are common in younger children (King, 2018). *Pseudomonas aeruginosa* colonises the lungs and leads to a decline in lung function (Harun et al., 2016).

Other manifestations of CF result from the impaired chloride transport in the sweat glands and the exocrine glands of the pancreas. In the sweat glands, the reabsorption of chloride and the subsequent reabsorption of sodium into the ducts of the glands fail, leading to a high concentration of sodium chloride in the sweat of the person with CF. The pancreatic and biliary ducts are affected by impaired sodium, chloride and potassium resorption, resulting in thick mucus production which blocks the pancreatic ducts. This in turn blocks the flow of digestive enzymes, leading to malabsorption of proteins, fats, carbohydrates and vitamins. Degenerative and fibrotic changes occur in the pancreas and gastrointestinal tract and diabetes can develop from the destruction of beta cells in the pancreas.
Diagnosis is based on clinical signs and diagnostic tests. Newborn screening includes a sweat chloride test to detect sodium and chloride levels in the sweat in excess of 60 mEq/l and blood tests for **immunoreactive trypsinogen** (IRT) (Farrell et al., 2017). IRT is a pancreatic enzyme precursor and it is elevated in infants and children with CF. Genetic testing for mutations in the **CFTR** gene can also be carried out (Farrell et al., 2017).

**Interstitial lung disease**

*Interstitial lung disease (ILD)* refers to a group of diffuse parenchymal lung disorders associated with substantial morbidity and mortality (Antoniou et al., 2014). They exert their effects on the collagen and elastic connective tissue found in the interstitium of the alveolar walls (Behr, 2012). ILD may occur in isolation or it may coexist alongside systemic disease (Behr, 2012); diminished lung compliance resulting in ‘stiff’ lungs that are difficult to inflate is the definitive hallmark of the condition. Increased work of breathing ensues as greater pressures need to be generated to inflate the lungs. Impaired gas exchange and hypoxaemia result due to damage of the alveolar epithelium and interstitial vasculature. As the disease progresses, respiratory failure may develop and may be associated with pulmonary hypertension or cor pulmonale.

ILD is initiated by some form of injury to the alveolar epithelium that causes an inflammatory response involving both the alveoli and the interstitium. Persistent injury leads to an accumulation of inflammatory and immune cells that cause damage to the lung tissue and development of fibrous scar tissue. Further development of fibrosis occurs as alveolar macrophages secrete a range of fibrogenic factors (e.g. fibroblast growth factor, platelet-derived growth factor) that attract fibroblasts and encourage their multiplication. Destruction of type I alveolar cells alongside an increase in type II alveolar cells chemotactically attracts further macrophages, cytokines and growth factors that further contribute to fibrotic changes.

Two of the most common ILDs are **idiopathic pulmonary fibrosis** (IPF) and sarcoidosis, therefore we will discuss both these disorders as they are frequently encountered in the people we care for.

**Idiopathic pulmonary fibrosis**

Idiopathic pulmonary fibrosis (IPF) is the most common disorder diagnosed among people with ILD; Antoniou et al. (2014) report that it is the most lethal disorder amongst ILDs. IPF is a chronic, progressive ILD of unknown aetiology that is difficult to diagnose and usually requires collaborative expertise to do so (NICE, 2017d). NICE (2017d) identifies clinical features that should be considered so that an early diagnosis can be made (Box 14.2). Two-thirds of those affected are people over the age of 60 years at the time of presentation. It affects men more than women. Risk factors for the disease include smoking and some occupations (e.g. farming, hairdressing, stone cutting and metal cutting) (Behr, 2012).

**Box 14.2 Clinical features of IPF**

- Age over 45 years
- Persistent breathlessness on exertion
- Persistent cough
- Bilateral inspiratory crackles when listening to the chest
- Clubbing of the fingers
- Normal spirometry or impaired spirometry, usually with a restrictive pattern but sometimes with an obstructive pattern
People presenting with IPF experience symptoms of breathlessness/dyspnoea initially on exertion and a cough (productive or non-productive); over time they will develop decreased lung function, cyanosis and cor pulmonale and have a reduced quality of life and ultimately death.

The rate of disease progression can vary greatly from person to person; the median survival time of someone with IPF is approximately 3 years from time of diagnosis, however 20% of people may live for more than 5 years (NICE, 2017d). Meyer (2014) acknowledges that ILD with extensive fibrosis is difficult to treat but appropriate therapies, including immunosuppressive anti-inflammatory therapies, oxygen therapy and pulmonary rehabilitation, can have a positive impact on quality of life and symptom palliation.

**Sarcoidosis**

Sarcoidosis is a systemic granulomatous disease process that may impact on any organ (in particular the lung) and can mimic other disease processes, especially malignancy or infection, making it difficult to diagnose (Parker et al., 2016). The disease usually affects people younger than 40 years but can occur in older people, affecting more females than males. The aetiology of sarcoidosis remains unclear. However, it is thought it may be linked to defective **human leucocyte antigen (HLA)** genes located in the **major histocompatibility complex (MHC)** (Baughman et al., 2011). The pathogenesis of sarcoidosis seems to involve the interplay of antigen, HLA class II molecules and T-cell receptors, with specific combinations of these three facets required for sarcoidosis to develop (Baughman et al., 2011). Sarcoidosis most likely requires exposure to one or more exogenous antigens. Infectious agents have long been suspected as possible causes of sarcoidosis; although it is unclear at present what these are specifically, it is thought that mycobacteria or *Propionibacterium acnes* may contribute to the disease (Baughman et al., 2011). Baughman et al. (2011) believe that the triggering antigen may vary depending on ethnicity, geographic location and individual genetic background.

The immune response is focused on the alveoli and is characterised by chronic inflammation, starting with polarisation of T lymphocytes to a Th1 phenotype, followed by cellular recruitment, especially macrophages and lymphocytes, that multiply and differentiate with the subsequent formation of the sarcoid granulomas (Baughman et al., 2011). The sarcoid granulomas do not show evidence of necrosis, however Chen et al. (2010) demonstrated that granulomas in sarcoidosis are characterised by extensive deposition of **serum amyloid**, a protein capable of starting an immune response and triggering cytokine release.

Signs and symptoms are variable, progression of the disease is unpredictable and any organ can be affected although mostly lungs, eyes and skin are involved. People may present with respiratory symptoms such as dyspnoea, non-productive cough and chest pain. They may also complain of fever, diaphoresis, anorexia, weight loss, fatigue and myalgia. The presence of skin plaques and **papules** is noted when there is skin...
involvement; involvement of the eyes leads to inflammation of the middle layer of the eye, i.e. uveitis. The person may experience periods of progressive chronicity, activity interspersed with periods of remission.

DISORDERS OF PULMONARY CIRCULATION

Disruption to blood flow through the lungs can be caused by the occlusion of blood vessels, increased pulmonary vascular resistance and destruction of the vascular bed that can lead to ventilation/perfusion mismatching. The effects of disrupted blood flow can range from mildly dysfunctional to severe and life-threatening. Two major problems associated with disruption in pulmonary blood flow are pulmonary embolism and pulmonary hypertension, both of which will be discussed.

Pulmonary embolism

Pulmonary embolism (PE) occurs when a blood-borne substance lodges in a branch of the pulmonary artery, occluding pulmonary vasculature (Figure 14.7). PE can originate from numerous sources, including deep vein thrombosis (DVT) (commonly in the lower extremities), tumours, fat (e.g. from a fracture), amniotic fluid, foreign bodies, air and sepsis (NICE, 2018). The most common source of PE is DVT; thrombus formation in the venous system occurs as a result of venous stasis, trauma (endothelial injury) and hypercoagulability. Collectively, these factors are known as Virchow’s triad (Merli et al., 2018). Risk factors for the development of PE are presented in Table 14.6.

![Figure 14.7 Pulmonary embolism](Illustrated by Shaun Mercier © SAGE Publications)
Table 14.6 Risk factors for the development of pulmonary embolism

<table>
<thead>
<tr>
<th>Stasis of blood flow</th>
<th>Prolonged immobilisation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Long aeroplane or other journeys</td>
</tr>
<tr>
<td></td>
<td>Diagnosis of DVT</td>
</tr>
<tr>
<td></td>
<td>Varicose veins</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Endothelial injury</th>
<th>Surgery within last 2 months</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fractures</td>
</tr>
<tr>
<td></td>
<td>Hypertension</td>
</tr>
<tr>
<td></td>
<td>Contact with substances that promote coagulation: e.g. implants, medical devices, cell membranes (platelets, monocytes in chronic inflammation)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hypercoagulability</th>
<th>Advancing age</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Obesity</td>
</tr>
<tr>
<td></td>
<td>Malignancy</td>
</tr>
<tr>
<td></td>
<td>Genetic factors: factor V Leiden, prothrombin gene mutations, inherited coagulation disorders</td>
</tr>
<tr>
<td></td>
<td>Hormones: pregnancy, use of oral contraceptive pill, hormone replacement therapy</td>
</tr>
</tbody>
</table>

In PE, the lung tissue is ventilated but not perfused, causing a V/Q mismatch that creates intrapulmonary dead space, resulting in impaired gas exchange and loss of alveolar surfactant. Over a period of several hours the alveoli collapse, resulting in worsening hypoxaemia. There is a reduction in blood flow to a cross-sectional area of the pulmonary bed that leads to the elevation of pulmonary arterial pressure and decreased cardiac output. The affected area of the lung is no longer perfused and may infarct, although this only happens on rare occasions as oxygen continues to be supplied by bronchial circulation and the airways (Merli et al., 2018).

Signs and symptoms of PE depend on the size and location of the obstruction. If the embolus is small and lodged in peripheries of the pulmonary artery, it may go unnoticed and be clinically silent, particularly in the elderly or acutely ill. If the embolus is moderate in size, the person may complain of chest pain, dyspnoea and a sense of apprehension. In some cases, they may present with haemoptysis or syncope. Persons with a large embolus usually present with a sudden collapse or crushing substernal chest pain; they may be cyanotic, tachycardic, hypotensive and diaphoretic; they may also have distension of the jugular vein. PEs are potentially life-threatening if they completely occlude the pulmonary vasculature as this can lead to right ventricular failure, cardiac arrest and ultimately death.

Pulmonary hypertension

Pulmonary hypertension is defined as an increase in mean pulmonary arterial pressure (PAPm) greater than 25 mmHg at rest as assessed by right heart catheterisation (Galiè et al., 2016). It is a relatively common complication of chronic obstructive pulmonary disease and diffuse pulmonary lung disease (including ILD) that may have serious implications for the function of the right ventricle (Tseng et al., 2018). Pulmonary hypertension causes profound functional limitations and results in a poor quality of life (Babu et al., 2016). It can be categorised into five groups (Galiè et al., 2016):

1. Pulmonary arterial hypertension
2. Pulmonary hypertension due to left heart disease
3. Pulmonary hypertension due to lung disease with or without hypoxia
4. Chronic thromboembolic pulmonary hypertension
5. Pulmonary hypertension with unclear and/or multifactorial mechanisms.

**ACTIVITY 14.3: GO DEEPER**

**Classification of pulmonary hypertension**

To find out more about the five groups of pulmonary hypertension, read the 2015 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension. The article is openly available online but if you are using the eBook just click on the icon to access it.

The pathophysiology of pulmonary hypertension focuses on endothelial dysfunction and the interplay between the overproduction of powerful vasoconstrictors (e.g. thromboxane, endothelin) and the underproduction of the vasodilators (e.g. prostacyclin and nitric oxide). Remodelling (fibrosis and thickening) of the vessel walls occurs due to the release of growth vascular factors and subsequent narrowing of the lumen and abnormal vasoconstriction (Gao and Raj, 2017). Resistance to pulmonary blood flow ensues, thereby increasing pressure within the pulmonary arteries and right ventricle; there is a reduction in lung volumes and gas exchange is impaired. Right ventricular workload is increased as pressure and resistance continue to rise, resulting in right ventricular hypertrophy followed by right-sided heart failure.

Signs and symptoms of pulmonary hypertension are non-specific and are mainly related to the progression of right-sided heart failure (Galiè et al., 2016). Initially symptoms are induced by exertion and include dyspnoea, shortness of breath, fatigue, weakness, angina and syncope. On occasion, the person may present with a dry cough and exercise-induced nausea and vomiting (Galiè et al., 2016). As the disease progresses and reaches advanced stages, symptoms begin to occur at rest, with increased right ventricle dysfunction, abdominal distension and ankle oedema more likely to develop.

**ACUTE RESPIRATORY DISORDERS**

Acute respiratory disorders refer to disruptions in gaseous exchange that are life-threatening with a high morbidity and mortality rate. Acute respiratory disorders include acute lung injury (ALI), acute respiratory distress syndrome (ARDS) and acute respiratory failure.

**Acute lung injury and acute respiratory distress syndrome**

Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) are manifestations of an inflammatory response of the lung to an insult either directly or indirectly (Ragaller and Richter, 2010). ALI and ARDS are characterised by severe hypoxaemia, hypercapnia, diffuse infiltrate on chest X-ray and substantial reduction in lung compliance. ALI can be defined as an acute lung disease with bilateral pulmonary infiltrate consistent with oedema and with no evidence of left atrial hypertension (Ragaller and Richter, 2010).
ARDS can be defined as an acute inflammatory lung injury associated with increased pulmonary vascular permeability, increased lung weight and loss of aerated tissue (Bellani et al., 2016). The conditions are differentiated by the difference in the extent of hypoxaemia determined by the ratio of partial pressure of oxygen in arterial blood (PaO$_2$) to the fraction of inspired oxygen (FiO$_2$) (Saguil and Fargo, 2012). ALI and ARDS can be caused by a variety of insults, including aspiration of gastric contents, trauma, sepsis, acute pancreatitis, disseminated intravascular coagulation and reactions to drugs/toxins.

The pathophysiology of ALI and ARDS is unclear although both local and systemic inflammatory responses occur. Mathiay et al. (2012) suggest that dysregulation of the inflammatory response, the accumulation of neutrophils, uncontrolled activation of coagulation pathways and altered permeability of the endothelium and disruption of endothelial barriers play a role in their development. Activation and migration of neutrophils across the alveolar epithelial surfaces result in the release of cytokines, proteases and reactive oxygen species that cause increased permeability and damage to alveolar type I and type II cells. Increased permeability allows the movement of fluid, plasma protein and blood cells from the vascular compartment to move into the interstitium and alveoli of the lung. Pulmonary oedema, hyaline membrane formation and the loss of alveolar surfactant decrease lung compliance, increasing intrapulmonary shunting of blood (V/Q mismatching), impairing gas exchange and resulting in hypoxaemia. At the bedside, ALI and ARDS culminate in life-threatening hypoxia, hypercapnia, acidosis and pulmonary hypertension, and require a fast and goal-oriented therapy without further lung damage (Ragaller and Richter, 2010).

**Acute respiratory failure**

Respiratory failure refers to the failure of the respiratory system to oxygenate the body or to eliminate carbon dioxide from the body. It may be due to acute disorders or trauma or can develop as a result of the course of a chronic disease. Respiratory failure is divided into two types: hypoxaemic respiratory failure (type I) and hypercapnic/hypoxaemic respiratory failure (type II) (Saguil and Fargo, 2012).

- **Type I (hypoxaemic):** characterised by low O$_2$ and normal or low PCO$_2$, often due to a dysfunction of gaseous exchange. Two major factors contribute to a decrease in oxygen level: V/Q mismatching and impaired diffusion. V/Q mismatching occurs when areas of the lungs are perfused but not ventilated, or ventilated but not perfused. This could be due to hypoventilation or decreased cardiac output. Impaired diffusion refers to the disruption in gaseous exchange between the alveoli and pulmonary circulation due to permeability of the alveolar surface or an increase in the distance for diffusion.
- **Type II (hypercapnic):** characterised by low O$_2$ with high PCO$_2$, often caused by a dysfunction of alveolar ventilation. Type II respiratory failure is usually caused by conditions that occur outside of the respiratory system, including depression of the central nervous system (drug/alcohol induced, brain injury); conditions that affect nerve supply to the respiratory system (Guillain-Barré syndrome, spinal cord injury); disorders of the respiratory muscles (muscular dystrophy); or thoracic cage disorders (scoliosis).

Signs and symptoms of acute respiratory failure are associated with hypoxaemia or hypercapnia and the clinical manifestations that occur because of this.

**CHAPTER SUMMARY**

In this chapter you will have learned about disorders of the respiratory system across the life span and how they impact not only physiologically but also how they influence health-related quality of life. To be an effective person-centred practitioner, you need to be relational when considering these disorders; being compassionate and working with someone’s belief system and values will enable you to care for them within the context of their culture and social structure.
**KEY POINTS**

- Hypoventilation refers to inadequate alveolar ventilation compared to the metabolic demands of the body. It occurs when the minute volume is reduced and is caused by changes in the mechanics of breathing or due to changes in the neurological control of breathing.

- Hyperventilation refers to increased alveolar ventilation that exceeds the metabolic demands of the body. It occurs when the excretion rate of CO$_2$ exceeds that which is produced by cellular metabolism.

- Hypoxia refers to a reduction in tissue oxygenation and can result from pulmonary alterations, or other abnormalities that are not related to pulmonary function, e.g. decreased cardiac output. Hypoxaemia refers to a reduction of oxygen in arterial blood and is caused by pulmonary dysfunction and may lead to hypoxia.

- Hypercapnia refers to an increase in CO$_2$ content of arterial blood and is usually caused by disorders that lead to hypoventilation or V/Q mismatching.

- Respiratory disease can be categorised into three phases: respiratory impairment, respiratory insufficiency and respiratory failure.

- Respiratory tract infections (RTIs) refer to infectious diseases that can affect any part of the respiratory tract; they tend to be discussed in terms of upper respiratory tract infections (URTI), i.e. infection of nose, oropharynx or larynx, or lower respiratory tract infections (LRTI), i.e. lower airways and lungs.

- Infections of the upper respiratory tract include: the common cold, influenza, rhinitis, sinusitis and otitis media. Infections of the lower respiratory tract include: bronchiolitis, pneumonia and tuberculosis.

- Pneumonia refers to infection of the pulmonary parenchyma, i.e. bronchioles and alveoli. It occurs when the normal defence mechanisms are bypassed and a pathogen gains access to the lower respiratory tract, causing an extensive inflammatory response. Pneumonias can be classified in a number of ways based on the pathogen (typical or atypical) or the area of infection (lobar pneumonia or bronchopneumonia). Pneumonias may also be classified according to the setting in which they occur and referred to as community-acquired pneumonia (CAP), hospital-acquired pneumonia (HAP), health care-associated pneumonia (HCAP) or ventilator-associated pneumonia (VAP).

- Tuberculosis (TB) is an infection caused by *Mycobacterium tuberculosis* (MTB) (an acid-fast bacilli), usually affecting the lungs but it may invade other body systems. It is highly contagious and the spread is airborne by means of droplet nuclei that are harboured in the respiratory secretions of persons with active tuberculosis. It is the ninth leading cause of death worldwide and is the leading cause of death from a single infectious agent.

- A pneumothorax refers to the presence of air in the pleural cavity caused by rupture of either the parietal or visceral pleura. A pneumothorax can cause either partial or complete collapse of the affected lung.

- Pleural effusion refers to an abnormal collection of fluid in the pleural cavity. Types of pleural effusions are characterised by the presence of substances in them: hydrothorax (serous fluid, transudative or exudative), empyema (pus), chylothorax (chyle) and haemothorax (blood).
• Obstructive lung disorders are a group of diseases caused by obstruction or limitation to airflow. The main disorders of obstructive lung disease are asthma, chronic bronchitis and emphysema.

• Chronic obstructive pulmonary disease (COPD) is an umbrella term for a group of disorders that cause airway obstruction, particularly chronic bronchitis and emphysema. Tobacco smoke is a primary risk factor for COPD.

• Bronchiectasis is characterised by the permanent dilation of the bronchi caused by destruction of the bronchial wall and elastic supporting tissue.

• Cystic fibrosis is an autosomal recessive disorder of defective chloride transport, resulting in thick mucus production in the airways and the glandular ducts.

• Interstitial lung disease (ILD) refers to a group of diffuse parenchymal lung disorders that exert their effects on the collagen and elastic connective tissue found in the interstitium of the alveolar walls. Two of the most common ILD are idiopathic pulmonary fibrosis and sarcoidosis.

• Pulmonary embolism (PE) occurs when a blood-borne substance lodges in a branch of the pulmonary artery occluding pulmonary vasculature.

• Pulmonary hypertension is defined as an increase in mean pulmonary arterial pressure (PAPm) greater than 25 mmHg at rest as assessed by right heart catheterisation.

• Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) are manifestations of an inflammatory response of the lung to an insult either directly or indirectly. They are characterised by severe hypoxaemia, hypercapnia, diffuse infiltrate on chest X-ray and substantial reduction in lung compliance.

• Respiratory failure refers to the failure of the respiratory system to oxygenate the body or to eliminate carbon dioxide from the body. It can be divided into two types: hypoxaemic respiratory failure (type I) and hypercapnic/hypoxaemic respiratory failure (type II).

In this chapter you will have learned about a variety of disorders of oxygenation and carbon dioxide elimination; these can be complex and in order to check your understanding, the following questions will help you confirm your knowledge and application.

Answers are available online. If you are using the eBook just click on the answers icon below. Alternatively go to https://study.sagepub.com/essentialpatho/answers

1. Identify and briefly explain the different types of hypoxia.

2. Identify and briefly explain the defence mechanisms of the respiratory tract function.

3. Briefly discuss the pathophysiology of pneumonia, indicating the different types.

4. Identify the risk groups and factors for developing tuberculosis.
What are the signs and symptoms of tuberculosis?

Briefly explain what a pleural effusion is and identify the different types.

What are the different types of atelectasis?

Explain the inflammatory response in atopic asthma.

Describe the physiological mechanism of ‘air trapping’.

Explain the inflammatory mechanisms responsible for mucous overproduction in chronic bronchitis.

Describe the types of emphysema.

Explain the physiological mechanisms that lead to thick mucus secretions in cystic fibrosis.

Identify the sources that can cause pulmonary embolism.

Differentiate between Type I and Type II respiratory failure.

REVISE
• Further revision and learning opportunities are available online

ACE YOUR ASSESSMENT
• Test yourself away from the book with Extra multiple choice questions
• Learn and revise terminology with Interactive flashcards

REFERENCES


Galiè, N., Humbert, M., Vachiery, J-L., Gibbs, S., Lang, I., Torbicki, A. et al. (2016) 2015 ESC/ERS guidelines for the diagnosis and treatment of pulmonary hypertension: the Joint Task Force for the Diagnosis and Treatment of Pulmonary Hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS). Endorsed by the Association for European Paediatric


My name is Liam. I am 16 years old and have chronic kidney disease. I have dialysis four times a week and know how vulnerable I am to infection. I joined a patient group and used the internet to get information about patient safety in dialysis units. We have hand-washing drummed into us by the staff all the time! Everyone wears gloves and uses those alcohol gels. I showed my nurse a website on my iPad after finding some patient information on gloves and how they can be dirtier than hands. I was worried about this, but she was really good and explained why gloves were worn and how staff should use them. Now I am much happier! I can see that everyone does the same thing and that everyone is treated the same as everybody else.

Liam, patient

Infection prevention and control is the responsibility of every healthcare professional – it is very important that germs with the potential to cause infections are not passed on to patients. Knowing and adhering to your hospital’s policies on IPC is a must; we need to protect our patients from dangers seen and unseen.

Charlie Clisby, NQN

Before reading this chapter, it will be helpful to:

- read key policies relating to infection prevention and control in your placement and any relevant information relating to hygiene, glove use, dermatitis or latex allergy
- spend a couple of hours with a local specialist IPC nurse to discuss hand hygiene and glove use and accompany them if audits of practice are being undertaken
- find out who your local hospital waste manager is and arrange to spend time with them to discuss the waste hierarchy and how this is being met.

Visit https://study.sagepub.com/essentialnursing2e to access a wealth of online resources for this chapter – watch out for the margin icons throughout the chapter. If you are using the interactive eBook, simply click on the margin icon to go straight to the resource.
INTRODUCTION

The prevention of infection is central to patient safety and underpins the safe provision of care in every nursing field and care setting. This chapter covers several practical clinical skills that are fundamental to nursing care and which you will need to incorporate into your everyday practice. IPC is a huge field, so it is not possible to cover all of the relevant issues within this chapter, but we can ensure that you understand the essential principles.

IPC can succinctly be described as the practical application of microbiology in clinical practice. Humans have always lived alongside micro-organisms, a relationship which can, at times, be beneficial and, at times, harmful to the host (us humans) – and possibly, and occasionally, you, as a nurse caring for a patient. Bacteria, viruses and fungi (commonly referred to as germs) deserve the greatest respect at all times. Our challenge as nurses is to manage their presence in the context of care settings wherever these may be, all of which offer opportunities for risks to patients and, on occasion, ourselves. It is important to remember that we can only ever reduce, not eradicate, the presence of germs which can lead to infection.

This chapter will assist you in thinking about the care you deliver to each patient in terms of IPC risks. Whatever care setting you are in – acute or community – and no matter what your field of nursing, the fundamental principles apply.

IPC AND STANDARD PRECAUTIONS

Several essential and commonly performed clinical practices are central to what is known as ‘standard precautions’. This term is generic and describes practices that, when used consistently and routinely for all patients, regardless of known or unknown infection status, are effective at interrupting the transmission of micro-organisms. Note: this is not the same as assuming all patients are infectious! When used properly, these precautions prevent the transfer of germs between patients and staff, and, of course, vice versa. This principle of interrupting the development of infection is central to the ‘chain of infection’.

Standard precautions commonly include but are not limited to:

- hand hygiene
- appropriate use of personal protective clothing (PPE) such as gloves, disposable aprons, respiratory masks and visors/goggles (as required for individual patients only based on the situation and risk presented)
- safe handling and disposal of sharps
- waste disposal
- management of linen.

We will start with hand hygiene, as this is a core element of standard precautions.

HAND HYGIENE

Hand hygiene is the most frequently talked about and contentious of IPC practices. As a student, you will witness both good and poor hand hygiene practice, so it is important that you have a sound understanding of the theoretical and practical aspects of hand hygiene and are confident both in your own practice and in recognising when others may be putting patients at risk. Think of your hands as tools that are used for work – they need to be clean, safe and cared for to maintain their use.
For the purposes of this chapter, we will define hand hygiene as a process for reducing or destroying the number of micro-organisms present on the hands through hand-washing or the use of hand sanitisers such as alcohol gel.

Hand hygiene on its own as an intervention to prevent infection will not be successful. It is one of a number of core practices that need to be undertaken together as a ‘package’ to protect both patients and staff. In practice, hand hygiene is frequently combined with other practices, such as the use of gloves, aprons and masks and aseptic technique. Glove use in particular is integral to hand hygiene, as gloves also have direct contact with patients and can become contaminated through care activities. Think gloves – think hands!

The importance of hand hygiene

Hand hygiene is important both within healthcare and in wider non-healthcare settings as a public health intervention to reduce the spread of communicable diseases (e.g. influenza) and infections caused via the faecal–oral route, such as norovirus.

The patient environment (be it their own home or another care setting) is important as a factor in the spread of infection. Contamination of the environment by the bacterial flora (for example, from the skin, bowel or respiratory tract) of patients or staff occurs easily due to their presence and the care activities undertaken. As staff move from one patient to another, or have contact with a patient’s immediate care environment (the area immediately surrounding the patient’s cot/bed/chair), hands ‘pick up’ micro-organisms which, if not removed through hand hygiene, can be passed to the next patient or deposited elsewhere. One of the most challenging aspects of hand hygiene is that the impact of not undertaking it may not become apparent for some time after the event – even if it can ever be conclusively attributed to a lack of hand hygiene. This absence of immediate visible consequences for patients, together with a lack of visible contamination of hands by micro-organisms, provides challenges to improving hand hygiene compliance.

“Frequent hand-washing is vital for the protection and safety of all patients. If infection is spread it can be life-threatening, especially to the most vulnerable of patients. As nurses who follow the Code we are there to do no harm, and I believe excellent hand hygiene skills are an element of upholding it.”

Alice Rowe, NQ RNMH

Gloves are a central element of standard precautions and are classified as personal protective equipment (PPE) when used for the protection of staff, but not when used to protect the patient. You will see gloves being used in nearly every care setting and speciality. Concerns exist, however, that gloves are used inappropriately in nursing practice, with their use and overuse putting both patients and staff at risk.

Serious risks exist when multiple care tasks are undertaken with the same patient, which involve moving from one part of the patient’s body to another. An excellent example of this is the provision of wound care following urinary catheter care. If hand hygiene is not performed following the urinary catheter care, any bacteria present on the hands (or gloves) of staff will be directly transferred to the wound area, which may result in infection with bacteria from the patient’s groin/perineal area.
Understanding why hand hygiene helps reduce the risk of infection

Our hands always have germs present on them. It is impossible to physically remove or destroy all of them, although we can significantly reduce their numbers. Germs present on hands are predominantly bacteria, but occasionally may include spores (from fungi or bacteria) or viruses (e.g. the common cold) if hands are in contact with a contaminated surface, body area or fluid. The bacteria on our hands can be divided into two categories: resident and transient bacteria. Resident bacteria are those that live on our hands permanently. The resident bacterial flora (also known as microbiota) play a protective role by helping to prevent non-resident bacteria from establishing permanently on the skin.

Transient bacteria are those most frequently associated with the spread of infection in healthcare and also the home or social environments. These transient bacteria become loosely attached to the outer skin layers of the hands following physical contact with people, equipment or environmental surfaces and, if not removed or destroyed, they will easily be transferred from one place or person to another, as highlighted in Table 22.1.

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**WHAT’S THE EVIDENCE?**


In this seminal paper, Casewell and Phillips (1977) demonstrated a link between nursing care activities, the contamination of hands and the colonisation or infection of patients. The study focused on obtaining evidence of how one specific bacteria (*Klebsiella spp*) was being transmitted between patients and staff.

**Table 22.1** Contamination of nurses’ hands by different *Klebsiella spp* following care procedures

<table>
<thead>
<tr>
<th>Patient</th>
<th>Care activity</th>
<th><em>Klebsiella</em> types colonising patients</th>
<th><em>Klebsiella</em> types recovered from nurses’ hands</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Lifting patient</td>
<td>21, 47, 10</td>
<td>47, 10</td>
</tr>
<tr>
<td>B</td>
<td>Taking blood pressure and pulse</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>Physiotherapy</td>
<td>21</td>
<td>21, 28</td>
</tr>
<tr>
<td></td>
<td>Washing patient</td>
<td>21</td>
<td>24, 28</td>
</tr>
<tr>
<td></td>
<td>Taking oral temperature</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>General nursing</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td>C</td>
<td>Radial pulse</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>Touching shoulder</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>Touching groin</td>
<td>21</td>
<td>21</td>
</tr>
<tr>
<td>D</td>
<td>Washing patient</td>
<td>21, 45, 9</td>
<td>21, 45</td>
</tr>
<tr>
<td>E</td>
<td>General nursing</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td><strong>Extubation</strong></td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>Touching groin</td>
<td>15</td>
<td>15, 19</td>
</tr>
<tr>
<td>F</td>
<td>Touching hand</td>
<td>55, 47</td>
<td>55</td>
</tr>
</tbody>
</table>

*Source: Adapted from Casewell and Phillips (1977)*
The results showed that Klebsiella spp were recovered from the hands of nurses and body sites of patients. As is shown in Table 22.1, specific nursing procedures were identified that resulted in the contamination of nurses’ hands following contact with patients. The introduction of routine hand-washing with chlorhexidine by staff resulted in sustained reductions over time of colonisation of patients with Klebsiella spp.

- Reflect on the essential care activities that you undertake, such as those illustrated in Table 22.1. Do you ever think about how contaminated your hands might be, even when there are no visible signs of dirt?
- When you are next on placement, for a four-hour period keep a record of how often you wash your hands and the reasons for doing so. Reflect on the results: do you think you washed your hands too infrequently, too often, or was your practice in line with local policies?
- Now read the rest of the chapter and see if you are correct!

How and when to perform hand hygiene

In this chapter, we are considering what is known as ‘social hand-washing’ with non-medicated soap. There is another form, which is known as surgical hand-washing (or scrubbing). This has not been included because social hand-washing is suitable for nurses in most situations, apart from the preparation of hands for invasive procedures undertaken with strict aseptic techniques, such as in operating theatres.

The aim of hand-washing is to remove transient germs (bacteria, viruses, fungi and spores) from the surface of the hands using mechanical friction as a result of the application, rubbing and removal of soap and water during the process of washing. Hand drying using paper towels also physically removes germs from the hands. This contrasts with the application of hand sanitisers, which chemically destroy or inactivate bacteria present on the surface of the hands. Currently, the use of hand sanitisers is not promoted for patients suspected or known to have viral gastroenteritis (e.g. norovirus) or an infection due to Clostridium difficile. Hand-washing with soap and water is recommended in these circumstances.

Hand-washing

A technique adapted from research evidence provides a framework for hand-washing. This technique (see Figure 22.1) enables good coverage of the hands with soap or hand sanitiser and a systematic technique to support effective hand hygiene.

As a further aid to effective hand-washing, Taylor (1978) identified areas of the hands which were most frequently missed during hand-washing. Her findings are still used today to help teach and improve hand-washing techniques (see Figure 22.2).

Guidelines

There are various guidelines and expectations relating to practice that you must be aware of when cleaning your hands. These include:

- infection control policies
- local programmes such as hand hygiene campaigns/strap lines.
To ensure you always wash your hands effectively, follow the steps outlined in Clinical Skill 22.1.

![Ayliffe's six-step hand hygiene technique](image)

**Figure 22.1** Ayliffe’s six-step hand hygiene technique

*Source: Adapted from Ayliffe et al. (1978)*

Sometimes missed

Frequently missed

![Areas of the hands most frequently missed during hand-washing](image)

**Figure 22.2** Areas of the hands most frequently missed during hand-washing
ACTIVITY 22.1: REFLECTION

Reflect on the effectiveness of the technique you use to wash your hands:

- Do you follow the six steps outlined by Ayliffe et al. (1978) (Figure 22.1)?
- Do you miss the areas identified by Taylor (1978) (Figure 22.2)?

Use of hand sanitisers

The use of alcohol-based hand rubs and foams has increased dramatically over the past ten years. They are far more effective than soap and water in reducing the numbers of transient bacteria on hands. The application and distribution of hand sanitisers can follow a similar technique as for the application of soap and water (Figure 22.1); however, hand sanitisers must be left to dry naturally on the hands and not washed off, because the action of evaporation is important.

Hand sanitisers are a convenient and highly effective method for the decontamination of hands, as you are able to apply it, rub it into your hands and move to the next patient or physical location without stopping to do hand-washing. This was a key factor in the promotion and adoption of hand sanitisers in healthcare facilities in the UK, in addition to the fact that they also offer a convenient portable alternative in environments where soap and water may not be readily available, such as in remote locations or ambulances.

It is important to remember that hand sanitisers are not suitable to use in all circumstances and that the physical contamination of hands by dirt or body fluids must be managed by first removing this by hand-washing (or use of hand wipes in community settings). This is most important as organic material is known to inactivate alcohol. Hand sanitisers can be applied once the hands are clean to ensure they are safe for the next patient. To ensure you always do this effectively, follow the steps outlined in Clinical Skill 22.2.

When to perform hand hygiene

When to perform hand hygiene remains a matter of much debate. Central to this are the risks that touching different potential sources of contaminants, either patient or environmental, bring. Long lists of indications about when to wash your hands have been replaced with an emphasis on assessing the potential risk. The concept of ‘reference points’ for hand hygiene linked to space/time (Sax et al., 2007) led to the development of the World Health Organization (WHO, 2009) framework of the ‘5 moments for hand hygiene’, which has been widely but not exclusively adopted as a contemporary standard to apply:

1. Before touching a patient
2. Before clean/aseptic procedure
3. After body fluid exposure
4. After touching a patient
5. After touching patient surroundings.

One of the advantages of the ‘5 moments’ is that it takes a patient-centred approach. It can also be adapted to situations outside of hospitals where the focus remains on the patient, be it a patient sitting in a chair or ambulance, or a baby in a cot.
STEP-BY-STEP CLINICAL SKILL 22.1:
HAND-WASHING

☑ Before you start
Consider, is it beneficial to inform the patient that you intend to wash your hands? The patient and their relatives or carers may be reassured that you are taking steps to protect them from the transmission of infection via hands.

☑ Essential equipment
Running tepid water, soap, hand towels (preferably disposable, but patients may offer you a clean hand towel in community settings).

☑ Field-specific considerations
Washing your hands is an essential skill within the care of patients from all fields. The principles outlined in this skill will not vary depending on the field.

☑ Care-setting considerations
Facilities for hand-washing will vary considerably between care settings and patients’ homes.
   - Always be prepared for a lack of running water, soap and clean hand towels.
   - In community settings, carry your own supply of hand towels or hand wipes to support hand-washing.
   - Always carry hand sanitiser for situations when this is appropriate.
   - Keep hand sanitisers out of the reach of children or those with impaired mental capacity. Refer to Clinical Skill 22.2, below.

☑ What to watch out for and action to take
Do not apply soap directly to dry hands as this can result in sore hands and poor coverage of soap.
   - Staff with broken skin should cover it with a plaster. Staff unable to perform hand hygiene (because of sore hands) should not be working in clinical environments due to the risks to patients and themselves.
   - Staff suffering from dermatitis and/or sore hands should seek advice from their local occupational health department.
   - Always use the foot pedal of the bin (if available) – never dispose of hand towels by lifting the lid using your fingers because this will result in recontamination of your hands.
Identify the need for hand hygiene to be performed
Undertake an assessment to ascertain whether there is a need for hand-washing to take place

Turn on taps and select a comfortable temperature
Water that is too hot or too cold can impact on compliance with the hand-washing technique

Wet hands
Prepare hands to receive soap and facilitate an even covering of soap for the next stage

Apply soap
Apply one dose of liquid soap to cupped hands.
If bar soap is the only option available, then this may be used, depending on its quality. Community staff may carry small amounts of soap with them in containers

Rub hands together and evenly distribute soap coverage following steps set out in Figure 22.1
Rubbing hands together produces mechanical friction. This results in all areas of the hands coming into contact with soap and transient micro-organisms being lifted from the outer layers of the skin into the soap solution on the hands

Rinse hands
To remove the transient micro-organisms present in the soap solution from the hands

Dry hands
Dry the skin of the hands and remove any remaining transient organisms as a result of mechanical friction. Ensure all areas of the hands are dry

Dispose of hand towels
Dispose of used materials correctly without re-contaminating your hands

It should be noted, however, that even though this approach offers a theoretical improvement, its application in practice for complex interactions, such as the delivery of a baby, remains controversial, and 100 per cent compliance is doubtful and unachievable in all situations.

Monitoring hand hygiene compliance
As a nursing student, it is almost certain that you will have your hand hygiene compliance monitored at some point, most likely in an inpatient setting. Hand hygiene is a priority area for the improvement and monitoring of infection prevention as it is thought to reflect overall standards of infection prevention.

Audits of hand hygiene vary in their methodology, but direct observation remains the current standard. This method, however, is not practical in settings where staff work alone – for example in GP surgeries, practice nurse or midwife clinics, or community settings such as patients’ own homes. Direct observation is also fraught with practical difficulties and bias, particularly if staff are aware they are being observed.

Healthcare organisations frequently report high compliance scores for hand hygiene, but audits undertaken by infection prevention teams often show much lower results.
STEP-BY-STEP CLINICAL SKILL 22.2
USING HAND SANITISER

✔️ Before you start
Consider whether it is appropriate to inform the patient that you are going to use sanitiser on your hands so the patient and their relatives or carers are reassured that you are taking steps to protect them from the transmission of infection via hands.

✔️ Essential equipment
Hand sanitiser (this may be carried personally, available at the point of care or wall-mounted).

✔️ Field-specific considerations
Ensuring your hands are free from transient micro-organisms is an essential skill within the care of patients from all fields. The steps outlined in this skill will not vary depending on the field.

✔️ Care-setting considerations
Facilities for hand hygiene will vary considerably between care settings and patients’ homes.
   - Always be prepared for a lack of running water, soap and clean hand towels. In community settings, carry your own supply of hand towels or hand wipes to support hand hygiene. Always carry hand sanitiser for situations in which this is appropriate.
   - Keep hand sanitisers out of the reach of children or those with impaired mental capacity. The ingestion of alcohol hand sanitisers that has resulted in the death of patients has been recorded (HM Coroner, 2017).

✔️ What to watch out for and action to take
Any cuts, open wounds or dry skin on hands will sting following the application of hand sanitiser. Staff with broken skin should cover it with a plaster. Staff unable to perform hand hygiene (because of sore hands) should not be working in clinical environments due to the risks to patients and themselves. Staff suffering from dermatitis and/or sore hands should seek advice from their local occupational health department.
Identify the requirement for hand hygiene to be performed
Assess whether hand hygiene needs to take place.

- Confident that the hand sanitiser will be effective to decontaminate hands.
- Remember, if a patient has diarrhoea or a gastrointestinal infection such as C. difficile, hand sanitiser may not be effective. Wash hands first, if possible, then apply hand sanitiser if needed. Visibly soiled hands should be cleaned with soap and water, if available, or a hand wipe prior to application of sanitiser.
- Able to access hand sanitiser at the point and time of need

Apply the hand sanitiser to all surfaces of the hands and rub hands together to support evaporation following steps set out in Figure 22.1
All surfaces of the hands come into contact with the hand sanitiser to ensure transient micro-organisms are destroyed.

- This six-step technique is more effective than other less standardised techniques (Reilly et al., 2016)

Allow the hand sanitiser sufficient time to dry (evaporate) prior to next patient contact
The hand sanitiser needs adequate time to be effective and destroy micro-organisms on hands

Source: Loveday et al. (2013); NICE (2012)

Improving hand hygiene
A number of barriers to hand hygiene have been recognised, which include:

- A perception that other patient needs take priority
- Time pressures
- The impact of hand hygiene products on staff skin
- Poor role models promoting hand hygiene
- Inadequate staffing
- Scepticism about the benefits of hand hygiene
- Inappropriate glove use.

ACTIVITY 22.2: REFLECTION

- How would you feel if you observed another member of staff not complying with hand hygiene or appropriate use of gloves?
- Do you have a responsibility to raise this issue on every occasion or just those that you feel are particularly important?
- How would your actions impact on the risk to patients?
Patient and public knowledge

Patient empowerment and knowledge of hand hygiene have increased tremendously over the past ten years, as Liam’s voice at the start of the chapter showed us. This has arisen due to greater public awareness of infection prevention, media messages and education relating to hand hygiene. For patients, the focus should be both on supporting them to clean their hands so as to prevent endogenous infection, for instance wound infections caused by removing or picking at dressings, and on encouraging good hygiene practices such as hand-washing before meals and after using the toilet. Within both hospital and home or community settings, practical obstacles exist in encouraging good patient hand hygiene.

"I lead by example by regularly washing my hands; I give them advice and use gentle persuasion."

Julie Davis, LD nursing student

CASE STUDY 22.1: GRAZIELLA

Being admitted to hospital can be a very frightening time for both patients and their families. We put our trust and faith in the staff caring for us. Therefore, it is important to make patients feel safe and secure and feel that everything possible is being done.

Of course, no one wants to be in hospital; patients are there to be treated for a variety of conditions and in most cases the outcome will be fine. However, this is not always the case – complications may occur, prolonging an admission, which can make patients more susceptible to a whole host of problems. Infections are the major problem. I know this only too well because someone very dear to me sadly lost her life due to one of these infections.

My grandmother went into hospital to be treated for a urine infection, something that should have been easy enough to treat; however, antibiotics, a less than clean environment and staff who were not adhering to IPC measures were to blame for my grandmother contracting _C. difficile_ infection. Within a period of ten weeks, my grandmother fought hard to overcome many problems but she could not fight _C. difficile_. She died as a result of the infection.

Watching her suffer in the way she did is something I will never forget. Of course, we all have to die, and at the age of 93 my grandmother had lived her life, but no one should have to suffer in the way she did.

Could my grandmother’s infection have been avoided? I think so. Thirty patients became infected with _C. difficile_ during the period in which my grandmother was in hospital. Staff did not know how to control it but something as simple as washing their hands could have prevented so many patients becoming infected.

Hand hygiene is one of the most basic ways of helping to keep patients safe. Something that only takes a couple of minutes of your time can be the key in ensuring that your patient is being protected from infection. That is the least any patient can expect from staff looking after them.

- Next time you are busy caring for patients, time exactly how long it takes you to perform hand hygiene.
- Performing effective hand hygiene has the potential to save a patient’s life. Are you really so busy that this is a risk worth taking?
STANDARD PRECAUTIONS AND GLOVES

As mentioned previously, gloves are a core element of what is currently known as ‘standard precautions’. Gloves provide a physical barrier between the user (you) and the substance originating from the patient (e.g. excreta, bodily fluids or chemicals used in care practices that they may come into contact with). The potential harm from biological risks relates to micro-organisms that may be present (e.g. blood-borne viruses or bacteria) which, when transmitted, are capable of causing infection in either other patients or staff.

We will differentiate between the two main glove types by using the term ‘examination gloves’ to describe disposable single-use gloves used for routine clinical practice, and ‘protective gloves’ to refer to single-use gloves that are used to protect the wearer from exposure to harmful chemicals or drugs. The latter are required to meet additional testing standards to those of standard ‘examination’ gloves, to prevent the risk of permeation of chemicals. A common example is ‘nitrile’ gloves. We will not be discussing the use of sterile gloves for surgical procedures.

It is important to understand the different types of gloves available and the differences between them. On your placements, you will come across many different nursing activities, and gloves may vary considerably between settings and patient groups.

Ideally, different glove types should be provided, to enable nurses to choose which to use based on a risk assessment of the requirements of the activity being undertaken. Within healthcare organisations, different staff groups will have different needs – for example, porters will need heavy-duty protective gloves when collecting or transporting waste; theatre staff require surgical gloves; nurses administering chemotherapy agents may require ‘protective’ gloves because of their exposure to hazardous drugs. In some settings, such as mental health, gloves may be very infrequently worn and contact with chemicals will be limited. In other nursing fields, the use of gloves may be more common.

Being clear on employer and employee responsibilities

Health and safety legislation requires both employers and employees to undertake risk assessments of potential risks in the workplace, and to eliminate these where possible. In the case of healthcare, it is not possible to remove these risks, such as caring for a patient and the micro-organisms they may be carrying, or a patient’s need for chemotherapy drugs. Where risks cannot be eliminated or managed by engineering controls (e.g. safety needles), measures should be put in place to reduce the impact of those risks that remain. Gloves are, therefore, when used correctly, one example of a control measure used to protect both staff and patients from risk.

Glove types and sensitivity to their components

Single-use disposable gloves may be manufactured using different components – natural rubber latex, vinyl or synthetic rubbers such as nitrile and neoprene are all frequently used. Gloves may also be powdered or unpowdered and sterile or unsterile.

Sensitivity and reactions to glove components are important issues for those working in healthcare, particularly nurses. The most well-known example is natural rubber latex, but many people do not realise that sensitivity can also develop to other, non-latex gloves. Sensitivity to chemicals used in the manufacturing process of gloves is another example. Natural rubber latex proteins are considered hazardous to health (HSE, 2012) and therefore need to be managed under the Control of Substances Hazardous to Health Regulations (COSHH) guidelines (2002, updated 2013). Currently, the use of latex gloves is not banned but many organisations have moved away from, or are in the process of moving away from, latex.
When to use gloves

As with hands, gloves can act as a vehicle for the transmission of micro-organisms and therefore it is important to change gloves if moving from one patient to another, or when undertaking multiple care tasks on the same patients. Gloves are not a substitute for hand hygiene!

The use of gloves has increased tremendously over the past 30 years and indiscriminate use is now recognised as a significant risk to both patients and nurses (Loveday et al., 2013) which undermines hand hygiene (Fuller et al., 2011).

Indications for glove use are:

- when anticipating contact with blood or body fluids
- when anticipating contact with non-intact skin or mucous membranes
- when a risk of contact with hazardous chemicals or drugs exists.

Reasons for the unprecedented rise in glove use are not really known; however, in discussion with students and registered nurses, it appears that some nurses feel they must wear gloves with all patients regardless of any established risk. This means that nurses are often observed wearing gloves for routine bed-making, feeding patients or even bathing them – when no contact with specific risks, as highlighted previously, exists. It seems that a perception of a ‘dirty task’ overrides the rationale and evidence base that hand hygiene is sufficient for all other ‘non-identified risk’ situations.

While it is our duty to ensure that our practice is up to date, at times you will experience working with practice educators who have been trained differently to us and as such may well use gloves in different circumstances to us or will not wear gloves. If this occurs and you are unsure what to do read the local policies to guide your practice or simply ask your practice educator why they are/aren’t wearing gloves. At all times ensure that you are working within best practice guidelines.

Alice Rowe, NQ RNMH

Which gloves and when?

Table 22.2 outlines some issues and considerations frequently associated with glove selection. Note: some healthcare organisations have made the decision to only provide one type of examination glove.

<table>
<thead>
<tr>
<th>Issue</th>
<th>Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>The activity to be performed</td>
<td>Be clear on why gloves are needed and select the appropriate glove type</td>
</tr>
<tr>
<td>Anticipated contact and compatibility with chemicals and chemotherapeutic agents (e.g. cytotoxic drugs or disinfectant solutions or wipes)</td>
<td>Are gloves required to protect the skin of staff? These should comply with the relevant EU standards to meet the Personal Protective Equipment Directive</td>
</tr>
<tr>
<td>Latex or other sensitivity</td>
<td>Alternative gloves should be available</td>
</tr>
<tr>
<td>Glove size required</td>
<td>Gloves should always be available in small, medium and large sizes</td>
</tr>
<tr>
<td>Local policies for creating a latex-free environment</td>
<td>Refer to local infection prevention and occupational health policies for further guidance</td>
</tr>
</tbody>
</table>
STEP-BY-STEP CLINICAL SKILL 22.3:
WHEN TO REMOVE YOUR GLOVES AND WHY

Before you start
Ensure you have undertaken an assessment to determine if gloves can be retained or should be changed.

Essential equipment
Hand hygiene equipment (soap and water or hand sanitiser).

Field-specific considerations
Mental health - gloves are infrequently used in mental health settings but may be required at times. Indications may include caring for incontinent patients, phlebotomy or dressing wounds. Differences may also be present in practice depending on whether you are working in an inpatient or community setting.

Learning disability - depending on the patients, glove-use need will vary. For those with physical needs, the indications for glove use are the same as for adult general nursing.

Child - indications for glove use in children’s settings are the same as for adults, and this includes neonates. Newborn babies may look ‘clean’ but the same principles and risks apply.

CHANGING GLOVES

1 When there is an indication for hand hygiene
Wearing gloves may afford you some protection but the patient remains vulnerable if you do not consider the risk of transfer of micro-organisms via gloves in the same way as hands
Whenever an indication for hand hygiene occurs, and gloves are being worn, these should be removed, hand hygiene performed and then clean gloves applied. This is particularly relevant when multiple care activities are undertaken on the same patient

2 When glove integrity is breached or suspected
Gloves are not a complete barrier and defects may be present unknown to the wearer. Gloves reduce but do not eliminate risks

3 When
a. the actual or potential contact with blood, body fluids or mucous membranes is finished
b. contact with hazardous drugs or chemicals has finished
c. contact with a contaminated body site or device (e.g. infected wound, urinary catheter bag) has finished

Once the activity is complete, gloves should be removed, disposed of and hand hygiene performed. This removes potential contamination from the hands of the nurse, protecting both them and the next patient

Source: Loveday et al. (2013); NICE (2012); RCN (2012)
for use by all staff (e.g. nitrile) as a result of local risk assessment (for example, the need to deliver chemotherapy in multiple patient settings as opposed to dedicated chemotherapy suites).

Knowing when to use gloves appropriately, however, is only half the story. It is as important to understand when gloves need to be removed and either changed or disposed of. Clinical Skill 22.3 identifies when removal of gloves is required and explains why.

**Glove use and hand hygiene compliance**

As we have already discussed, gloves are not a substitute for hand hygiene and hand hygiene must be performed after gloves are removed, as the act of removing gloves can contaminate hands. Additionally, gloves may not provide a complete barrier to micro-organisms or chemicals (due to the presence of small breaches in glove integrity) and therefore hands may become contaminated during care activities. Most hand hygiene audits do not routinely collect data on glove use as a factor in determining hand hygiene compliance, and therefore this represents a gap in both our understanding of the extent of the issue and the available data on which to assess improvements in hand hygiene practice.

**ACTIVITY 22.3: REFLECTION**

There are many potential occasions when gloves may be required:

- Compile a list of occasions recently when you have determined it is necessary to wear gloves.
- Which of these were to protect the patient, and which to protect you?
- Now reflect on whether you wore gloves when perhaps they might not have been required - under what circumstances did this occur, and were you influenced by the behaviour of others to act in a similar way?

**WHAT’S THE EVIDENCE?**

Loveday et al. (2013) recommend that hand hygiene should be performed before and after donning gloves:

- Do you comply with this and have you seen other staff do this?
- What are the risks to you and the patient if you do not perform hand hygiene as recommended with gloves?
- Now read the guidelines and consider how you can best apply the recommendations to your practice.

**ACTIVITY 22.4: REFLECTION**

The promotion of patient hand hygiene and a culture of encouraging patients to challenge staff to perform hand hygiene remain core elements of many care organisations’ local hand hygiene programmes. This is an extract from an article in the *Daily Telegraph*, from 18 July 2007:
Patients in hospital should challenge doctors and nurses to wash their hands before consultations, the Chief Medical Officer said yesterday. Good hand hygiene is the key to reducing hospital acquired infections such as MRSA and NHS staff are often too complacent, Sir Liam Donaldson said in his annual report. Patients on wards should be issued with their own alcohol hand gel and should ask doctors and nurses to use it before examinations and procedures, he said.

Estimates suggest that one patient is infected every two minutes in hospitals and one dies every two hours from healthcare-associated infections. Sir Liam said that even in the best hospitals compliance with hand hygiene rules is rarely above 60 per cent - yet up to three quarters of all patients do not feel comfortable challenging staff.

- What would your reaction be if you were challenged by a patient to wash your hands?
- Consider different patient groups in different settings - which groups of patients do you think might be at a disadvantage in challenging staff?
- How can this be overcome?

Skin health

In addition to placing patients at risk of infection, inappropriate (excessive) use of gloves can have a detrimental effect on the health of nurses’ skin. Nurses are vulnerable to developing dermatitis due to a number of factors, including:

- exposure to a ‘cocktail’ of chemicals such as natural rubber latex or accelerators, disinfectant or detergent wipes, soap and alcohol hand sanitisers
- ‘wet work’ – where hands are frequently exposed to water, such as in bathing, showering or washing up
- frequent hand-washing or use of hand sanitisers.

Prolonged or frequent use of gloves can cause the skin to become over-hydrated and can also be a risk factor in the development of dermatitis. Combined, these potential factors place staff at risk of dermatitis on their hands (RCN, 2012), as shown in Figure 22.3.

![Dermatitis and reddening of skin](https://www.hse.gov.uk/skin/imagelibrary.htm)

**Figure 22.3 Dermatitis and reddening of skin**

Source: www.hse.gov.uk/skin/imagelibrary.htm. Copyright of HSE, contains public sector information published by the Health and Safety Executive and licensed under the Open Government Licence v1.0
CASE STUDY 22.2: IDRIS

Idris has just returned from placement on a neonatal care unit. He is about to start working on a children's oncology ward but the alcohol hand sanitiser is really stinging, so he has stopped using it. He can't wash his hands properly as his hands are so sore.

- What actions should Idris take and why?
- Do you think Idris should continue caring for patients if he can't perform hand hygiene?

What is dermatitis and why is it important?

Simply put, dermatitis is inflammation of the skin on the hands, a type of eczema. It may occur naturally or as a result of contact with substances which cause a reaction, known as ‘contact dermatitis’. Different terminology is used to describe different types of dermatitis.

Not all dermatitis occurs as a result of work, and some factors associated with life outside of work, such as having children under the age of 5 or washing dishes by hand, can contribute to the risk of developing dermatitis.

Roles and responsibilities relating to occupational dermatitis

Both employers and employees have responsibilities when it comes to reporting and managing occupational dermatitis. Employers have a responsibility under health and safety laws to protect staff from illness caused as a result of work, and this includes contact dermatitis. Healthcare workers (that’s you) have a legal responsibility to cooperate with employers regarding health and safety matters. This includes following your organisation’s policies and procedures (for example, those around standard precautions, skincare and the appropriate use of PPE), and reporting any incidences of hand dermatitis in a timely manner to your manager and occupational health department.

Always follow the glove good practice guidelines:

- Gloves are single-use items – which means wear for one task then take them off!
- Hand hygiene should be performed prior to putting gloves on.
- Hands should never be washed with gloves on.
- Wearing gloves is not a substitute for hand hygiene.
- Gloves can transmit micro-organisms capable of causing infection in the same way hands can.
- Gloves must only be used if necessary, following a risk assessment for both patient and nurse.
- Hand hygiene should always be performed after the removal of gloves.
- Gloves should not be routinely used for bed-making.
- Only powder-free gloves should be used.
- Always wear the correct glove size for you and report a lack of suitable glove types or sizes to the person in charge.
- Be aware of the signs of dermatitis and report any that occur.
- Never wear gloves ‘just in case’.

YOUR RESPONSIBILITIES AS A WASTE PRODUCER

The production of waste as a result of healthcare activities is inevitable. The waste items we will cover in this chapter are healthcare waste and used linen.
While the management of waste may not strike you as a nursing responsibility, nursing accounts for approximately 70 per cent of the UK healthcare workforce and the production of waste is an unavoidable consequence of the role and the care tasks undertaken. The International Council of Nurses (ICN) states that ‘nurses must understand the hazardous consequences of improper waste handling, the “cradle to grave” waste cycle and methods that mitigate the negative impact of waste on the environment’ (ICN, 2009).

The principles of good waste management – the waste hierarchy

The waste hierarchy describes options that should be considered as part of an overall strategy for waste management, so it is not just applicable to healthcare organisations. The principle of the hierarchy is that waste production should be avoided wherever possible. The overall aim, as identified in Figure 22.4, is therefore to reduce the actual amount of waste that is generated for disposal, regardless of the end point, such as landfill or incineration.

![Figure 22.4 The waste hierarchy](image)

Classification of healthcare waste

Many different types of waste are produced as a result of healthcare. Management of waste is governed by a multitude of regulations arising from both UK and EU requirements. Different types of waste are produced, some harmful and some not. Types of waste include laboratory waste (including chemicals or growth media); human anatomical waste (e.g. limbs, used medical devices such as syringes, nebulisers); infectious waste; and household waste.
The RCN (2014) describes how the main types of waste may be classified simply into the following categories:

- clinical waste – most, but not all, of this is classified as hazardous waste. Sub-categories of clinical waste exist, such as infectious waste, medicinal waste and sharps. In healthcare, the term includes:
  - waste which contains viable micro-organisms or their toxins which can cause disease in humans or other living organisms
  - waste which contains or is contaminated with a medicine that contains a biologically active pharmaceutical agent, or is a ‘sharp’, or a body fluid or other biological material (including human tissue) containing or contaminated with a dangerous substance
- offensive waste (or hygiene waste) – this is best described as waste that is non-infectious but which may be deemed to be ‘offensive’ due to its contents (e.g. sanitary waste, soiled incontinence pads, etc.) and possibly smell
- municipal (household) waste – waste similar to that produced in your own home, e.g. waste packaging, dead flowers, etc.

The waste producer

The waste producer is the person who generates the waste (you), whether this is a sharp after you give an injection or packaging that you are disposing of. The producer of the waste is responsible for:

- assessing what type of waste they have generated
- placing the waste in the correct, colour-coded container.

Waste in healthcare is often ‘over-classified’ as clinical or infectious (RCN, 2011). The consequences of this is a lack of compliance with waste legislation and a huge burden on healthcare costs, as it is currently more expensive to dispose of clinical waste than offensive or household waste. This situation has arisen partly due to efforts to ensure waste is safe ‘just in case’, hence over-classification, or because offensive waste bags and bins have not been made readily available to staff to support their use. In reality, the vast amount of waste produced in healthcare is not infectious.

What type of waste do you produce?

It is important that the waste produced is assessed at the time of its production, separated correctly and placed in the right container. Nurses often note confusion about the classification of infectious and offensive waste. Table 22.3 offers advice on this.

Colour-coded segregation exists to help identify which type of waste is present in a container or bag. In this way, it is possible to identify sharps, medicinal waste, waste containing cytotoxic chemicals or offensive waste, which is important for the purposes of transporting waste to its final destination. This colour scheme can vary from country to country in the UK.

The main categories of bagged waste you are most likely to come across are illustrated in Figure 22.5. Always check your local policies for more detail.
Table 22.3  Classification of common waste according to ‘infectious properties’

<table>
<thead>
<tr>
<th>Waste contains</th>
<th>Proposed general classification</th>
<th>Examples of waste</th>
<th>Exceptions to this rule</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine, faeces, vomit and sputum</td>
<td>Offensive (where risk assessment had indicated is present, and no other risk of infection exists)</td>
<td>Urine bags, incontinence pads, single-use bowls, nappies, PPE (gloves, aprons, etc.)</td>
<td>Gastrointestinal and other infections that are readily transmissible in the community setting (for example, verocytotoxin-producing <em>Escherichia coli</em> (VTEC), campylopacter, norovirus, salmonella, chicken pox/shingles)(^1)</td>
</tr>
</tbody>
</table>
| Blood, pus and wound exudates  | Infectious unless assessment indicates no infection present. If no infection, and no other risk of infection, then offensive | Dressings from wounds, wound drains, delivery packs | Blood transfusion items
Dressings contaminated with blood/wound exudates assessed not to be infectious
Maternity sanitary waste where screening or knowledge has confirmed that no infection is present and no other risk of infection exists

Note: \(^1\)Potential hazards from the use of cytotoxic and cytostatic medicines may also be relevant in some instances and with some drugs. This would also prevent the waste being considered offensive.

Source: RCN (2014)

Figure 22.5  Categories of bagged waste

ACTIVITY 22.5: REFLECTION

Find and read your local waste policy:

- What different types and colours of bags and bins does it mention?
- Have you seen all of these when on placement?
- Do you think that waste could be segregated more effectively and recycling improved?

Waste production in the community

Waste disposal as a result of nursing patients in their own homes poses some unique challenges and considerations.
Risk assessment of waste in community settings is the same as in hospital settings and the waste classification applies. The management of waste (storage, collection, etc.), however, is managed differently and will vary from one geographical area to another. This sometimes means nurses have to carry healthcare waste in their cars, or even take it with them on ferries in more remote communities. Use of the patient’s waste bins is permissible for some waste produced, such as ‘soft non-infectious waste’, where the amount is comparable to what a patient would normally produce and has been deemed non-infectious (RCN, 2014). Infectious and sharps waste must be managed separately and must not be disposed of via the patient’s home.

“Waste management is just as important in the community as in hospital but the set-up and polices are just different. You would need to read and make yourself familiar with the policies when starting a new job.

Sarah Parkes, LD nursing student”

**ACTIVITY 22.6: REFLECTION**

- Do you think community nurses should carry waste in their cars?
- Why do you think community nurses may have been asked to do this?
- Can you identify any improvements as to how healthcare waste is managed in the community?

**Top tips for good waste management in all settings**

If you follow these principles, it is possible to manage healthcare waste safely and effectively in all settings:

- Risk-assess all waste you produce, and segregate accordingly – do not be tempted to put everything in one bag regardless.
- In hospital and GP surgery settings, waste must be stored securely and separately to other items such as used linen.
- Waste bags should be removed, sealed and transferred to a storage facility when two thirds full – do not allow bags to overfill, otherwise they cannot be sealed safely, thus placing others at risk.
- Ensure different types of waste are segregated appropriately in the appropriate storage area.
- Label waste so the area where it originates can be identified. Tags should be available to use locally. Do not ‘borrow’ other areas’ labels, as traceability is important should an issue be identified.
- Waste must not be allowed to accumulate in public areas – always use dedicated storage areas or move directly to large rigid containers.
- When handling waste, aprons and gloves should be worn; they should be removed immediately after the task is complete, followed by hand hygiene.
- Report issues with waste collection or storage immediately.
MANAGEMENT OF USED LINEN

The management of used linen includes handling and disposal and is mostly associated with hospital settings. Used linen is rarely implicated in outbreaks of infection, but it does pose risks to both other patients and staff on occasion.

Some units, such as neonatal or long-term care units, may have their own washing machines to enable them to wash patients’ items locally. While these may seem convenient, they do pose risks, and outbreaks of infection have occurred that have been associated with washing machine use. Most used linen is processed via a central (usually off-site) laundry which has to comply with national standards for temperature controls and processes.

How to handle linen appropriately

When handling used linen, the following applies:

- Always wear a plastic apron for handling used linen to prevent contamination of your uniform.
- Do not routinely wear gloves for bed-making unless the patient is in isolation or being ‘barrier nursed’ with a risk of infection. Always perform hand hygiene after bed-making.
- Handle used linen carefully to avoid shaking it and disturbing skin cells and bacteria that will be present.
- Do not carry used linen through a ward or department. Always place it immediately into a linen skip and transfer to the storage area as soon as possible.
- Do not mix clean and used linen.

Segregation of used linen

As with waste, different colour coding applies to linen categories. The two main category codes are red and white bags:

- Red linen bags denote an infection risk and involve placing the linen in a red inner ‘alginate’ (water-soluble) bag before it is placed in a thicker plastic or cotton outer bag. This is because of the need to prevent laundry workers coming into contact with the linen (it is placed directly into the machine in the ‘alginate’ bag, which then dissolves). Linen should be segregated into red bags if a patient is being isolated with an infection or infectious condition, or if the linen is soiled with blood or body fluids.
- White linen bags are the most common type, used for uncontaminated linen.

Used linen bags should be sealed before they are overfull and stored appropriately at local level in a dedicated area.

CONCLUSION

This chapter identified the core infection prevention and control practices you need to develop to protect both patients and yourself, wherever you are on placement. Bacteria, viruses and fungi deserve the greatest respect at all times. Our challenge as nurses is to manage their presence in the context of care settings, wherever they may be.

It is fundamentally important for you to have a good understanding of the theoretical and practical aspects of hand hygiene. Think of your hands as tools that are used for work – they need to be clean, safe and cared for to maintain their use.
Hand hygiene on its own as an action to prevent infection will not be successful. It is one of a number of core practices that need to be undertaken together as a ‘package’ to protect both patients and staff in all care settings. In practice, hand hygiene is frequently combined with other practices, such as use of gloves, aprons and masks, plus aseptic technique. Glove use in particular is integral to hand hygiene, as gloves are also in direct contact with patients and can become contaminated through care activities.

Waste management is very costly for organisations and is environmentally important. While the management of waste may not strike you as a nursing responsibility, waste production is an unavoidable consequence of our role and the care tasks undertaken.

**CHAPTER SUMMARY**

- Preventing infection relies on an awareness and knowledge of all the factors that contribute to the complexity of decision-making regarding when to perform hand hygiene or wear gloves.
- Ensure that you are always able to identify both poor practice and positive role models for practice.
- Always reflect on incidents relating to poor infection prevention and control to enable you to improve your practice. It is important to know where to access infection prevention and control-related information and have the confidence to seek support from specialists.
- Ensure you develop and maintain an awareness of research findings, so the care you provide to patients has a sound evidence base.
- While the management of waste may not strike you as a nursing responsibility, the production of waste is an unavoidable consequence of our role and the care tasks undertaken.
- If you follow the principles, it is possible to manage healthcare waste safely and effectively in all settings.

**CRITICAL REFLECTION**

**Holistic care**

Infection prevention and control are important in providing holistic care for a patient. Review the chapter and note down all the instances where the care actions outlined can help you meet a patient’s wider physical, psychological, social, economic and spiritual needs. Think of a variety of different patients across the fields, not just within your own field. You may find it helpful to make a list and refer back to it next time you are in practice, and then write your own reflection after your practice experience.

**GO FURTHER**

**Books**


This guidance represents the practical application of national guidance such as HTM 07-01 and applies it to nursing practice. It has been written in easy-to-read language and reflects the needs of care in a range of settings.


**SAGE journal articles**

Go to https://study.sagepub.com/essentialnursing2e for free online journal articles for this chapter. If you are using the interactive ebook, simply click on the book icon in the margin to go straight to the resource.
Lee, K. (2013) ‘Student and infection prevention and control nurses’ hand hygiene decision making in simulated clinical scenarios: A qualitative research study of hand washing, gel and glove use choices’, *Journal of Infection Prevention*, 14(3): 96–103. This article describes how final-year nursing students and specialist IPC nurses verbalised their hand hygiene decision-making while working through clinical scenarios on a computer, to understand what factors they were taking into account in choosing to use hand-washing or alcohol-based hand-rub/gel or to wear gloves.


**Other journal articles**

Fuller, C., Savage, J., Hayward, A., Cookson, B., Cooper, B. and Stone, S. (2011) ‘The dirty hand in the latex glove’: A study of hand hygiene compliance when gloves are worn’, *Infection Control and Hospital Epidemiology*, 32(12): 1194–9. This article describes observed hand hygiene and glove use across a mix of secondary care wards. The authors describe how gloves were often worn when not indicated, and vice versa.

Reilly, J., Price, L., Lang, S., Robertson, C., Cheater, F., Skinner, K. and Chow, A. (2016) ‘A pragmatic randomized controlled trial of 6-step versus 3-step hand hygiene technique in acute hospital care in the United Kingdom’, *Infection Control and Hospital Epidemiology*, 37: 661–6. This publication provides the first microbiological comparison of two techniques used to apply alcohol-based hand rub (AHBR). The 6-step technique was identified as a superior technique.

**Weblinks**

Go to [https://study.sagepub.com/essentialnursing2e](https://study.sagepub.com/essentialnursing2e) for weblinks related to this chapter. If you are using the interactive ebook, simply click on the book icon in the margin to go straight to the resource.

Health and Safety Executive (n.d.) Choosing the Right Gloves to Protect Skin: A Guide for Employers. Available at: www.hse.gov.uk/skin/employ/gloves.htm. This web page is a useful resource in helping students to gain a broader understanding of issues relating to gloves and guidance for employers. It applies to healthcare as well as other industries, such as hairdressing.

Loveday, H., Wilson, J., Pratt, R., Golsorkhi, M., Tingle, A., Bak, A., et al. (2013) ‘EPIC 3: National evidence-based guidelines for preventing healthcare-associated infections in NHS hospitals in England’, *Journal of Hospital Infection*, 86S1: S1–S70. Available at: www.his.org.uk/files/3113/8693/4808/epic3_National_Evidence-Based_Guidelines_for_Preventing_HCAI_in_NHSE.pdf. These guidelines were commissioned by the Department of Health and form the most up-to-date evidence-based IPC guidance available in the UK. They were developed after a systematic and expert review of all the available scientific evidence. Glove use and standard precautions are included.

National Institute for Health and Clinical Excellence (NICE) (2012) *Clinical Guideline 139, Infection: Prevention and Control of Healthcare-associated Infections in Primary and Community Care*. London: NICE. Available at: http://publications.nice.org.uk/infection-cg139. This guideline applies to all healthcare workers employed in primary and community care settings, including ambulance services, and should ensure safe practice is applied consistently. Much care is also delivered by informal carers and family members, and this guideline is equally applicable to them.

Royal College of Nursing (RCN) (2011) *Freedom of Information Report on Waste Management*. London: RCN. Available at: www.rcn.org.uk/professional-development/publications/pub-004108. This report highlights the significant variation in the classification of waste, with most waste disposed of via the ‘infected’ waste stream, although in reality only a small amount of waste produced by healthcare meets the infectious classification. The report identifies that significant financial savings could be made if waste was better segregated, and encourages better use of the offensive waste stream.

ACE YOUR ASSESSMENT

Review what you have learned by visiting the book’s online resources at: https://study.sagepub.com/essentialnursing2e If using your interactive ebook, just click on the icon in the margin to go straight there.

- Test yourself with multiple-choice and short-answer questions.
- Revise key terms with the interactive flash cards.

REFERENCES


Fuller, C., Savage, J., Hayward, A., Cookson, B., Cooper, B. and Stone, S. (2011) “‘The dirty hand in the latex glove’: A study of hand hygiene compliance when gloves are worn’, Infection Control and Hospital Epidemiology, 32(12): 1194–9.


My name is Jo and I have a 3-year-old son called Sam. Sam had an accident and needs to have his wound dressed by the nurses every few days. This is painful as sometimes the dressing sticks and Sam now recognises when we are going to have it done and can be quite difficult. It’s very upsetting to see him crying, but I know it’s for the best.

The nurse has a wonderful way with Sam and we always make sure he has had some pain medicine before we start. I help her by keeping his hands away from his dressing and together we make sure the wound is clean and securely bandaged afterwards. It takes time of course, but Sam is always so pleased to get his bravery reward when we have finished! Sam is very interested in what happens when his dressing is changed; he has learned the importance of asepsis – perhaps we have a future nurse in the making!

Jo, mother to Sam

Visit https://study.sagepub.com/essentialnursing2e to access a wealth of online resources for this chapter – watch out for the margin icons throughout the chapter. If you are using the interactive eBook, simply click on the margin icon to go straight to the resource.
INTRODUCTION

Asepsis and aseptic technique are two commonly used terms associated with processes to avoid or eliminate contamination of normally sterile body sites or specimens. They are linked to a number of nursing skills, including the collection of patient specimens, and are fundamental to the delivery of quality care because if these skills are undertaken incorrectly the patient may suffer adverse effects such as infection or inaccurate laboratory results. The need to avoid contamination of specimens is crucial, given the importance of these as part of decision-making points for patient treatment. This is particularly relevant to antibiotic prescribing, the main driver of antimicrobial resistance (AMR).

There are numerous reasons why specimens are requested, from assisting in the diagnosis of disease or infection, through monitoring a patient’s recovery from illness, to requirements outside of ‘traditional’ care roles, for example as part of drug and alcohol testing in sport or as part of a police investigation. The role of the nurse extends to many health, social and public arenas in which they can be required to support any such scenarios. However, as an introduction, the context of this chapter is the healthcare environment.

ASEPSIS AND ASEPTIC TECHNIQUE

An aseptic technique process can be used in a variety of scenarios (e.g. invasive device insertion sites), wounds and specimens. Historically, the aseptic technique originated in operating theatres but today is performed in clinical and non-clinical areas (such as patients’ own homes) using sterile equipment combined with other practices to prevent contamination entering the patient’s body.

History

Historically, a number of individuals have contributed to our modern-day interpretation of asepsis and aseptic technique, including Joseph Lister (1827–1912) (use of carbolic acid as an antiseptic in operating theatres), Ignas Semmelweiss (1818–65) (hand-washing), Oliver Wendell Holmes (1809–94) (transmission of puerperal fever) and Louis Pasteur (1822–95) (pasteurisation and the germ theory). The work of these individuals and others has contributed to procedures we still use today in operating theatres to prevent infection in surgical wounds.

Asepsis and aseptic technique today, however, are used in and adapted to situations outside of operating theatres where control of the environment and equipment to reduce contamination is easier to achieve. This brings a number of practical challenges to reducing the risk of contamination of vulnerable body sites. A key challenge is the use of different terminologies/language to describe the aseptic technique, which can result in confusion. However, the biggest challenge is that there is no evidence to inform us how to best undertake an aseptic technique in non-theatre settings, including hospital and community situations. All we can consider is evidence of the impact of non-compliance with some elements of the process (Hopper and Moss, 2010). So, at best, aseptic technique has to be defined by ‘best practice’ or consensus agreement regarding principles with different interpretations, rituals, languages and preferences, all of which have contributed to its evolution over time.

Aseptic versus ‘clean’ technique

The term ‘clean technique’ describes a process or processes to meet the needs of different care settings (originally community settings, but this has expanded to potentially any area) which reduces contamination of vulnerable sites (for example, in the management of chronic leg ulcers which are
already known to be heavily colonised with bacteria). This is, however, a good example of confusion regarding language, process and outcome expectations for both techniques (Flores, 2008; Gillespie and Fenwick, 2009), and in reality the two processes overlap.

How to perform an ‘aseptic technique’

In practice, this issue has become a very contentious one, due to the need to move away from the traditional ‘procedural approach’ towards one that focuses on principles to avoid risks to vulnerable patient sites or devices. As a nurse, it is imperative to understand the fundamental principles, which you can then apply to any situation, rather than just being able to follow a procedure without the ability to customise it to meet patient or environmental demands.

It is important to apply the principles of asepsis to the chain of infection (see Figure 23.1). The chain of infection is a useful way of thinking about how different elements can combine to put patients at risk.

![Chain of infection]

**Figure 23.1** The chain of infection demonstrating where contamination can occur if an aseptic technique is not used

“It is important to understand and apply aseptic techniques as they prevent cross infection or worsening conditions of an existing wound. If we do not use these techniques we will risk further infection of the wounds, prolonged healing time, cross infection for the patient and ourselves and others.”

*Julie Davis, LD nursing student*
An aseptic technique, when performed correctly, will ‘break’ the chain of infection and by doing so protect the patient. However, procedures involving aseptic technique principles are frequently ‘prescribed’ within individual tasks, such as dressing a wound, inserting an intravenous cannula or administering intravenous drugs. There are potential problems with such an approach, as elements of ritual can find their way into practice and differences of opinion, and therefore variations in practice can develop which do not reduce the risk of contamination or produce benefit for the patient. Two examples relevant to current practice and debate are (1) whether it is necessary to wear gloves to perform an aseptic technique; and (2) if so, whether the gloves should be sterile or non-sterile. Another question is whether use of a dressing trolley is preferable to a tray in clinical settings.

The chain of infection

The ‘chain of infection’ (Figure 23.1) describes the necessary series of events that need to take place for infection to occur, regardless of whether this is caused by a virus, bacteria or fungi. For an infection to occur, all links in the chain must be present. As nurses, we prevent this from happening by putting in place procedures or techniques to ‘break’ the chain of infection. Always applying an appropriate aseptic technique is an excellent way of breaking the chain.

**ACTIVITY 23.1: REFLECTION**

Reflect on the aseptic techniques you have observed being carried out by the nurses you have been working with:

- Did they all do it the same way?
- What elements were common to all procedures observed?
- Did the option to use equipment such as gloves or a dressing tray/trolley require discussion? If so, how did you reach a decision regarding what was used?
- Do you think any elements of the chain of infection were broken?

**ASEPSIS AND YOUR PRACTICE**

Please note: this chapter does not provide a prescribed step-by-step procedure outlining how you should perform an aseptic technique, due to the issues previously highlighted. It does, however, present the important principles you need to consider (see Clinical Skill 23.1) and apply in the care you deliver to patients.

To start with I found aseptic technique quite difficult – you have to concentrate the whole time, being very self-aware about what you are touching. I had to really think hard about when I put gloves on to overcome the temptation to wear them all the time just in case. My practice educator helped me to gain confidence by assessing any risks for blood and body fluids in advance according to what procedure I was doing. I now practice confidently knowing I have safe clean hands and my patients and I are both protected in the right way without wearing gloves unnecessarily.

Charlie Clisby, NQN
Guidelines

There will be various local guidelines of which you must be aware when undertaking aseptic procedures. Remember never to undertake this care unless you are, or have supervision from, a trained, experienced and competent person. The types of guidelines you need to consider will be contained in:

- infection prevention and control policies
- local organisational policies, e.g. wound dressing formulary.

CASE STUDY 23.1: TOM

Tom is 15 and has a learning disability. He has a chronic wound on his leg which is very painful and smells offensive. Tom is very conscious of the smell and dislikes having his dressing changed. He expresses his anxiety through his behaviour, which can be perceived as aggressive at times, especially by people who do not know him.

Tom really enjoys bathing, because he finds it relaxes him and he loves playing with his collection of boats, and plastic dinosaurs.

To make Tom’s dressing changes less stressful for him, your practice educator wants to use his bath as an opportunity to undertake his dressing change.

- What needs to be considered in order to prevent any contamination and support wound healing?

ACTIVITY 23.2: DECISION-MAKING

You and your practice educator are planning to dress a patient’s wound while they are sitting in the living room of their home. The room is very unclean - there are six cats who live in the house and they have used the living room carpet as a litter tray. The facilities for you to wash your hands are poor, the sink is dirty and there is no hand towel, no hot water and only a trickle of cold.

- What actions would you take to reduce contamination of the wound?

“

My name is Jan. I was admitted to hospital because I had diarrhoea which wouldn’t settle. Weeks I’ve had it. The docs think I might have an infection. I was given a foil container like a pie dish and told to do my business in it. How was I supposed to do that? And in a public toilet?

My nurse was wonderful. I didn’t have to tell him I was embarrassed or ask how to use it. He explained everything and helped set up the toilet so all I had to do was use it as usual. It’s a horrible thing to have to have somebody else handle your business but he was so professional and organised, there was no fuss and most of all I kept my dignity.

The results showed I did have an infection, and now I’m back to normal. I’m so grateful to him; I don’t think I could have faced doing it by myself.

Jan, patient

”
STEP-BY-STEP CLINICAL SKILL 23.1:
PRINCIPLES OF ASEPSIS

☑ Field-specific considerations

When caring for a patient with a learning disability, it is important to be mindful of their level of understanding, so that consent and cooperation for the procedure can be gained. You will need to allow time to explain what you are doing and consider whether it will cause discomfort or pain.

Patients who have impaired mental capacity may not understand why you need to undertake an aseptic procedure. They may therefore withhold consent and you may need to refer to local policies on presumed or assumed consent, which will reflect requirements of the Mental Capacity Act 2005 and best interest.

As younger children may not understand what you wish to do, you may need to modify your approach – it may be helpful to have the parents or carers present to assist.

☑ Care-setting considerations

Aseptic technique can be undertaken in any care setting, although you may need to think carefully about how best to manage the patient and the patient’s environment.

☑ What to watch out for and action to take

While undertaking an aseptic procedure, you should also assess:

- the general condition of the patient; specific elements will vary according to the procedure being undertaken (e.g. respiratory rate for care of a chest drain)
- their neurological condition – are they alert and responsive? Are they agitated?
- any signs or complaints of pain or discomfort
- the patient’s or relatives'/carers' views – for example, saying that their condition is ‘not quite right’ or they ‘don’t feel well’.

The information gained from these observations is additional to any assessment you make relating to, for example, the wound you are dressing and will enable you to fully assess the patient’s condition and institute appropriate treatment as necessary, escalating care needs to senior nurses and the medical team.

☑ Helpful hints

- Do I wear gloves and an apron? Gloves and aprons must only be worn if contact with blood/body fluids/excreta is anticipated or the patient is in source isolation for IPC requirements.
- Hand hygiene must be performed before touching a patient, before clean/aseptic procedures, after body fluid exposure/risk, after removal of gloves and after touching a patient or the patient’s immediate surroundings.
- Waste should be disposed of into the correct waste stream in line with a risk assessment.
## ASEPTIC TECHNIQUE GUIDELINES

### BEFORE YOU START

1. **Before commencing any care activity, introduce yourself to the patient, explain the procedure and gain their consent**
   - Fully informed consent may not always be possible if the patient is a child or has impaired mental capacity or learning disabilities, but even in these circumstances, every effort should be made to explain what you are going to do in terms that the patient can understand. This is not only respectful of their individual human rights, but also helps to ensure that they will be more accepting of the treatment and that their anxieties are reduced.
   - For patients who are unable to provide consent because they are unconscious, refer to local policies.

2. **Assess the procedure and determine its complexity before you start, collecting all equipment that may be needed (and an assistant/chaperone if required)**
   - Ensures you are fully prepared; also avoids you having to leave the patient or interrupt the procedure.

3. **Consider what is going on around you - do you really need to do an aseptic technique now (even if planned)? Is the patient due to have other investigations that will cause you to rush, e.g. an x-ray?**
   - Ensures that the environment is conducive to undertaking an aseptic technique - for example, there will be a negative environmental impact if bed-making or cleaning is being undertaken in close proximity to a large wound dressing being undertaken.

4. **Ensure the patient is in a comfortable position where you can access the appropriate area**
   - Ensure the patient has received appropriate analgesia as required.
   - Promotes patient comfort.

5. **Clear sufficient space within the environment, e.g. around the bed space, chair or treatment area**
   - **Ensure the area is private**
     - Enables clear access for the patient and the nurse to work safely.
     - Maintains patient privacy, dignity and comfort as required - patients will feel exposed if others can see the care they are receiving.

6. **Transport equipment to the patient appropriately (consider a dressing trolley if available and appropriate)**
   - Ensures all equipment is to hand.
   - Maintains cleanliness of equipment and aids transport of all items safely.

7. **Perform hand hygiene and apply non-sterile gloves only if required**
   - Wearing an apron and gloves is a standard infection prevention practice when dealing with body fluids or patients in isolation if they pose a risk of infection to others.
   - Ensure your use of PPE is appropriate by considering the individual patient situation and the risk presented. Appropriate hand hygiene will assist in preventing and controlling infection.

(Continued)
REMOVE AND DISPOSE

1. If present, remove any soiled dressings, ‘contaminated’ or ‘dirty’ items and place in appropriate waste bag according to risk assessment.
   - In preparation for dressing (etc.) change. Ensure soiled, contaminated or dirty items are disposed of appropriately in the offensive or infectious waste stream depending on risk assessment.

2. Remove gloves and perform hand hygiene.
   - Appropriate hand hygiene will assist in preventing and controlling infection.

CREATE A ‘STERILE FIELD’

1. If using a dressing pack, open sterile items and create your ‘sterile field’ by placing only sterile items within this area.
   - Creating a sterile field avoids contamination through direct contact with non-sterile items. Remember, your hands are not sterile!

2. Apply sterile or non-sterile gloves as required.
   - A risk assessment will determine if sterile or non-sterile gloves are required.

3. Undertake procedure ensuring:
   - Only sterile items come into contact with the susceptible site.
   - Sterile and non-sterile items do not come into contact with each other.
   - In order to prevent and control infection.
TO CONCLUDE

1. After completing the required care, ensure the patient is in a comfortable position, with drinks and call bells available as necessary. Promotes patient comfort and ensures they are well nourished and hydrated.

2. Dispose of all waste and any single-use equipment, discard PPE (if used) and perform hand hygiene. Clean any equipment used as per the relevant policy every time it is used. Prevents cross-infection and maintains equipment in working order.

3. Record the care provided in the patient’s record. Maintains patient safety and accurate records.

4. If any abnormal findings are observed, report to your practice educator or a registered nurse immediately. It is vital to report abnormal findings to a registered nurse immediately so they can ensure care is escalated; failure to do so can result in the patient’s condition deteriorating and potentially preventable adverse outcomes.

Source: Loveday et al. (2013); NICE (2017); WHO (2009)

SPECIMEN COLLECTION

Analysis of a specimen will determine important clinical decisions regarding a patient’s condition or disease progress. Results also influence choices regarding ongoing or new treatment such as the prescribing of antibiotics. A specimen received at the laboratory in poor condition or taken inappropriately could therefore have profound implications for patient care, diagnosis and timeliness of any subsequent treatment. Therefore, the quality of a specimen is very important.

Nurses commonly take or handle specimens such as:

- wound swabs
- faeces
- urine
- blood (cultures or samples for analysis)
- sputum
- nasal and throat swabs (for detection of infection or screening for presence of clinically important bacteria, e.g. MRSA, Group A Streptococci).

As previously outlined, specimens are tested for a multitude of reasons, which can be summarised as:

- screening – for disease or the presence of micro-organisms such as malarial parasites, MRSA or Carbapenemase Producing Enterobacteriaceae (CPE)
- case finding – similar to screening but in a defined population such as blood tests for BRCA1 and 2 genes for breast cancer
SKILLS FOR NURSING CARE

- monitoring – for example, to reveal levels of drugs in the body, e.g. Gentamicin, Vancomycin
- diagnosis – to confirm or exclude the presence of disease.

I would discuss with the patient the reasons why we need to collect a specimen and gain consent. I would then discuss with them to see if they are happy to take their own nasal swab. If not I would explain what I need to do and collect the specimen as quickly and confidently as I could. Before collecting the specimen I would read up on the procedure and discuss with my practice educator, role playing it through with them if necessary.

Laura Grimley, adult nursing student

The importance of multi-disciplinary relationships

As mentioned previously, patient outcomes are directly influenced by the quality of specimens. The collection, transport, processing, analysis and results reporting of specimens all require efficient and reliable multi-disciplinary relationships to exist. Those involved in this process include nurses, porters (or transport drivers in community settings), laboratory reception staff, laboratory staff, technicians and biomedical scientists, plus doctors to interpret and communicate results. Each element of this relationship is of equal importance and a problem in any part has the potential to adversely affect the accuracy of the result of the specimen testing.

WHAT’S THE EVIDENCE?

Historically, evidence has developed to inform laboratory techniques and ‘standard operating procedures’, but these do not in themselves often reflect the patient experience. One example of where patient experience can be adversely affected is the contamination of blood culture specimens, for example when MRSA is identified as a result of poor technique due to skin contamination at the venepuncture site. If we think about what Jan said in his patient voice, however, he makes it very clear that patients value the support, advice and experience a nurse can offer to ensure that specimen collection is as simple and convenient as possible.

Laboratory testing

As there are endless different ways in which a specimen can be analysed, a summary of common laboratory types and the tests they undertake are presented in Table 23.1. Not all healthcare providers will have all types of laboratory services: refer to your local provider’s website for detailed information on what laboratory services are available.

Note: this chapter does not include the use of rapid diagnostic tests – use of these is subject to local policies and procedures in line with the manufacturer’s guidance.

Principles common to the effective collection of all types of specimen

There are a number of principles which are applicable to the collection of all specimens.
ASEPTIC TECHNIQUE AND SPECIMEN COLLECTION

There are various national and local guidelines of which you must be aware when collecting specimens. Remember never to undertake collection of any type of specimen unless you are, or have supervision from, a trained, experienced and competent person. The types of guidelines you need to consider will be contained in:

- infection prevention and control policy
- local pathology policies/guidelines.

Once you are aware of these, you need to undertake a number of steps that will be common for the collection of all types of specimen (see Clinical Skill 23.2).

The usual procedures for collecting common specimens are described below, but remember that this should be read in conjunction with relevant local policies. At all times, complete the common steps (Clinical Skill 23.2) and ensure appropriate adjustments are made to suit the needs of the individual patient – local guidelines will be helpful for this.

**ACTIVITY 23.3: CRITICAL THINKING**

Tilly is three months old and has persistently loose, watery and offensive-smelling bowel motions. Her mother has been asked to collect a stool specimen so it can be tested, but is uncertain how she should do this.

- How would you explain the procedure?
STEP-BY-STEP CLINICAL SKILL 23.2: COMMON STEPS FOR THE COLLECTION OF ALL TYPES OF SPECIMEN

☑ Essential equipment – depends on the specimen but is likely to include one or more of the following:
- specimen container, specimen bag and laboratory form
- swabs as appropriate.

☑ Field-specific considerations
When collecting a specimen from a patient with a learning disability, it is important to know their level of understanding so that consent and cooperation can be gained. You will need to allow time to explain what you are doing, why you are doing it and whether it will cause discomfort or pain.

Patients who have impaired mental health may not understand why you need to collect a specimen. They may therefore withhold consent and you may need to refer to local policies on presumed or assumed consent, which will reflect requirements of the Mental Capacity Act 2005 and best interests.

Younger children may not understand why you need to collect a specimen. You will need to adopt an appropriate approach for consent. It may be helpful to have the parents or carers present to assist.

☑ Care-setting considerations
- Consideration must be given to any equipment needs, e.g. the refrigeration of viral transport media.
- Specimens can be collected in most care settings.

☑ Key points to remember
- There is a clear clinical need for the specimen.
- Explain the rationale to the patient and gain consent (refer to Chapter 6).
- The specimen must be obtained without contamination.
- The specimen must be stored appropriately or transferred to a laboratory as soon as possible.
- Check result and act on it accordingly.

☑ Helpful hints
- Gloves and aprons must be worn only if contact with blood/body fluids/excreta is anticipated or the patient is in isolation.
- Hand hygiene must be performed before touching a patient, before clean/aseptic procedures, after body fluid exposure/risk, after touching a patient and after touching a patient’s surroundings.
- Waste should be disposed of in a waste bag if it is contaminated with blood/body fluids/excreta in line with risk assessment for waste, e.g. offensive or infectious waste.
### SPECIMEN COLLECTION GUIDELINES

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<td>The first step of any procedure is to introduce yourself to the patient, explain the procedure and gain their consent. Fully informed consent may not always be possible if the patient is a child or has impaired mental health or learning disabilities, but even in these circumstances, every effort should be made to explain the procedure in terms that the patient can understand. This is not only respectful of their individual human rights, but also helps to ensure that they will be more accepting of the treatment and that their anxieties are reduced. For patients who are unable to provide consent because they are unconscious, local policies should be referred to.</td>
<td>Clear sufficient space within the environment where the specimen is to be collected, for example around the bed space or chair. Enables clear access for the patient and the nurse to safely use the equipment required.</td>
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<td>Ensure that it is an appropriate time to collect the specimen. The quality of the specimen can be affected by the time of collection and length of time before it reaches the laboratory. To make sure the specimen is of the best quality, ensure that it will reach the laboratory quickly once it has been collected and that it is the best time of day to collect the specimen. For example, it is best to collect a urine sample from the first voided urine in the morning for mycobacterial culture as this will contain the highest concentration of bacteria present.</td>
<td>Standard precautions should be used whenever there is a need to collect specimens. Wearing an apron and gloves as part of personal protective equipment (PPE) is a standard infection-control procedure when there is contact with body fluids or a patient is in isolation. Ensure your use of PPE such as gloves and disposable aprons is appropriate by considering the individual patient’s situation and the risk presented. Do not use ‘just in case’.</td>
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<td>Gather the equipment required to collect the specimen; ensure this is clean and in working order. Reduces the chance of inaccurate results. All lids, containers and specimen bags should be checked to ensure there are no leaks or breaches which could result in spillage during transportation. Containers used for the collection and transportation of specimens should be CE-marked as this confirms that the container complies with essential requirements – only approved containers should contain specimens for laboratory analysis. This reduces the chance of infection and helps maintain the quality of the specimen.</td>
<td>Patients need to be in a private, comfortable and appropriate position and surroundings. Maintain patient privacy, dignity and comfort as required. To promote patient comfort and reduce anxiety.</td>
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<td>Complete the appropriate laboratory forms. The information provided on specimen or laboratory forms is very important. Incorrectly spelled or wrong patient names and identifying information could result in the wrong result being placed in a patient’s notes. Alternatively, poorly completed forms could result in specimens being rejected by the laboratory, with significant implications for the patient. Some organisations use electronically generated specimen request forms and specimen labels to support laboratory tests. Always check local policies for more information.</td>
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The laboratory request form must include the following information:

- patient surname and forename (care should be taken to avoid use of nicknames)
- date of birth
- gender
- NHS or hospital number – refer to local policies regarding patient unique identifiers and their use
- location of where specimen obtained (if relevant)
- requesting clinician or consultant in charge
- sample date and time
- name or initial of the person taking the specimen
- clinical information relevant to the specimen – this helps laboratory staff to interpret the clinical significance of specimen results. Examples include symptoms, possible or confirmed diagnosis, any current treatment (e.g. antibiotics) and other pertinent history such as foreign travel

Sample type | Details
--- | ---
Wound swab | Skill 23.3
Faecal specimen | SAGE edge
Urine sample | SAGE edge
Blood sample | p. 379
Sputum sample | Skill 23.4
Nasal swab | Skill 23.5
Throat swab | Skill 23.6

Specimens for microbiological investigation should ideally be taken before antibiotic therapy is commenced

If the patient is already on antibiotics before a specimen is taken, this may have a significant impact on identification of the causative organism (bacteria). The laboratory must be informed on the laboratory form of all therapy the patient is receiving or has recently received. Where sepsis is suspected, patients should have specimens taken if possible before commencing treatment, however this should not delay the administration of antibiotics.

Specimens for viral investigation can require special transport media

Viruses are generally quite fragile and die easily

Examples include chickenpox (varicella), chlamydia, influenza, norovirus

Where specimens are taken directly from lesions, such as vesicles of herpes or chickenpox, then the swab must be placed inside special viral transport media to preserve any viral particles during transport to the laboratory. Viral transport media may require refrigeration and will have an expiry date. Refer to local policies for more information.

Double-check to ensure the patient is correctly identified – ask the patient (where possible) to state their full name and date of birth. Use patient identifiers (e.g. wristbands) where possible to confirm

Prevents you from taking a specimen from the wrong patient. Never ask ‘are you ...?’ Always ask the patient to state their name and date of birth

Some patients may not wear wristbands, e.g. neonates, those living in care homes or those with amputated limbs – check your local policies for alternatives to wristbands

Ensure specimen is collected in line with local policy

Using an aseptic technique reduces the risk of contaminating the specimen

Further details relating to taking the following specimens are available within the following:

Label container and seal in the specimen bag along with the laboratory request form, in line with local policy

Ensures the specimen and laboratory form are retained together and avoids loss of either during transport

Specimens should be transported to the laboratory and processed as soon as possible

Once a specimen is obtained, any micro-organisms present have been removed from their ‘natural’ habitat; therefore in order to preserve micro-organisms, transport to the laboratory should take place as soon as possible. If there is a delay in transportation,
some specimens may be refrigerated in a designated refrigerator (do not put in a food fridge) until collection. This is preferable to leaving them at room temperature, which could interfere with the laboratory interpretation of results.

For some specimens, delays of over 48 hours are considered unsatisfactory as the specimen will have deteriorated. Check local policy for further guidelines.

After collecting the specimen, ensure the patient is in a comfortable position, with drinks and call bells available as necessary.

Promotes patient comfort and ensures they are well nourished and hydrated.

Discard PPE, any single-use equipment and other used materials as per policy. Clean any equipment used as per the relevant policy and perform hand hygiene. Prevents cross-infection and maintains equipment in working condition.


Source: WHO (2009)

Collecting a blood sample

Nursing students in the UK would not take blood cultures or other blood specimens. However, knowledge of the process will enable you to support a patient when they are having a blood sample taken:

- Blood cultures are taken to determine the presence of bacteria or fungi in the blood (bacteraemia or fungaemia, respectively). Blood cultures should not be routinely taken, only when clinically indicated.
- Haematological investigations are undertaken to ascertain, for example, the patient’s haemoglobin or platelet levels.
- Biochemical investigations are undertaken to ascertain, for example, the patient’s potassium and sodium levels.

While blood cultures should be taken using a strict aseptic technique; there is less emphasis on this for routine blood tests (haematology or biochemistry). The other common steps and fundamental principles still apply, however, and there needs to be careful attention paid to prevent any sharps injuries. Many healthcare staff undertake venepuncture training but require additional knowledge and skills to be assessed as competent to provide this care.

ACTIVITY 23.4: CRITICAL THINKING

Jasmin is 3 years old and requires a blood sample to be taken:

- What might help Jasmin feel safe and comfortable so that the sample can be collected?
- Jasmin’s mum is with her, and is very anxious and concerned about the blood test. How can you ensure that her worries do not impact on Jasmin’s care?
STEP-BY-STEP CLINICAL SKILL 23.3: TAKING A WOUND SWAB

Wounds include surgical and traumatic wounds, burns, ulcers, folliculitis and invasive device insertion sites such as an intravenous cannula or wound drain.

**Indications for taking the specimen**

Wound infection, cellulitis (in the presence of a break in the skin) and/or the presence of pus.

The presence of bacteria in a wound without signs and symptoms of infection reflects colonisation only, and is common in chronic wounds (such as leg ulcers in community settings).

**WOUND SWAB GUIDELINES**

1. Perform steps 1-8 of the common steps (see Clinical Skill 23.2)
   To prepare the patient and yourself to undertake the task

2. Dip swab in transport media (if present with swab) or moisten with sterile saline
   To preserve any bacteria present during transportation to the laboratory
   Moisten swab to avoid **dessication** of any bacteria present

3. **If pus is present, collect pus (via aspiration) or use a moistened swab**
   To preserve any bacteria present during transportation to the laboratory

4. Take swab from the part of the wound exhibiting symptoms of infection
   This area will produce the best results

5. Using an aseptic technique, perform a ‘zig-zag’ motion while gently rotating the swab between the fingers
   To ensure good contact by the swab with the wound

6. Place the wound swab immediately back into the container
   To prevent contamination

7. Perform steps 10-16 of the common steps (see Clinical Skill 23.2)
   To ensure that:
   - the patient is safe and comfortable
   - the specimen has been correctly collected and documented in the patient’s records
   - the equipment is clean and in working order

Source: PHE (2016a)
STEP-BY-STEP CLINICAL SKILL 23.4: COLLECTING A SPUTUM SAMPLE

- **Indications for taking the specimen**
  Upper and lower respiratory tract infections, including pneumonia.
  Micro-organisms normally present in the upper respiratory tract can contaminate the usually sterile lower respiratory tract and cause infection.
  **Green sputum does not necessarily mean the patient has an infection!**

**SPUTUM SAMPLE GUIDELINES**

1. **Perform steps 1–8 of the common steps (see Clinical Skill 23.2)**
   To prepare the patient and yourself to undertake the skill

2. **The patient is required to expectorate in order to produce a specimen of sputum – saliva is not suitable**
   Patients who have difficulty coughing or expectorating may need a physiotherapist to help them produce a sample

3. **As necessary, place sample in specimen container, carefully avoiding contamination of the outside of the pot**
   A minimum of 1 ml of sputum is required

4. **Samples should be sent to the laboratory as soon as possible (sputum may be refrigerated for up to 2-3 hours)**
   Some bacteria die easily and overgrowth of other bacteria occurs quickly at room temperature, which will produce false results

5. **Perform steps 10-16 of the common steps (see Clinical Skill 23.2)**
   To ensure that:
   - the patient is safe and comfortable
   - the specimen has been correctly collected and documented in the patient’s records
   - the equipment is clean and in working order

Source: PHE (2016b)
STEP-BY-STEP CLINICAL SKILL 23.5:
TAKING A NASAL SWAB

**Indications for taking the specimen**
To detect clinically important bacteria in the nose, for example to determine if the patient is colonised with a bacteria such as meticillin-resistant *Staphylococcus aureus* (MRSA) or meticillin-sensitive *Staphylococcus aureus* (MSSA).

**NASAL SWAB GUIDELINES**

1. **Perform steps 1–8 of the common steps (see Clinical Skill 23.2)**
   - To prepare the patient and yourself to undertake the skill

2. **Dip swab in transport media (if present with swab) or moisten with sterile saline**
   - One swab can be used to swab both nostrils
   - To preserve any bacteria present during transportation to the laboratory
   - Moisten swab to avoid dessication of any bacteria present

3. **Swabs must be taken from the anterior nares of the nose (see Figure 23.2).**

4. **Perform steps 10–16 of the common steps (see Clinical Skill 23.2)**
   - The swab should be inserted just inside the nostrils and then directed gently upwards back towards the tip of the nose and rotated to ensure gentle contact with the mucosal surface
   - The anterior nares are the external part of the nostrils (see Figure 23.2)
   - To ensure that:
     - the patient is safe and comfortable
     - the specimen has been correctly collected and documented in the patient’s records
     - the equipment is clean and in working order

Source: Dougherty and Lister (2015); PHE (2015a)

*Figure 23.2 Nasal swab*
STEP-BY-STEP CLINICAL SKILL 23.6: TAKING A THROAT SWAB

**Indications for taking the specimen**
To detect a throat infection or carriage of clinically important bacteria, such as MRSA, or occasionally for screening in outbreak or contact situations with, for example, Group A Streptococci, *N. meningitides*.

**THROAT SWAB GUIDELINES**

1. Perform steps 1–8 of the common steps (see Clinical Skill 23.2)
   To prepare the patient and yourself to undertake the procedure

2. Depress the tongue to expose the fauces of the tonsils (see Figure 23.3) and gently and quickly rub the swab over the affected or inflamed area
   Ensure you have good lighting present to enable you to see into the throat
   The fauces or ‘pillars of fauces’ are two membranous folds which enclose the tonsils

3. Perform steps 10–16 of the common steps (see Clinical Skill 23.2)
   To ensure that:
   - the patient is safe and comfortable
   - the specimen has been correctly collected and documented in the patient’s records
   - the equipment is clean and in working order

**CASE STUDY 23.2: MRS NEUmann**

Mrs Neumann is 58 and is undergoing a pre-operative assessment prior to a total hip replacement. As part of this, she requires a ‘screen’ for MRSA. This is local hospital policy. Mrs Neumann is very anxious about this and has read about ‘superbugs’ in the newspapers. She is worried that the fact she is having a screen means that she may have MRSA.

- How will you reassure Mrs Neumann regarding the need for an MRSA screen and gain her consent for the test (a nose swab)?
- What additional information can you provide for her while she awaits the result?
Fully prepare the patient about what you need to do so they are relaxed and can cooperate. Ensure you have all the equipment needed and you are very familiar with the procedure you are carrying out. If you are unsure about anything ASK. Don’t worry and relax, the patient is usually more anxious then you are.

Julie Davis, LD nursing student

CONCLUSION

Maintaining asepsis and the accurate collection of specimens from patients are two skills fundamental to the role of the nurse.

It is most important when you are performing an aseptic technique to understand the principles, which you can then apply to any situation. This is a much safer way to work than just being able to follow a procedure without the ability to customise it to meet a patient’s or their immediate environment’s specific needs.

The ‘chain of infection’ describes a series of events that need to take place for infection to occur, regardless of whether this is caused by a virus, bacteria or fungi. For an infection to occur, all links in the chain must be present. An aseptic technique, when performed correctly, will ‘break’ the chain of infection and, by doing so, protect the patient.

There are numerous reasons why we would request a specimen from a patient, ranging from assisting in the diagnosis of disease or infection, through monitoring recovery from illness, to the need to take specimens outside of traditional healthcare settings, such as in professional sport. Always ensure that contamination is avoided whenever a specimen is collected, as this is critical to obtaining accurate results.
**CHAPTER SUMMARY**

- An aseptic technique protects the patient by preventing contamination of vulnerable body sites or contamination of specimens.
- Considerable confusion exists between the use of clean and aseptic techniques.
- It is important to understand the principles of asepsis, so you can apply them appropriately to the care required by patients.
- Specimens should only be sent when clinically indicated.
- Specimen or laboratory forms must be fully completed, with all relevant clinical information included.
- Storage, transport and time taken for specimens to reach the laboratory can impact on results.

**CRITICAL REFLECTION**

*Holistic care*

Maintaining asepsis and the appropriate and correct specimen collection are important in providing holistic care for a patient. Review the chapter and note down all the instances in which the care actions outlined can help you meet a patient’s wider physical, psychological, social, economic and spiritual needs. Think of a variety of different patients across the fields, not just within your own field. You may find it helpful to make a list and refer back to it next time you are in practice, and then write your own reflection after your practice experience.

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Knell, C., Pellow, C. and Potter, J. (2009) ‘Long-term urethral catheter audit in patients’ own home’, *Journal of Infection Prevention*, 10(2): 62–5. This article describes risks associated with infection and indwelling urinary catheters. It acknowledges the importance of focusing on care in settings outside acute healthcare facilities and describes a programme of audit in order to improve the care that patients receive who have these devices in situ. Issues relating to urine specimen collection were identified as an area of practice to improve.

Jones, P. (1995) ‘Pioneers of the transition from antiseptic to aseptic surgery’, *Journal of Medical Biography*, 3: 201–6. This article describes the historical development of asepsis within the theatre context.
Other journal article


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NHS Choices (2014) How Should I Collect and Store a Urine Sample? Available at: www.nhs.uk/chq/Pages/how-should-i-collect-and-store-a-urine-sample.aspx?CategoryID=69&SubCategoryID=692. This is useful information for patients and the general public on specimen collection and storage of urine samples, one of the most common samples requested.


National Institute for Health and Care Excellence (NICE) (2014) Quality Standard QS 61: Infection Prevention and Control. Available at: www.nice.org.uk/guidance/qs61. This NICE Quality Standard describes ambitious standards that organisations should aim to meet to support ongoing improvements in IPC and reductions in associated HCAI.

ACE YOUR ASSESSMENT

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- Test yourself with multiple-choice and short-answer questions.
- Revise key terms with the interactive flash cards.

REFERENCES


Chapter 9  Respiratory diseases

Chapter aims

After reading this chapter you will be able to:

• describe the risk factors, pathophysiology and clinical features of asthma and chronic obstructive pulmonary disease (COPD);
• relate the signs and symptoms of asthma and COPD to the underlying pathophysiology;
• explain how drugs for asthma and COPD exert their actions and can cause side-effects;
• describe the important drug interactions, cautions and contraindications of these drugs;
• explain how current guidelines are used to choose different drugs for patients with asthma and COPD.

Case study

Peter, a retired teacher aged 65 years, first developed symptoms of chronic obstructive pulmonary disease (COPD) five years ago. Prior to his diagnosis Peter smoked around 20 cigarettes per day. He started smoking cigarettes at school when he was 14 years old. Since his diagnosis of COPD he has managed to stop smoking.

Peter first noticed symptoms of breathlessness on exertion and a cough with the production of sputum (referred to as a productive cough). He initially put these down to the effects of ageing along with his smoking habit. As his breathlessness worsened his wife encouraged him to see his GP. The practice nurse completed a lung function test using a spirometer (see under ‘Lung function tests’). This indicated mild COPD. Peter was also given a blood test to rule out anaemia as a cause of his breathlessness. He was prescribed a salbutamol inhaler to be used as required.
Introduction

Peter is typical of someone presenting with COPD. In this chapter we examine COPD and asthma, both of which are common lung disorders. Asthma often starts in childhood but COPD is predominantly diagnosed in those in their 50s or older. Both asthma and COPD are classified as obstructive pulmonary disorders. Obstructive pulmonary disorders are characterised by difficulties in breathing, especially breathing out (exhalation). There is an ‘obstruction’ to airflow which may be airway inflammation as is present in both asthma and COPD, or damage to lung tissue which is also characteristic of COPD.

Other common lung diseases can be classified as restrictive. Restrictive diseases include pulmonary fibrosis of which there are various types, including pneumonia and asbestosis (Chapter 2). Restrictive diseases are characterised by a ‘restriction’ on lung expansion causing a reduction in lung capacity. Restriction may be due to destruction of lung tissue, as is the case in pulmonary fibrosis where chronic inflammation and scarring destroys lung connective tissue. Restriction can also be due to problems outside the lung, including problems with the breathing muscles or the nerves supplying these. This is the case with, for example, Guillain–Barré and motor neurone disease. Other important respiratory conditions include upper and lower respiratory infections (such as the common cold, tuberculosis, influenza; Chapter 3); lung cancer; cystic fibrosis.

We begin this chapter by reviewing the structure and function of the respiratory system. We focus on aspects of anatomy and physiology which are needed to understand the clinical presentations of asthma and COPD. We then look at the risk factors, pathogenesis and clinical manifestations of asthma and COPD. We then examine the drugs used to relieve symptoms of asthma and COPD, and how different drugs are chosen for patients. We will also consider the side-effects, contraindications, cautions and interactions.

Review of the normal structure and function of the respiratory system

The respiratory system along with the cardiovascular system functions to deliver oxygenated blood to the cells of the body for cellular respiration. The two systems also function to remove carbon dioxide from the body to maintain the acid–base balance of the blood. The respiratory system draws air into the lungs in a process called inhalation, and removes air by exhalation. Together, the movement of air into and out of the lungs is known as pulmonary ventilation.

The upper respiratory tract includes the nasal cavity, pharynx and larynx (Figure 9.1). The lower respiratory tract includes the conducting airways making up the tracheobronchial tree. This is made up of the trachea, the bronchi and bronchioles. With the exception of the smallest bronchioles, these airways contain smooth muscle
tissue in their walls. The smooth muscle regulates the diameter of the airways and plays an important role in the response to environmental allergens in asthma (see under 'Asthma', below). The airways are lined with a ciliated epithelium whose function is to trap inhaled dust particles and remove these from the airways. The ciliated epithelium contains goblet cells which secrete mucus. Inhaled dust particles entering the lungs during inhalation are trapped by the mucus. The cilia beat continually to move the mucus upwards and out of the lungs.

*Figure 9.1* (A) Main organs of the respiratory system. (B) Bronchioles, respiratory bronchiole and alveoli. (C) Smooth muscle is shown in the walls of the bronchioles.
Airway resistance and compliance are important terms needed to understand the difficulties in breathing experienced by patients with asthma and COPD. Airway resistance describes the resistance to airflow through the airways. The diameter of the airways is an important factor in determining airway resistance. Compliance is a measure of the ease with which the lungs and chest cavity can expand. Lung compliance is affected in patients with emphysema, one of the component diseases in COPD.

The smaller (respiratory) bronchioles and alveoli are the site of gas exchange. The alveoli are microscopic air sacs. These contain very thin walls made up of a single layer of epithelial cells lined with fluid. There are approximately 500 million air sacs in each lung, providing in total an extensive surface area for the exchange of oxygen and carbon dioxide. Each collection of air sacs is supplied with a network of blood capillaries from the pulmonary circulation.

The barrier or ‘interface’ between the air and the blood is known as the **respiratory membrane**. It consists of the wall of the alveolus and the wall of the blood capillary, each of which are only one cell thick (Figure 9.2). Between the two layers of cells is a thin layer of connective tissue called the interstitium. The respiratory membrane is extremely thin to allow oxygen to diffuse from the air in the lungs to reach the blood. Carbon dioxide diffuses in the opposite direction – from the blood into the air in the lungs to be exhaled. Any damage or thickening of the respiratory membrane will reduce the diffusion of gases and make gas exchange less effective. Gas exchange is driven by the concentration gradients of oxygen and carbon dioxide across the respiratory membrane. The role of breathing is to maintain these diffusion gradients (Tortora and Derrickson, 2017).

![Respiratory membrane diagram](image)

**Figure 9.2** A single alveolus showing the structure of the respiratory membrane across which oxygen and carbon dioxide pass by diffusion
Chapter 9

The lungs must also be effectively perfused. The pulmonary circulation supplies blood to the lungs for gas exchange. The pulmonary circulation is a low pressure circuit arising from the right side of the heart. Blood passing through the pulmonary capillaries becomes fully oxygenated. The pulmonary veins return oxygenated blood to the left atrium.

Many diseases of the lungs will affect the process of gas exchange. In turn, altered gas exchange can lead to alterations in blood gases such as hypoxaemia (low oxygen levels) and hypercapnia (elevated carbon dioxide levels). Gas exchange can be affected through reducing the volume of air moved during breathing, by altering the respiratory membrane to reduce the effectiveness of gas exchange or by reducing lung perfusion.

Breathing is regulated through the respiratory centres in the brainstem. The most important stimulus to increase breathing rate and depth is the acid–base balance of the blood. Respiratory acidosis evokes a strong stimulus to increase the breathing rate (Tortora and Derrickson, 2017). Retention of carbon dioxide due to difficulties in breathing is one of the main causes of respiratory acidosis. This is important to bear in mind when we consider asthma and COPD.

In the next section, we describe asthma, one of the most common respiratory disorders.

Asthma

Case study

Rachel, a 35-year-old musician, was admitted to the medical admissions unit with severe breathlessness for 2 hours. Rachel has a past history of asthma since childhood. Her asthma is normally reasonably well controlled with medication. Two days ago, she developed an upper respiratory tract infection which is a known trigger factor for her asthma.

On the medical assessment unit, she was unable to complete a whole sentence and her neck muscles were prominent when inhaling. Rachel was notably anxious. She struggled to produce a peak flow of 105 L/minute – her normal peak flow is 400 L/minute. Her heart rate was 132/minute and respiratory rate 30/minute. She had a distinct expiratory wheeze. Her oxygen saturation was 91%. Rachel was given oxygen therapy to maintain oxygen saturation at 94–98%, a short-acting β₂-agonist through a nebuliser and an oral corticosteroid.

Rachel has experienced a severe acute asthma exacerbation. Fortunately for her, these exacerbations are rare. Rachel has a good understanding of her asthma and the need for maintaining her regular medication. Her personalised asthma plan enables her to act appropriately when her asthma worsens and seek help quickly.
Asthma is a chronic inflammatory condition affecting the airways. Typical symptoms include breathlessness, tightness in the chest, coughing and wheezing. The prevalence of asthma is increasing and asthma is more common in developed countries. Some of the highest rates are found in New Zealand, Australia and the UK. The reason for this is not fully understood. In the UK, 5.4 million people are receiving treatment for asthma and 1.1 million of these are children. Asthma is estimated to cause 1000 deaths a year in the UK, with 90% of these attributed to preventable factors (BLF, 2018). Although the death rate from asthma is low, asthma causes considerable distress and contributes to days off school for children and days off work for adults. Anxiety and depression are up to six times higher in those with asthma than the general population.

At a physiological level, asthma has three characteristics.

- Airflow limitation which is normally reversible with treatment.
- Airway hyper-responsiveness to a number of stimuli or trigger factors. Bronchospasm occurs in response to a number of stimuli and is the sudden contraction of the smooth muscle in the wall of the bronchi. It results in narrowing of the airways and obstruction to breathing.
- Inflammation of the bronchi. Chronic inflammation of the bronchi is present in asthma. This is characterised by the presence of eosinophils (Chapter 4). These contribute to the long-term manifestations of asthma.

The aetiology of asthma is complex involving both genetic and environmental factors. Asthma is often provoked by exposure to environmental stimuli. Table 9.1 gives examples of possible environmental stimuli that can provoke asthma.

<table>
<thead>
<tr>
<th>Stimulus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Environmental allergens, e.g. pollen, house dust, animal hair, latex</td>
</tr>
<tr>
<td>Respiratory tract infections</td>
</tr>
<tr>
<td>Exercise</td>
</tr>
<tr>
<td>Occupational sensitisers, e.g. isocyanates (from polyurethane varnishes)</td>
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<tr>
<td>Cold air</td>
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<tr>
<td>Ingestion of NSAIDs</td>
</tr>
<tr>
<td>Emotional stress</td>
</tr>
<tr>
<td>Exposure to bronchial irritants, such as cigarette smoke, perfume</td>
</tr>
</tbody>
</table>

Table 9.1  Environmental stimuli provoking asthma

In common with many conditions we have included in this book, including dementia, cancer and heart failure, asthma is seen as an ‘umbrella’ term for a range of different diseases. This reflects our increasing understanding of the different mechanisms by which the disease or condition arises.
In asthma there are many different immune mechanisms which can produce a clinical picture of asthma. Recent research has highlighted a number of different asthma ‘phenotypes’. Asthma phenotypes represent different presentations of asthma, and include: early onset atopic, late onset atopic, obesity-related non-atopic and elderly (non-atopic) (Kuruvilla, 2018). If you remember from Chapter 4, ‘atopic’ or ‘atopy’ refers to the production of IgE antibodies in response to an allergen. Atopy is the hallmark of a type-1 hypersensitivity reaction, which is responsible for a wide-range of allergies. Atopy in turn, is driven by what was referred to in Chapter 4 as a Th2 response. For reasons not fully-elucidated, those people who develop allergies or asthma, produce T helper cells of type 2 (Th2) as opposed to T helper cells of type (Th1). It is the Th2 cells that cause production of IgE antibodies through the different cytokines they produce. The atopic/non-atopic distinction is one of the most important means of classifying asthma. This principle is important in choosing the most important treatment and management strategies for the patient. This has proved especially important in treating the patients with more severe forms of asthma in which monoclonal antibody therapy against IgE may be used (Eller et al., 2018).

For the purposes of this chapter we will focus on the pathogenesis of atopic asthma because it is one of the most common types of asthma and one of the best understood.

**Pathogenesis**

In atopic asthma, a sensitisation phase occurs in which exposure to an allergen causes the development of a Th2 response. Th2 cells release cytokines IL-4, -5 and -13. These in turn lead to B cell production of IgE antibodies, the recruitment of white blood cells called eosinophils. The IgE antibodies bind to mast cells. With respect to asthma there are many mast cells located within the lining (or mucosa) of the airways. The binding of IgE antibodies to mast cells lining the airways sensitises an individual to further exposure to the allergen.

On subsequent exposure to the same allergen, the allergen will be recognised and bind to the IgE molecules on the mast cells. This signals the immediate release of histamine – a potent inflammatory mediator and a range of other inflammatory mediators from the mast cells. Other mediators released include leukotrienes, prostaglandins and pro-inflammatory cytokines (Chapter 2). These mediators produce an immediate allergic reaction within seconds of exposure. This immediate reaction results in airway swelling and oedema, and stimulates the mucus glands and goblet cells in the airways to increase mucus secretion.

Bronchospasm is caused by a mixture of direct stimulation of the smooth muscle cells by the mediators and by reflexes involving the nervous system. The airways contain smooth muscle tissue which regulates the diameter of the airway. The contraction of the smooth muscle is under control of the autonomic nervous system. Nerve fibres from the sympathetic division of the autonomic nervous system cause bronchodilation – or
widening of the bronchi – through relaxing the smooth muscle. Stimulation from the parasympathetic division causes bronchoconstriction through causing the smooth muscle to constrict. During an asthma attack, local sensory fibres (probably of type C; Chapter 6) are stimulated and cause a reflex firing of parasympathetic fibres. This causes the bronchospasm characteristic of asthma. We will return to the regulation of the airways by the autonomic nervous system when we discuss β₂-agonists in the section on pharmacological treatments of asthma.

The immediate allergic reaction in asthma results in the narrowing of the airways and increased resistance to air flow through three mechanisms.

- Airway swelling due to inflammation and fluid exudates (oedema).
- Increased mucus secretion which can form mucous plugs in the airways.
- Bronchospasm.

Narrowing of the airways increases the resistance to air flow and the clinical features of asthma (described below).

Following the immediate reaction, patients may experience a late reaction, 8–12 hours later. This involves the infiltration of immune cells including eosinophils, lymphocytes (especially Th2 cells) and neutrophils. Much focus has been made on the role of eosinophils in asthma. Eosinophil infiltration is characteristic of asthma and a key cause of chronic inflammation of the airways. Eosinophils release harmful proteins and reactive oxygen species which damage the lining of the airways. This causes airway ‘re-modelling’ which describes the thickening of the walls due to mucous gland hypertrophy and the build-up of scar tissue. Chronic inflammation is also believed to make the airways hyper-responsive to other irritants.

Figure 9.3 illustrates the changes occurring in the airways in asthma during an asthma attack.

Clinical features

Rachel presented with typical symptoms of an asthma exacerbation. This included: wheezing, cough, chest tightness and breathlessness. The frequency and duration at which individuals experience asthma exacerbations varies greatly. Patients often experience prolonged expiration and hyper-inflation of the lungs may be visible. Nasal flaring and accessory muscle use are often evident. Children may show ‘retraction’ of the substernal, subcostal regions. Respiratory rate and pulse rate will be high. The reduced gas exchange brought about by the difficulties breathing can lead to low oxygen levels, raised carbon dioxide levels and low blood pH. These changes to blood gas levels and lowered pH in turn stimulate peripheral and central chemoreceptors to raise breathing rate and pulse (Tortora and Derrickson, 2017). The increased work of breathing may lead to exhaustion.
Asthma is characterised by the symptoms being intermittent and often worse at night. Symptoms will be provoked by the factors described in Table 9.1. However, some patients present without severe exacerbations but have chronic symptoms of cough and wheeze.

Management

An important aspect of asthma management is patient and family education. It is important for individuals with asthma to be taught the correct inhaler technique, to monitor their peak flow at regular intervals and, where possible, to avoid environmental triggers. Anyone with asthma needs to avoid exposure to tobacco smoke. NICE (2013a) recommends that everyone with asthma receive a written personalised action plan as part of structured education on asthma. This is where the educational role of the nurse is particularly important. Each person with asthma should be able to monitor and recognise when their symptoms deteriorate. They need to be aware of the actions to take should their asthma deteriorate. For some individuals, it will be appropriate for parents or a carer to be involved with review of the plan. This will be the case for children, those with learning difficulties and some older adults. In children, height and weight should be measured at least once a year because corticosteroid use can affect growth rate.

Before examining the drugs used to treat asthma, we examine the pathophysiology of COPD.
Chronic obstructive pulmonary disease (COPD)

COPD occurs following progressive lung damage and gradual worsening of lung function (NICE, 2018b). It is characterised by airflow obstruction which is not fully reversible. This absence of reversible airflow obstruction can help distinguish COPD from asthma. Most people with COPD are diagnosed in their fifties. NICE (2018b) state that there is an estimated 1.2 million people in the UK with COPD and many more people remain undiagnosed.

The main cause of COPD is smoking or exposure to environmental pollution. There is a rare genetic cause in those who inherit a faulty gene for α-antitrypsin (whose role in COPD is described below under ‘Pathogenesis’). Despite the link with tobacco smoking, it is of note that only a minority of smokers develop COPD. Therefore it is likely that as yet unidentified genetic factors play a role in the development of COPD.

Clinical features

Peter in the case study at the beginning of the chapter had mild COPD. Peter presented with a productive cough and breathlessness on exertion. He is likely to have experienced chest tightness and a wheeze. Patients with COPD are susceptible to frequent lung infections which may cause exacerbations in symptoms. Severe COPD causes patients to be breathless even at rest, with a prolonged expiration (‘out breath’). Patients will use their accessory muscles of breathing; chest expansion is poor and the lungs are likely to be ‘hyper-inflated’. This can lead to a ‘barrel-chest’ appearance. The patient may sit forward in a hunched position to aid their breathing. There may also be peripheral and central cyanosis.

In advanced disease, there may be weakness and skeletal muscle wasting.

Diagnosis

A diagnosis of COPD is made based upon the patient’s history. This will almost invariably include a history of smoking or exposure to environmental irritants. The following symptoms are likely to be present (NICE 2018b):

- exertional breathlessness
- chronic cough
- regular sputum production
- frequent winter ‘bronchitis’
- wheeze.

A diagnosis will be supported by spirometry which is used to determine lung function. The lung functions measured by spirometry include lung volumes and the rate at which air can
be exhaled from the lungs. Spirometry is used in diagnosing and monitoring a number of respiratory diseases. Its use is described under ‘Lung function tests’ in the box below.

**Lung function tests**

A spirometer is a device for measuring lung function. There are various makes of spirometer but they all have a mouthpiece into which the patient breathes ([http://patient.info/health/spirometry-leaflet](http://patient.info/health/spirometry-leaflet)). The patient is asked to blow into the spirometer as hard and as fast as possible. A nose clip may be used to prevent air escaping through the nose.

The most important measurements for obstructive pulmonary disease are: forced expiratory volume in one second (FEV$_1$) – the maximum volume of air the subject can blow out within one second, and forced vital capacity (FVC) – the total volume of air the subject can blow out in one breath. It is of note that these measurements are taken after administration of a bronchodilator medication to ease airflow and give the optimum result (NICE, 2018b).

With COPD or other obstructive disease, FEV$_1$ is reduced due to airway obstruction. FVC on the other hand may be relatively normal, indicating that lung volume has not changed significantly. Restrictive diseases also produce a lowered FEV$_1$. However, the FVC is reduced as well indicating significant loss of lung volume. Due to this, the ratio FEV$_1$/FVC is important for diagnostic purposes.

Figure 9.4 shows a spirometry tracing from a normal healthy individual, an individual with an obstructive disease and one with a restrictive disease.

Those with COPD have an FEV$_1$ less than or equal to 80% predicted (for age, height, sex) and FEV$_1$/FVC is less than 0.7 (NICE 2018b). The following table categorises COPD from mild to very severe according to the percentage of predicted FEV$_1$. The degree of severity has implications for treatment and management.

**Categorisation of COPD severity**

<table>
<thead>
<tr>
<th>Airflow obstruction</th>
<th>% of predicted FEV$_1$ (NICE 2018b)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>≥ 80%</td>
</tr>
<tr>
<td>Moderate</td>
<td>50–79%</td>
</tr>
<tr>
<td>Severe</td>
<td>30–49%</td>
</tr>
<tr>
<td>Very severe</td>
<td>&lt; 30%</td>
</tr>
</tbody>
</table>
Pathogenesis

COPD is a diffuse inflammatory disease of the lung tissue and airways. ‘Diffuse’ means spread throughout the lungs. COPD has two elements: emphysema and chronic bronchitis. Most cases involve a combination of the two. Inflammation in the lungs, particularly the small airways, is part of the normal immune response to smoking. However, in those with COPD the response to inhaled smoke or other toxins is magnified and causes damage to the lung tissue. Chronic bronchitis can result, which is inflammation of the bronchial tubes and is characterised by a productive cough. There is increased mucus secretion from goblet cells lining the airways. There is in addition hypertrophy of the mucus glands within the wall of the airway. The increased mucus secretion causes the characteristic productive cough. Airway inflammation results in swelling and oedema and contributes to airway obstruction.

Emphysema is characterised by an enlargement of the airspaces beyond the terminal bronchioles. This is accompanied by destruction of the alveolar walls. There are different
forms of emphysema according to the location of the alveolar destruction (Kumar et al., 2014). The one associated with smoking, centrilobular emphysema, causes damage to the respiratory bronchioles (Figure 9.5). This leads to the development of air spaces (or bullae) in the lungs. This reduces the total surface area available for gas exchange and may lead to reduced oxygenation of blood and reduced removal of carbon dioxide.

Current theory proposes that harmful chemical imbalances develop in the lungs of those with COPD: a protease/anti-protease imbalance; and an oxidant/anti-oxidant imbalance (Figure 9.6). These lead to the eventual tissue destruction characteristic of emphysema. Cigarette smoke and other environmental irritants activate the epithelial cells lining the airways and macrophages in the lungs to release chemotactic factors (Chapter 2). These factors attract CD8 lymphocytes and neutrophils from the circulation. Neutrophils and macrophages produce proteases. These are enzymes that break down proteins, particularly the elastic and collagen fibres of lung tissue. In normal lungs there is a significant presence of anti-protease enzymes, especially α₁-antitrypsin. The anti-proteases normally act to balance the proteases and limit their destructive effects. In emphysema, the balance is tipped in favour of the proteases partly because of the infiltration of a large number of neutrophils.

![Figure 9.5](image-url)  
*Figure 9.5* Damage to the alveoli shown in emphysema. (A) Shows the normal structure of the respiratory bronchiole and alveoli. (B) Centrilobular damage to the wall of respiratory bronchioles causing enlargement of the airspace.
At the same time oxidants from smoke and reactive oxygen species released from inflammatory cells act to inactivate anti-proteases. This further contributes to tipping the balance in favour of the proteases. The excess of oxidants represents another imbalance. The lungs of healthy individuals normally contain antioxidants that minimise oxidative damage. Tobacco smoke contains a range of reactive oxygen species which outweigh the antioxidants. Activated neutrophils also release reactive oxygen species into the alveoli.

Pathophysiology

Airflow obstruction results from the inflammation and narrowing of the airways and the presence of inflammatory exudates. The loss of elastic tissue characteristic of emphysema causes the airways to narrow and collapse. The elastic tissue of the lungs normally provides a traction (‘pull’) force that keeps the airways open. In addition, following inhalation, elastic recoil of the lungs normally drives air out of the lungs. In emphysema elastic recoil is reduced. This makes the lung easier to inflate (i.e. increases lung compliance) but makes breathing out more difficult.

Summary of events leading to alveolar wall destruction

Cigarette smoke and other environmental irritants activate the epithelial cells lining the airways and macrophages in the lungs to release chemotactic factors. These factors attract CD8 lymphocytes and neutrophils from the circulation which produce proteases. In normal lungs, anti-proteases normally act to balance the proteases and limit their destructive effects. In emphysema, the balance is tipped in favour of the proteases. Oxidants from smoke and reactive oxygen species released from inflammatory cells act to inactivate anti-proteases. This further contributes to tipping the balance in favour of the proteases.

Figure 9.6  Pathogenesis of emphysema
During exhalation, airway obstruction traps air in the lungs. Airway obstruction and the loss of elastic tissue result in airway closure during exhalation. Air which should have been exhaled remains in the lungs. This results in hyperinflation of the lungs in which a larger than normal volume of air remains in the lungs following exhalation. Hyperinflation in turn reduces the volume of air that can be inspired. This can cause marked breathlessness in COPD, especially during exertion.

Airway obstruction and collapse can lead to arterial hypoxemia (low arterial blood oxygen levels) in advanced disease. Hypoxemia can present with or without hypercapnia (increased levels of carbon dioxide in the blood). Some patients are able to maintain blood oxygen levels by increasing their respiratory effort. Other patients fail to maintain respiratory effort and develop hypercapnia. This raised carbon dioxide (and associated respiratory acidosis) normally stimulates breathing rate. However, over time, patients with hypercapnia develop insensitivity to raised carbon dioxide levels in the blood. Low oxygen levels become the main stimulus for breathing. Low oxygen levels in the blood in turn stimulate red blood cell production (polycythaemia) and fluid retention. Patients appear cyanosed and ‘bloated’ due to fluid retention. Oxygen must be administered carefully to patients with hypoxemia and carbon dioxide retention. The main stimulus to breathe in these patients is the low oxygen level. If administered with too much oxygen, their drive to breathe decreases. Carbon dioxide levels then worsen as their breathing rate diminishes.

In advanced disease, patients may develop pulmonary hypertension. Blood vessels supplying parts of the lung which remain unventilated automatically constrict. This causes increased resistance to blood flow and raises the pulmonary blood pressure. Pulmonary hypertension can eventually lead to enlargement of the right ventricle (cor pulmonale) due to the increased work needed to pump blood through the pulmonary system. This results in weakness of the right ventricle and right ventricular failure.

Patients may experience exacerbations in COPD often due to lung infections. Increased airway inflammation and reduced gas exchange can lead to severe respiratory failure and death.

Activity 9.1  Reflection

Over the past five years, Peter has experienced a number of exacerbations of his COPD and his lung function has declined. He has been admitted to the respiratory ward where you work as a nurse. He is very breathless and wheezing. This causes great anxiety for him. How would you help to alleviate his anxiety?

A suggested answer is given at the end of the chapter.
Pharmacological treatment of asthma

Scenario

Rachel has recovered well from her asthma attack. Today she visits you in the nurse-led asthma clinic at the surgery. She tells you she is on a blue relieving inhaler and a brown inhaler for prevention. She uses the blue one only when it is needed but she uses the brown one every day. Her FEV₁ is 90% of that predicted. She has not had an asthma attack since her brown inhaler was increased three months previously. She also tells you that she generally has no ‘wheeziness’ or cough in the day or at night. You are happy with her progress and continue the treatment.

The scenario shows that Rachel’s asthma is currently well controlled. Pharmacological treatments are the main treatment type for asthma. The blue ‘relieving’ inhaler mentioned by Rachel is a salbutamol inhaler. Salbutamol is a type of β₂-agonist. The brown ‘preventative’ inhaler is a corticosteroid inhaler. We will look at the differences in how these inhalers act to explain both their uses in asthma and their side-effects.

β₂-agonists

In the section on pathogenesis of asthma, we introduced the action of the two divisions of the autonomic nervous system: sympathetic and parasympathetic, which regulate the diameter of the airways. Most organs of the body receive a nerve supply from both divisions. As is the case with the airways, the two divisions usually work in opposite ways to regulate the organs they supply.

To understand how β₂-agonists work in asthma, and what their potential side-effects are, we need to examine the neurotransmitters released from the sympathetic and parasympathetic nerve endings. We also need to know the names of the receptors for these neurotransmitters and how they are distributed in the various body organs.

Sympathetic fibres release neurotransmitters adrenaline and noradrenaline. Adrenaline and noradrenaline are adrenergic receptor agonists (Chapter 1). There are two main types of adrenergic receptors: alpha (α) and beta (β). These are further divided into subtypes – α₁, α₂, β₁, β₂ – based on the response they produce and according to which drugs bind to them. By contrast, parasympathetic fibres release the neurotransmitter acetylcholine. Acetylcholine is a muscarinic receptor agonist. Table 9.2 summarises the effects of these neurotransmitters on various organs and tissues of the body.
Chapter 9

<table>
<thead>
<tr>
<th>Target</th>
<th>Effect of sympathetic stimulation (adrenaline/noradrenaline) (type of adrenergic receptor is given in brackets)</th>
<th>Effect of parasympathetic stimulation (acetylcholine) of muscarinic receptors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lungs – bronchial smooth muscle</td>
<td>Relaxation causing bronchodilation ($\beta_2$)</td>
<td>Bronchoconstriction</td>
</tr>
<tr>
<td>Stomach and intestines – smooth muscle of wall</td>
<td>Decrease in motility and tone ($\alpha$ and $\beta_2$)</td>
<td>Increase in motility and tone</td>
</tr>
<tr>
<td>Heart – (cardiac) muscle</td>
<td>Increased rate and force of contraction ($\beta_1$)</td>
<td>Reduced rate and force of contraction</td>
</tr>
<tr>
<td>Arterioles – smooth muscle</td>
<td>Relaxation or contraction depending upon the organ: arterioles to kidney and gastrointestinal tract contract producing constriction and reduced blood flow; arterioles to skeletal muscle, heart, liver, adipose tissue relax producing dilation and increased blood flow ($\alpha$ and $\beta$)</td>
<td>No known effect</td>
</tr>
<tr>
<td>Liver</td>
<td>Synthesis and release of glucose (from glycogenolysis and gluconeogenesis) (Chapter 12) ($\alpha$ and $\beta_2$)</td>
<td>No known effect</td>
</tr>
<tr>
<td>Salivary glands</td>
<td>Little or no increase in saliva production</td>
<td>Increased secretion of a watery mucus</td>
</tr>
<tr>
<td>Eye – radial muscle of iris</td>
<td>Contraction leading to pupil dilation ($\alpha_1$)</td>
<td>No known effect</td>
</tr>
<tr>
<td>Eye – circular muscle of iris</td>
<td>No known effect</td>
<td>Contraction leading to pupil constriction</td>
</tr>
</tbody>
</table>

Table 9.2  Effects of sympathetic and parasympathetic stimulation of various body organs and tissues

Side-effects and considerations for practice

$\beta_2$-agonists used in asthma, such as salbutamol and terbutaline, work directly to stimulate $\beta_2$ receptors on the smooth muscle of the airways causing bronchodilation. This reverses the bronchoconstriction seen in asthma. However, these drugs are often not completely specific for the lung or one type of $\beta$ receptor. For example, salbutamol also activates cardiac muscle $\beta_1$ receptors to some degree which can lead to the side-effect of tachycardia.

Activity 9.2  Critical thinking

According to the World Anti-Doping Agency (2015), high doses of salbutamol are banned in sport. Use Table 9.2 to consider what unfair advantages salbutamol might provide for an athlete. Bear in mind that salbutamol activates both $\beta_2$ and $\beta_1$ receptors.

A suggested answer is given at the end of the chapter.
Activity 9.2 shows how stimulating the sympathetic nervous system with salbutamol can enable us to predict its actions and side-effects. It helps to explain why somebody who overuses their salbutamol inhaler might describe symptoms such as a ‘racing heart’. Another important side-effect, unrelated to the effects on the nervous system, is hypokalaemia (low blood potassium). This is more commonly seen with higher doses from nebulised solutions. β₂-agonists must therefore be used with care with other medications, such as diuretics, which also cause hypokalaemia.

Using β₂-agonists with β-blockers (Chapter 8) can cause problems. β-blockers are β₂-antagonists and therefore block the action of β₂-agonists. β-blockers are contraindicated in asthma as they may actually precipitate asthma attacks. NSAIDs are also contraindicated in asthma (Chapter 6).

Salbutamol and terbutaline are short-acting β₂-agonists (SABAs) with a relatively short duration of action (salbutamol 4–6 hours). As a consequence they may need to be used up to four times a day to relieve symptoms. Long-acting β₂-agonists (LABAs) include salmeterol and formoterol. These take longer to work but have a longer duration of action (salmeterol 12 hours). They are able to relieve symptoms for much longer and are inhaled twice a day. Short-acting β₂-agonists are used for immediate relief of symptoms. Long-acting β₂-agonists are used as prophylactic treatments to prevent the development of symptoms. Their side-effects and drug interactions are similar to those described for short-acting β₂-agonists.

Corticosteroids

Corticosteroids have anti-inflammatory and immunosuppressant effects (Chapter 2). Corticosteroids are used in asthma to reduce inflammation, and have been shown to reduce the number of eosinophils in the circulation. This makes corticosteroids useful in asthma prophylaxis. They are not useful for symptoms of an acute asthma attack, where short-acting β₂-agonists should be used. Examples of corticosteroids used in inhalers are: beclomethasone, budesonide and fluticasone. There are many different inhaler types and strengths available. The current UK asthma guidance classifies the different strengths as very low (children’s dose), low (usual adult starting dose), medium and high. The guidance contains a useful table outlining these different strengths (BTS/SIGN, 2016).

Activity 9.3 Critical thinking

You are a nurse on the ward. One of the junior doctors has just written up a drug chart for John who has been newly admitted. You notice that on the drug chart is written ‘Beclometasone inhaler two puffs twice a day’. What other information would you need on the drug chart so that you can order it from the pharmacy?

A suggested answer is given at the end of the chapter.
Activity 9.3 shows the importance of being aware of different inhaler types, brands and strengths. Some inhalers also contain a mixture of drugs, such as a corticosteroid and a $\beta_2$-agonist. It is also very important that patients are using their inhalers correctly. Hickey (2014), listed in Further reading at the end of the chapter, gives advice on inhaler technique.

Side-effects and considerations for practice

You learned about the adverse side-effects of corticosteroids in Chapter 2. Steroid inhalers are used in asthma where possible, rather than oral medicines. This is to minimise absorption of the corticosteroid into the systemic circulation. This helps to reduce side-effects, although it does not completely eliminate them as some of the corticosteroid will be absorbed. The amount absorbed will increase as the dose of inhaler increases. High doses can cause adrenal suppression and, if a patient is on a high inhaled dose, a steroid card may be needed. Throat irritation and oral candidiasis can be a particular problem with corticosteroid inhalers. This is especially true if inhaler technique is poor as more corticosteroid is deposited in the mouth rather than the lungs. Rinsing the mouth after use or spacer devices can help.

Leukotriene receptor antagonists

Leukotrienes are inflammatory mediators released by mast cells, eosinophils and basophils during an inflammatory reaction (Chapter 2). In asthma, leukotrienes contribute to bronchospasm by directly stimulating contraction of the smooth muscle in the airways.

Blocking leukotrienes with leukotriene receptor antagonists can improve asthma symptoms and reduce exacerbations. They have an additive effect when used with corticosteroids. Examples include montelukast and zafirlukast. They are taken by mouth and are generally well tolerated. Side-effects include abdominal pain, headache, dizziness and sleep disturbance.

Xanthines

This group of oral medications includes theophylline and aminophylline which are related to caffeine. Aminophylline is a mixture of theophylline and ethylenediamine. They are usually given as slow release preparations, for example Phyllocontin Continus®, which is a slow release aminophylline preparation. They are only used if asthma control is poor despite inhaler therapy. Intravenous aminophylline preparations can also be used during severe asthma attacks.

The precise mechanism of action is unclear but they cause bronchodilation by relaxing smooth muscle in the bronchioles. They may also have some anti-inflammatory actions (Barnes, 2005).
Side-effects and considerations for practice

Xanthines stimulate the central nervous system and can cause tremor, nervousness and poor sleep as a result. They also stimulate the heart, causing tachycardia and palpitations, so must be used with caution in patients with cardiac arrhythmias or severe hypertension.

Aminophylline and theophylline have a narrow therapeutic range (Chapter 1). The concentrations in the blood must be carefully monitored. If the level in the blood is too high serious central nervous system and cardiovascular toxicity can result.

Activity 9.4  Research

In Appendix 1 of the BNF there is a section on drug interactions. Look up theophylline and note down some of the common drugs theophylline interacts with. How does the number of drug interactions compare with a drug like paracetamol?

A suggested answer is given at the end of the chapter.

The activity demonstrates that xanthines interact with many different medications. Xanthines are metabolised by cytochrome P450 enzymes in the liver (Chapter 1). Other drugs affecting cytochrome P450 enzymes will alter the rate of metabolism of xanthines. This is problematic because xanthines have a narrow therapeutic range. The interacting drugs cause a change in plasma level which can move the levels of xanthine out of its narrow therapeutic range. For example a cigarette smoker will need a higher dose of a drug like aminophylline because the cigarette smoke increases P450 enzyme action. The antibiotic erythromycin inhibits cytochrome P450 enzymes and will increase blood levels of xanthine.

Choosing drugs to treat asthma

As we have seen there are many drugs used in asthma to both prevent and relieve symptoms. Choosing which ones a patient needs depends on the severity of asthma. A stepwise approach is used (Joint British Thoracic Society/Scottish Intercollegiate Guidelines Network (SIGN) guidance, 2016). This is illustrated in Figure 9.7.
### Activity 9.5 Critical thinking

A more detailed look at Rachel’s inhalers shows us that she takes a salbutamol 100 microgram inhaler two puffs four times a day if needed. She is also on a Clenil 100 microgram aerosol inhaler and she takes this regularly at a dose of two puffs twice a day. What step is Rachel on in the asthma guideline? If her symptoms got worse what might be considered next in her treatment? What might happen if her symptoms continued to be well controlled?

* A suggested answer is given at the end of the chapter.

The activity shows how the stepwise approach to treatment is applied to individuals to improve asthma control with the aim of achieving complete control. Asthma attacks can be fatal. For some people achieving control is very difficult. We can see from the table that these people are likely to be taking more medicines and may even be on oral corticosteroid tablets which can cause many side-effects.

### Acute asthma attacks

Acute asthma attacks can be life-threatening and the patient may need admission to hospital. Hospital treatment would consist of a nebulised short-acting $\beta_2$-agonist (usually salbutamol), oxygen and an oral or IV corticosteroid. The short-acting antimuscarinic agent ipratropium might also be added as this will act together with the salbutamol to further improve dilation of the airways. Nebulisers deliver larger doses
Respiratory diseases

to the lungs than inhalers can. If you consider a salbutamol inhaler, the dose from one puff is 100 micrograms. The dose of a salbutamol nebule is 2.5 mg or 5 mg which is 25–50 times more. In the same way the corticosteroid given orally or IV will deliver a higher dose to the lungs than an inhaler can. Asthma attacks can occur if a person is not using their inhalers properly. Some people have poor inhaler technique. Others may be underusing their ‘preventer’ inhalers (corticosteroids) and overusing their ‘reliever’ inhalers (β₂-agonists). Nurses have a vital role to play in educating patients about the different types of inhalers, what they are for and how to use them.

Pharmacological management of COPD

Many of the inhalers used to treat COPD are the same as those used to treat asthma, but as we shall see, there are important differences. It should be remembered that the pharmacological treatments help to improve symptoms and reduce exacerbations but they do not prevent progression of COPD.

Bronchodilators

Irreversible airway obstruction occurs in COPD. Bronchodilators improve breathlessness and reduce hyperventilation. The use of bronchodilators will not return the airways to normal and may not improve the FEV₁ greatly. However the quality of life of many patients is often improved with these drugs.

Short-acting β₂-agonist (SABA) and long-acting β₂-agonist (LABA)

These bronchodilating drugs have already been introduced under asthma. Drugs such as salbutamol (a short-acting β₂-agonist) and salmeterol (a long-acting β₂-agonist) are used in COPD to relax smooth muscle in the lungs and cause bronchodilation.

Short-acting muscarinic antagonists (SAMA) and long-acting muscarinic antagonists (LAMA)

If we revisit Table 9.2 we can see that blocking (or antagonising) muscarinic receptors is another way of causing bronchodilation. This is how drugs such as ipratropium (a short-acting muscarinic antagonist) and tiotropium (a long-acting muscarinic antagonist) work. These drugs block the effect of acetylcholine on muscarinic receptors leading to bronchodilation. Theoretically these drugs could be used in the routine treatment of asthma but in practice they are less effective than β₂-agonists (Rodrigo et al., 2005). Muscarinic antagonists are sometimes used in acute asthma where other drugs have failed. They are, however, used very often for COPD, often in addition to β₂-agonists.
Side-effects and considerations for practice

Side-effects can be predicted from the pharmacology (see Table 9.2) and include dry mouth and constipation. Contact with the eyes, for example when nebulised solutions are used, can cause blurred vision and glaucoma.

Xanthines

Oral aminophylline or theophylline are also used in COPD that has not responded to other treatments. They are used for their bronchodilating effects in stable COPD. They are used for exacerbations only if nebulised bronchodilators have not worked (NICE, 2018b). They are not routinely used as they have a narrow therapeutic range so monitoring can be complicated. Also not all studies show they are effective for exacerbations.

Inhaled corticosteroids (ICS)

Inhaled corticosteroids can be used in the treatment of COPD but they are much less effective than in the treatment of asthma. Although COPD is also an inflammatory condition the pathophysiology is different. Neutrophils are implicated in COPD and these inflammatory cells are relatively insensitive to corticosteroids, unlike in asthma where eosinophils are implicated which are sensitive to corticosteroids. However many patients with COPD are prescribed corticosteroids inhalers and do appear to benefit from them (NICE, 2018b). They should not be used alone but in combination with a bronchodilator in a combination inhaler. An example of such an inhaler is Symbicort which contains budesonide (a corticosteroid) and formoterol (a LABA). They must be used with care as they cause a greater incidence of pneumonia.

Oxygen

As COPD progresses patients can become hypoxaemic (have low blood oxygen levels). Oxygen therapy can help to increase exercise capacity and provide relief of breathlessness. It can also be used during exacerbations of COPD. It should be started by a specialist as there are many risks that need to be considered. A full discussion is outside the scope of the book. Further information can be found in the full NICE guidelines (2018b).

Stepwise COPD treatment

The NICE guidelines (2018b) for COPD set out a stepwise approach to the treatment of COPD (see Further reading at the end of the chapter). Initial treatment choice would be a short-acting β₂-agonist (SABA) such as salbutamol or a short-acting muscarinic antagonist (SAMA) such as ipratropium. The next scenario helps to illustrate how such guidelines are used in practice.
Scenario

John is a 63-year-old ex-smoker with a diagnosis of COPD. He comes to see you at your nurse-led clinic. He is currently on an ipratropium inhaler and he takes two puffs four times a day. You check his FEV$_1$ and FVC and ask him questions about how his breathlessness affects his activities. You also check his inhaler technique. He tells you that he is currently feeling more breathless, especially when walking fast or uphill. You decide to move John onto the next stage of therapy. The next stage of treatment depends on whether a patient also has features of asthma, which means that their condition might be responsive to inhaled corticosteroids. You know that this is the case for John. You therefore suggest that his current inhaler is stopped and that Symbicort, a combination inhaler containing a LABA plus an ICS, is started. You ensure that John knows how to use his new inhaler before he leaves.

Acute exacerbations of COPD

Depending on the severity of symptoms, people with exacerbations of COPD may be treated at home or in hospital. The treatment for exacerbations include increasing the dose of short acting bronchodilators. This might be achieved by increasing the dose given by inhaler. In some cases nebulised ipratropium and salbutamol are used. This allows much larger doses to be given but requires specialised nebulising equipment. Short courses of systemic corticosteroid, usually prednisolone, are also used. The prednisolone would usually be prescribed for 7–14 days only, to minimise long-term side-effects of corticosteroids. An antibiotic would also be needed if a bacterial infection was suspected (NICE, 2018b).

It is now time to review what you have learned within this chapter by undertaking some multiple choice questions.

Activity 9.6 Multiple choice questions

1. ‘Shortness of breath’ is called:
   a) Asthma
   b) Dyspnoea
   c) Tachycardia
   d) Hypoxaemia

2. In a patient with COPD which clinical feature is least likely to be present?
   a) A history of smoking
   b) A slowly progressive disease

(Continued)
(Continued)

c) Airway obstruction that is reversible
d) Airway inflammation

3. Which of the following white blood cells is characteristic of the chronic inflammation in asthma?
   a) Eosinophil
   b) Neutrophil
   c) Monocyte
   d) Lymphocyte

4. Increased stimulation of sympathetic fibres to the airways brings about:
   a) Mucus secretion
   b) Bronchodilation
   c) Bronchoconstriction
   d) Swelling and oedema

5. According to NICE (2018b) COPD affects approximately how many people in the UK?
   a) 10.1 million
   b) 5.7 million
   c) 3.3 million
   d) 1.2 million

6. Corticosteroids should be delivered by which route in mild to moderate exacerbations of COPD?
   a) Inhaled via a dry powdered inhaler
   b) Nebulised
   c) Oral
   d) Intravenous

7. Which one of the following inhaled medicines is used to help prevent asthma attacks?
   a) Salbutamol
   b) Beclomethasone
   c) Ipratropium
   d) Salmeterol

8. Which one of the following inhaled medicines is used to help alleviate the symptoms of an acute asthma attack?
   a) Salbutamol
   b) Beclomethasone
   c) Ipratropium
   d) Salmeterol
9. Tiotropium is an inhaled medication used in maintenance treatment of COPD. It is an example of a:
   a) Short-acting $\beta_2$-agonist (SABA)
   b) Long-acting $\beta_2$-agonist (LABA)
   c) Short-acting muscarinic antagonist (SAMA)
   d) Long-acting muscarinic antagonist (LAMA)

10. Inhaled salbutamol can cause tachycardia as a side-effect, especially in higher doses. This occurs because:
   a) $\beta_2$ receptors on the heart muscle are blocked by salbutamol
   b) $\beta_2$ receptors on the heart muscle are stimulated by salbutamol
   c) Muscarinic receptors on the heart muscle are blocked by salbutamol
   d) Muscarinic receptors on the heart muscle are stimulated by salbutamol

Chapter summary

Asthma and COPD are obstructive pulmonary disorders. Asthma is a condition characterised by narrowing of the airways, hyper-responsiveness of the airways to triggers leading to bronchospasm and chronic inflammation. Airflow obstruction is reversible. COPD often develops later in life, is progressive and airflow obstruction is irreversible. Inflammation leads to damage of lung tissue, the development of air spaces and a loss of elasticity. Similar inhaled drugs are used in the treatment of both COPD and asthma. The smooth muscle in the walls of bronchi and bronchioles is controlled by the autonomic nervous system. Many drugs used in COPD and asthma cause bronchodilation by affecting the autonomic nervous system. $\beta_2$-agonists such as salbutamol act on $\beta_2$ receptors in the bronchi which leads to relaxation of smooth muscle. Muscarinic antagonists, such as ipratropium, block acetylcholine receptors to relax the smooth muscle. The xanthines, aminophylline and theophylline, are also bronchodilators used in the treatment of COPD and asthma. They are used less often as they have a narrow therapeutic range and are involved in many drug interactions. Corticosteroid inhalers are also used. They reduce inflammation in the lungs and are especially effective in asthma. They are less effective in COPD. Other drugs such as leukotriene receptor antagonists can also help reduce inflammation in asthma. Nurses have a vital role to play in educating patients, especially with so many different inhaler types.
Chapter 9

Activities: Brief outline answers

Activity 9.1 Reflection (p246)

It is important that healthcare professionals acknowledge the effect that COPD may have on patients like Peter, and their psychosocial well-being. Patients with chronic respiratory diseases are often at high risk of developing symptoms such as anxiety and depression. Psychological symptoms may result from a patient’s fear, which can be triggered by increasing breathlessness, anxiety over risk of acute exacerbations or concerns about lack of prognostic certainty.

Nursing strategies for managing breathlessness should adopt an integrated approach that does not separate psychological and physical aspects of breathlessness. Therapeutic interventions for rehabilitation and supportive care may focus on helping patients (and their carers) to (1) increase their fitness and tolerance of restricted lung function and reducing functional disability by recognising/reducing triggers to breathlessness and managing their breathlessness; (2) manage their anxiety during an episode of breathlessness through, for example, breathing retraining techniques; and (3) acknowledging the meaning of breathlessness in the context of their life-limiting condition. It is therefore important that, alongside pharmacological interventions for breathlessness, health promotion advice including breathing control, activity pacing, relaxation techniques and information about their condition, as well as emotional support, is provided to patients and their careers.

Activity 9.2 Critical thinking (p248)

Salbutamol causes vasodilation and greater air passage in the lungs. This will increase gas exchange and lead to greater oxygen levels in the blood oxygen in the body. This will enable greater aerobic respiration, for example in active skeletal muscle of an athlete. Blood glucose levels are also increased as salbutamol increases glucose release from the liver. From the table we can also see that salbutamol increases heart rate and force and causes vasodilation. This would all potentially increase blood flow to skeletal muscles. All these factors could unfairly enhance sporting performance.

Activity 9.3 Critical thinking (p249)

You would need to know the type of inhaler the patient is taking. Both dry powder and aerosol inhalers exist. In this case, the brand of inhaler is also important. Although it is often best practice for doctors to prescribe generically, sometimes this isn’t the case. Clenil Modulite® and Qvar® are two different brands of beclomethasone aerosol inhaler. Qvar® has extra fine particles and is twice as potent as Clenil Modulite® (BNF). If the brand is wrong you could overdose or underdose the patient. The type of inhaler device is also important. Qvar® comes as a standard aerosol inhaler, an autohaler or a Salamol Easi-breathe inhaler. These other types can be useful for people who find it difficult to use inhalers correctly. It is also vital to know the strength of the inhaler. Clenil Modulite® for example comes in strengths of 50 micrograms, 100 micrograms, 200 micrograms and 250 micrograms per metered dose inhalation (per puff).

Activity 9.4 Research (p251)

Examples include erythromycin, ciprofloxacin, antiepileptic drugs, calcium channel blockers and anti-fungals. Xanthines have a much longer list of drug interactions than that for many other drugs, for example paracetamol. It is therefore especially important to check for interactions if a patient is on aminophylline or theophylline.

Activity 9.5 Critical thinking (p252)

Rachel is currently on the regular preventer step of the asthma guidance. She is on a low dose of steroid inhaler. The next step would involve adding a long-acting beta agonist (LABA) such as salmeterol to her treatment. Combination inhalers are often used for this to ensure patients take the corticosteroid and the LABA. If a combination inhaler was used the corticosteroid
would need to be changed as beclometasone isn’t available in a combination inhaler. She could be started on a Seretide inhaler which contains fluticasone (corticosterooid) and salmeterol (LABA).

**Activity 9.6 Multiple choice questions (pp255–7)**

1. ‘Shortness of breath’ is called:
   - b) Dyspnoea

2. In a patient with COPD which clinical feature is *least* likely to be present?
   - c) Airway obstruction that is reversible

3. Which of the following white blood cells is characteristic of the *chronic inflammation* in asthma?
   - a) Eosinophil

4. Increased stimulation of sympathetic fibres to the airways brings about:
   - b) Bronchodilation

5. According to NICE (2018b) COPD affects approximately how many people in the UK?
   - d) 1.2 million

6. Corticosteroids should be delivered by which route in mild to moderate exacerbations of COPD?
   - c) Oral

7. Which one of the following inhaled medicines is used to help prevent asthma attacks?
   - b) Beclomethasone

8. Which one of the following inhaled medicines is used to help alleviate the symptoms of an acute asthma attack?
   - a) Salbutamol

9. Tiotropium is an inhaled medication used in maintenance treatment of COPD. It is an example of a:
   - d) Long-acting muscarinic antagonist (LAMA)

10. Inhaled salbutamol can cause tachycardia as a side-effect, especially in higher doses. This occurs because:
    - b) $\beta_2$ receptors on the heart muscle are stimulated by salbutamol

**Further reading**


This gives more information on the treatment of asthma including algorithms used to choose therapy.


This article gives useful information on inhaler technique.


A comprehensive textbook on pathology. Chapter 13 gives a detailed examination of the pathology of asthma and COPD.
Chapter 9

NICE (2018b) NG114. Chronic Obstructive Pulmonary Disease in Over 16’s: Diagnosis and Management. Available at: www.nice.org.uk/guidance/ng115/
This gives more information about treatment and diagnosis of COPD.

Useful websites

www.brit-thoracic.org.uk/
British Thoracic Society website. Provides information on asthma, COPD and other lung conditions.

www.asthma.org.uk/
A charitable organisation providing information and support for those with asthma.
Chapter 3
The patient who needs respiratory support

Desiree Tait

NMC Standards for Pre-registration Nursing Education

This chapter will address the following competencies:

Domain 3: Nursing practice and decision-making

Generic competencies:

3. All nurses must carry out comprehensive, systematic nursing assessments that take account of relevant physical, social, cultural, psychological, spiritual, genetic and environmental factors, in partnership with service users and others through interaction, observation and measurement.

4. All nurses must ascertain and respond to the physical, social and psychological needs of people, groups and communities. They must then plan, deliver and evaluate safe, competent, person-centred care in partnership with them, paying special attention to changing health needs during different life stages, including progressive illness and death, loss and bereavement.

Field-specific competencies:

3.1. Adult nurses must safely use a range of diagnostic skills, employing appropriate technology, to assess the needs of service users.

4.1. Adult nurses must safely use invasive and non-invasive procedures, medical devices, and current technological and pharmacological interventions, where relevant, in medical and surgical nursing practice, providing information and taking account of individual needs and preferences.

NMC Essential Skills Clusters

This chapter will address the following ESCs:

Cluster: Care, compassion and communication

3. People can trust the newly registered graduate nurse to respect them as individuals and strive to help them preserve their dignity at all times.

By entry to the register:

v. Is proactive in promoting and maintaining dignity.
Chapter aims

By the end of this chapter, you should be able to:

- identify why patients may need advanced respiratory (ventilatory) support;
- demonstrate an awareness of the importance of arterial blood gas analysis in the management of patients with respiratory failure;
- describe non-invasive ventilation (NIV) and mechanical invasive ventilation (MIV);
- demonstrate an awareness of the factors influencing the choice of appropriate respiratory support;
- describe the fundamentals of providing a safe holistic approach to caring for patients receiving NIV and MIV.

Introduction

In this chapter you are introduced to Mrs Jenny Matthews. She is 43 years old and for 20 years has had a history of acute exacerbation of asthma. She had eczema as a child and hay fever but wasn’t diagnosed with asthma until she was in her twenties. We will follow her on her journey through healthcare as she experiences an acute exacerbation of asthma. The scenario box below provides a summary of her admission to the emergency department.

Scenario: Jenny Matthews

Situation

Mrs Jenny Matthews, age 43 years.
Admitted to the emergency department with a seven-day history of shortness of breath and productive cough. She had previously been seen by her GP and treated with a combination of broad spectrum antibiotics and an increased dose of salbutamol. She was progressing well at home but a sudden onset of increased shortness of breath at 2 a.m. on the morning of the eighth day prompted a 999 call for help from her husband.

Background

History of acute asthma for 20 years. Jenny has been admitted to hospital on five occasions during the last seven years. On the last occasion she required emergency intubation and ventilation, staying in intensive care for 48 hours.
She did smoke 30 cigarettes a day for 22 years but has reduced this to ten a day. She has a strong family history of reactive airways disease.

Assessment in the emergency room

Airway (A): Patient is agitated and struggling to breathe.
Breathing (B): Dyspnoea, with use of her accessory muscles.
Unable to complete a full sentence when responding to questions.

(Continued)
Chest auscultation indicates an expiratory wheeze and bilateral crackles.

Respiration (R): 36 bpm.

SpO₂ 91%.

Blood gases showed:

- PH: 7.42
- PaO₂: 8.7 kPa
- PaCO₂: 3.6 kPa
- HCO₃⁻: 24 mmol/L

Peak expiratory flow rate (PEF): 120 ml/min (Jenny’s normal PEF: 300 ml/min).

Circulation (C): HR: 125/min.

BP: 125/72 mmHg.

Disability (D): Blood glucose: 6.8 mmol/L.

Alert and agitated.

Exposure (E): Temp: 37.4°C.

**Recommendation**

- Humidified high-flow oxygen 60%.
- Salbutamol 5 mg nebulisers continuously until improvement in PEF.
- Hydrocortisone 100 mg IV.
- Aminophylline infusion.
- IV antibiotics.
- Plan to transfer to high dependency for monitoring and evaluation of her treatment.

Jenny’s husband, Brendan, has arrived and when he is informed that she needs to be admitted to the high dependency unit he becomes very angry and starts shouting at his wife ‘I told you this would happen, I’ve had enough of this! Why didn’t you just give up smoking! I’m going home, you’re on your own now!’ Jenny seems not to be listening and Brendan Matthews is asked to leave. The staff invite him to stay in a quiet room and approach him for more information. At this point he refuses to stay and instead gives the GP’s address and Jenny’s parents’ phone number saying that he has had enough and he’s leaving her.

**Why did Jenny’s situation deteriorate at home and lead to an emergency admission to HDU?**

Less than 24 hours ago Jenny was seemingly making a good recovery from a chest infection when she experienced a sudden deterioration in her condition. Asthma is a chronic inflammatory disorder of the mucosal lining of the bronchi which is associated with bronchial hyper-responsiveness, reversible airway constriction and variable airflow obstruction (McCance and Huether, 2014). Factors involved in triggering an acute asthma attack can include:
• evidence of a family history of asthma;
• exposure to an allergen;
• urban residence;
• air pollution;
• tobacco smoke;
• recurrent respiratory tract infections;
• psychological factors and anxiety.

In Jenny’s case, after seven days of antibiotic treatment, she decided it was time to resume smoking, as this was her main way of coping with the stresses of life. This triggered a long and aggressive argument with her husband that continued over the course of the evening. It was later that night that Jenny developed the acute exacerbation of asthma reported in her story. According to Polosa and Thompson (2013) cigarette smoking in asthma is associated with a higher frequency and severity of exacerbations and a higher risk of mortality than non-smokers. There is also evidence to suggest that anxiety and depression is often associated with smoking and an increased frequency of exacerbations for patients with asthma (Leader et al., 2014). For Jenny, the combination of tobacco smoke, stress, a recent respiratory tract infection and her family history combined to trigger the asthma attack.

When Jenny was admitted to the emergency room she was presenting signs and symptoms of the ‘early response’ phase. According to McCance and Huether (2014) this phase is initiated by exposure to the inhaled irritant and triggers a cascade of inflammatory events that lead to acute and chronic airway dysfunction. The combined impact of mast cell activation releasing vasoactive mediators with degranulation of their inflammatory mediators and an immune activation, leads to:

• vasodilation and increased capillary permeability;
• vascular congestion;
• bronchospasm;
• increased contractile response of the bronchial smooth muscle;
• mucus secretion;
• thickening of airway walls.

This cascade of events leads to bronchial hyper-responsiveness and airway obstruction.

For Jenny the presence of a PEF of less than 50% of her predicted normal range, a respiratory rate of 36 bpm, a pulse of 125 bpm and an inability to complete a sentence in one breath indicated the presence of acute severe asthma (BTS and SIGN, 2014). With oxygen saturations of 91% and signs of type 1 respiratory failure indicated by her arterial blood gas result of $\text{PaO}_2$ 8.7 kPa (see Chapter 2), Jenny’s condition was becoming life threatening and she required high dependency (level 2) care (BTS and SIGN, 2014).
Why are the arterial blood gas results significant?

In the body, acids (substances that release hydrogen ions (H⁺) in solution) are constantly being produced as by-products of normal cell metabolism. For example, the metabolism of proteins produces acids such as sulphuric acid and hydrochloric acid. During the metabolism of carbohydrates about 15,000 mmol of carbon dioxide (CO₂) is produced each day, and although it is not an acid, it is influential in maintaining pH balance.

Carbon dioxide is transported in the circulation in the following ways.

- 20% of CO₂ is attached to haemoglobin and carried as carbaminohaemoglobin (HHbCO₂).
- 10% is dissolved in the plasma as carbonic acid (H₂CO₃).
- 70% of CO₂ is carried as a bicarbonate base (a substance that uses up hydrogen ions). Carbonic acid in the presence of an enzyme called carbonic anhydrase is converted to bicarbonate ions (HCO₃⁻) and hydrogen ions (H⁺).

The relationship between carbonic acid and bicarbonate is a very important factor in how the body regulates the pH (the calculated acidity of the blood) in the circulation, as well as other buffer systems such as the kidney and renal excretion of hydrogen ions. In order to maintain a pH of 7.4 (normal blood pH) the ratio between carbonic acid and bicarbonate should stay at one part carbonic acid to 20 parts bicarbonate (1 H₂CO₃: 20 HCO₃⁻ + H⁺). This means that if the amount of HCO₃⁻ in the blood falls so must the amount of H₂CO₃ in order to maintain a ratio of 1:20. The body achieves this by increasing the rate and depth of respiration so that more CO₂ is eliminated through respiration and the ratio is maintained. This is called respiratory compensation and can be seen in patients who are producing an excess of metabolic acids such as lactic acid in shock (Chapters 6 and 7) and ketone acid in diabetic ketoacidosis (Chapters 8 and 12).

Left in the circulation, an imbalance in acids or bases would destroy cells and organs, so it is imperative the body has ways to maintain a pH balance at a pH value of between 7.35 and 7.45 in order to maintain normal cell function (Hall, 2011). Should the pH value fall above or below this range, the impact on the body can be critical and in extreme cases lead to death. The body also needs to maintain acid-base balance inside cells so that cells continue to function effectively and intracellular proteins, such as haemoglobin, help to buffer acids inside cells.

What should we be monitoring?

The results obtained from analysis of arterial blood provides information about a number of factors involved in the process of acid-base balance as well as information about the amount of oxygen available to the cells. These include:

- pH value of arterial blood;
- the amount of O₂ in arterial blood (expressed as the partial pressure of oxygen or PaO₂);
The patient who needs respiratory support

- the amount of CO\textsubscript{2} in arterial blood (expressed as the partial pressure of carbon dioxide or PaCO\textsubscript{2});
- the amount of bicarbonate and bases available to buffer acids in arterial blood (expressed as mmol/l);
- arterial blood potassium levels;
- arterial haemoglobin;
- blood urea nitrogen, creatinine and glomerular filtration rate to monitor kidney function.

Why are these values important?

The body has a number of ways of maintaining the acid-base balance in health and we will look at five now.

Buffer systems are control mechanisms that can either increase or decrease the number of hydrogen ions in a solution, thus making the solution more acid if the hydrogen ions increase in number or more alkaline if the hydrogen ions are reduced in number (Mattson Porth and Matfin, 2009). These include:

- inside cells proteins act as buffer systems such as the plasma proteins;
- in the circulation it is the bicarbonate buffer system that converts a strong acid that releases large numbers of H\textsuperscript{+} to a weak acid that releases much fewer H\textsuperscript{+}. For example, hydrochloric acid (HCL: strong acid) can be substituted by carbonic acid (H\textsubscript{2}CO\textsubscript{3}: weak acid), thus reducing the overall H\textsuperscript{+}:

\[
\text{HCl + NaHCO}_3 \rightarrow \text{H}_2\text{CO}_3 + \text{NaCl} \quad \text{(sodium bicarbonate)} \quad \text{(sodium chloride)}
\]

This equation is reversible and is accelerated by the presence of the enzyme carbonic anhydrase. The carbonic acid produced dissociates into H\textsuperscript{+} and HCO\textsubscript{3}– (bicarbonate ions). The H\textsuperscript{+} combines with haemoglobin and the bicarbonate diffuses into plasma where it continues to participate in buffering acids.

Respiratory control mechanisms act as another line of defence against alterations in acid-base balance. An increase in ventilation decreases levels of CO\textsubscript{2} in the circulation and a decrease in ventilation increases CO\textsubscript{2} in the blood. Chemoreceptors in the brain stem, carotid and aortic bodies (see Figure 6.1, page 134) sense changes in CO\textsubscript{2}, hydrogen ions and O\textsubscript{2} and alter the respiratory rate accordingly. The respiratory control of pH is rapid and occurs within minutes of a change in pH balance but is only approximately 50–70% effective as a buffer system. It is the second line of defence against large changes in pH.

Renal control mechanisms are slower to react but can continue to function for days until the pH value has returned to the normal range. The mechanisms are:

- reabsorption of bicarbonate ions into the circulation;
- excretion of hydrogen ions from acids produced as a result of protein and fat metabolism.
Buffers are the body’s first line of defence

<table>
<thead>
<tr>
<th>The lungs are the body’s second line of defence</th>
<th>The kidneys are the body’s third line of defence</th>
</tr>
</thead>
<tbody>
<tr>
<td>They act within seconds</td>
<td>They act within seconds to minutes</td>
</tr>
<tr>
<td>They remove or release H⁺ to correct acid-base balance</td>
<td>They eliminate or retain CO₂ to maintain the ratio of carbonic acid to bicarbonate at 1:20</td>
</tr>
</tbody>
</table>

**Table 3.1: How the body defends against abnormal alterations in acid-base balance**

*Hydrogen-potassium exchange* when there is excess H⁺ in the blood, some is able to move into cells in exchange for potassium ions (K⁺), and when there is excess K⁺ in the blood, it moves into cells and exchanges with H⁺. Thus potassium levels and hydrogen levels can change dramatically in some clinical situations such as a patient with diabetic ketoacidosis (see Chapter 8).

*Blood urea nitrogen (BUN), creatinine and glomerular filtration rate through creatinine clearance*: the measurement of creatinine and glomerular filtration rate are very important measures of renal function and will give an indication of how efficient the patient’s kidney function is. If the patient has impaired kidney function the ability for the kidneys to act as the third line of defence in maintaining acid-base balance is impaired. A metabolic acidosis in the context of other indicators can suggest acute kidney injury or chronic renal disease (see Chapter 9).

**What do these values tell us?**

The pH value determines the presence of acidaemia and alkalaemia.

- **Acidaemia**: pH < 7.35 (a value below 7.35).
- **Alkalaemia**: pH > 7.45 (a value above 7.45).

The partial pressures of oxygen and carbon dioxide give a measure of respiratory function and the presence of respiratory acidosis/alkalosis.

- **Respiratory acidosis**:
  - pH < 7.35 and PaCO₂ > 6.0 kPa;
  - dyspnoea/increased or decreased respiratory function;
  - headache;
  - restlessness, confusion;
  - drowsiness/unconsciousness;
  - tachycardia and arrhythmias.

- **Respiratory alkalosis**:
  - pH > 7.45 and PaCO₂ < 4.9 kPa;
  - feeling light-headed;
The levels of bicarbonate and base excess give a measure of metabolic function and represent either a failure to buffer hydrogen ion concentrations with bases leading to acidosis or a failure to buffer bicarbonate concentrations with acids leading to an alkalosis.

- **Metabolic acidosis:**
  - pH <7.35 and $\text{HCO}_3^- < 22 \text{ mmol/L}$;
  - headache;
  - restlessness, confusion;
  - coma;
  - cardiac arrhythmias;
  - Kussmaul respirations (rapid shallow)/respiratory depression;
  - skin warm and flushed.

- **Metabolic alkalosis:**
  - pH >7.45 and $\text{HCO}_3^- > 27 \text{ mmol/L}$;
  - muscle twitching and cramps;
  - feeling dizzy;
  - confusion;
  - lethargy;
  - seizures/coma;
  - nausea and vomiting.

In Table 3.2 you will find clinical examples of patients who have experienced an acid-base imbalance.

**What does Jenny’s arterial blood gas result tell us about her condition?**

By using the step-by-step guide in Table 3.3, an analysis of Jenny’s arterial blood gas results and general condition indicate the following.

- **Step 1: Assess oxygenation**
  
  $\text{PaO}_2 = 8.7 \text{ kPa}$: there is evidence of hypoxaemia with $\text{SpO}_2 91\%$ with supplemental oxygen of 60%.

- **Step 2: Assess pH level**
  
  pH – 7.42: there is no evidence of respiratory acidosis or alkalosis.

- **Step 3: Assess respiratory component**
  
  $\text{PaCO}_2 = 3.6 \text{ kPa}$: this indicates that Jenny has been hyperventilating and expiring $\text{CO}_2$ in an attempt to cope with reduced volumes of air movement in her lungs due to bronchospasm. This is supported by her reduced peak expiratory flow rate (PEF) of 120 ml/min (her normal PEF: 300 ml/min).
<table>
<thead>
<tr>
<th>Arterial blood gas analysis</th>
<th>Patient examples</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Respiratory acidosis:</strong></td>
<td></td>
</tr>
<tr>
<td>pH &lt; 7.35</td>
<td>Gladys Cabrera (62 years) suffers from COPD and she is admitted to hospital with an acute exacerbation of her condition. She is unable to talk due to her breathlessness, rate of 40 bpm, SpO₂ is 72%, she is centrally cyanosed and she is unable to respond to commands. The results of an arterial blood sample are: pH 7.29, PaO₂ 4.8 kPa, PaCO₂ 8.4 kPa, HCO₃⁻ 28.5 mmol/L. Following a rapid assessment of Gladys’s condition she was admitted to intensive care for respiratory support and intensive treatment for type II respiratory failure (see Chapter 2).</td>
</tr>
<tr>
<td>PaCO₂ &gt; 6.0 kPa</td>
<td></td>
</tr>
<tr>
<td><strong>Respiratory alkalosis:</strong></td>
<td>Joan Butcher (50 years) suffers from anxiety attacks, and these have become worse since progressing to the menopause. On this occasion she has been involved in a minor road traffic collision and she has no obvious injuries. However, when the paramedics arrived at the scene they found her to be breathless and disorientated. She was complaining of pins and needles in her hands and arms, and she felt she couldn’t get her breath. Joan was taken to accident and emergency where her arterial blood gas result following admission was: pH: 7.49; PaCO₂: 3.2 kPa; HCO₃⁻: 24.2 mmol/L; BE (base excess): -1.0. Joan was hyperventilating and needed to be encouraged to reduce her respiratory rate and allow her carbon dioxide levels to rise back to normal levels.</td>
</tr>
<tr>
<td>pH &gt; 7.45</td>
<td></td>
</tr>
<tr>
<td>PaCO₂ &lt; 4.9 kPa</td>
<td></td>
</tr>
<tr>
<td><strong>Metabolic acidosis:</strong></td>
<td>Mary Bevan (58 years) was found by her neighbour lying at the front door in a drowsy and confused state. Mary has type 2 diabetes and has recently developed a severe infection on her leg. Mary’s neighbour called the emergency services and Mary was admitted to accident and emergency. Her arterial blood gas following admission was: pH: 7.24; PaCO₂: 3.8 kPa; HCO₃⁻: 15.1 mmol/L; BE: -13.7. Mary had Kussmaul respirations at a rate of 35 bpm and a blood glucose of 22 mmol/L. Mary had developed a metabolic acidosis secondary to infection that triggered an increase in blood glucose that necessitated management with insulin.</td>
</tr>
<tr>
<td>pH &lt; 7.3</td>
<td></td>
</tr>
<tr>
<td>HCO₃⁻ &lt; 22 mmol/L</td>
<td></td>
</tr>
</tbody>
</table>
**Metabolic alkalosis:**
\[ \text{pH} > 7.45 \]
\[ \text{HCO}_3^- > 26 \text{ mmol/L} \]

Gary Smith (54 years) has been suffering from indigestion-type pain for several days. Rather than go to the GP he has been treating himself with large doses of antacids such as bicarbonate of soda. That afternoon he felt nauseated, weak and tired and still had the persistent indigestion. He visited the GP who decided to admit him to hospital for an assessment of his chest pain. His arterial blood gas following admission was:
\[ \text{pH}: 7.49; \text{PaCO}_2: 5.6 \text{ kPa}; \text{HCO}_3^-: 29.7 \text{ mmol/L}; \text{BE}: +9.0. \]

**Respiratory and metabolic acidosis:**
\[ \text{pH} < 7.35 \]
\[ \text{PaCO}_2 > 6.0 \text{ kPa} \]
\[ \text{HCO}_3^- < 22 \text{ mmol/L} \]

Peter Baker (41 years) was admitted to an acute ward with a history of abdominal pain, nausea and vomiting. Peter’s condition deteriorated during the first 24 hours, and that evening he had a cardiac arrest. He was resuscitated and transferred to ICU for respiratory support and management of acute pancreatitis. His arterial blood gas following admission was:
\[ \text{pH}: 7.15; \text{PaCO}_2: 7.6 \text{ kPa}; \text{HCO}_3^-: 16.7 \text{ mmol/L}; \text{BE}: -9.8. \]

Peter has developed a combined acidosis as a result of his cardiac arrest (failed respiration) and severe sepsis associated with pancreatitis and lactic acidosis (see Chapter 7).

---

| Table 3.2: Clinical examples of patients with changes in acid-base balance | Metabolic alkalosis: 
Gary Smith (54 years) has been suffering from indigestion-type pain for several days. Rather than go to the GP he has been treating himself with large doses of antacids such as bicarbonate of soda. That afternoon he felt nauseated, weak and tired and still had the persistent indigestion. He visited the GP who decided to admit him to hospital for an assessment of his chest pain. His arterial blood gas following admission was:  
\[ \text{pH}: 7.49; \text{PaCO}_2: 5.6 \text{ kPa}; \text{HCO}_3^-: 29.7 \text{ mmol/L}; \text{BE}: +9.0. \]  
**Respiratory and metabolic acidosis:**  
Peter Baker (41 years) was admitted to an acute ward with a history of abdominal pain, nausea and vomiting. Peter’s condition deteriorated during the first 24 hours, and that evening he had a cardiac arrest. He was resuscitated and transferred to ICU for respiratory support and management of acute pancreatitis. His arterial blood gas following admission was:  
\[ \text{pH}: 7.15; \text{PaCO}_2: 7.6 \text{ kPa}; \text{HCO}_3^-: 16.7 \text{ mmol/L}; \text{BE}: -9.8. \]  
Peter has developed a combined acidosis as a result of his cardiac arrest (failed respiration) and severe sepsis associated with pancreatitis and lactic acidosis (see Chapter 7). |
Chapter 3

• **Step 4: Assess the metabolic component**

HCO$_3^-$ 24 mmol/L: this indicates that Jenny has no evidence of metabolic acidosis or alkalosis.

• **Step 5: Combine your findings**

Jenny is not experiencing any form of acidosis or alkalosis based on these blood gas results, however, the presence of a PaCO$_2$ of 3.6 kPa indicates that Jenny’s hyperventilation and lower than normal PaCO$_2$ is correcting any potential for acidosis.

• **Step 6: Clinical interpretation and recommendation**

Clinically Jenny is showing signs of type I respiratory failure (see Chapter 2), she is experiencing increased work of breathing and a reducing peak expiratory flow. The combination of salbutamol (bronchodilation) nebulisers and hydrocortisone will have a direct anti-inflammatory effect on her hypersensitive bronchi and should relieve her symptoms. Jenny, however, continues to be at risk of an escalation of her condition due to a secondary or late response to the initial trigger and requires close monitoring and support during this critical stage (BTS and SIGN, 2014; McCance and Huether, 2014).

### Always risk assess

<table>
<thead>
<tr>
<th>ABG: Step 1</th>
<th>Look: Listen: Feel: Measure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assess oxygenation.</td>
<td></td>
</tr>
</tbody>
</table>
| Normal: PaO$_2$ 11.5–13.5 kPa | • Is there evidence of hypoxaemia?  
|  | • Is there evidence of high levels of oxygenation?  
|  | • Is the patient receiving supplemental oxygen?  |
| ABG: Step 2 |  |
| Assess pH level. |  |
| Normal: 7.35–7.45 | • Is there evidence of acidosis? pH <7.35  
|  | • Is there evidence of alkalosis? pH >7.45  |
| ABG: Step 3 |  |
| Assess the respiratory component. |  |
| PaCO$_2$: 4.5–6.0 kPa | • Is the PaCO$_2$ <4.5 kPa?  
|  | • Is the PaCO$_2$ >6.0 kPa?  |
| ABG: Step 4 |  |
| Assess the metabolic component. |  |
| HCO$_3^-$: 22–27 mmol/L. | • Is the HCO$_3^-$ <22 mmol/L?  
|  | • Is the HCO$_3^-$ >27 mmol/L?  
|  | • The base excess level (BE) is the quantity of acid or base required to restore the pH to 7.4. Base excess will mirror the bicarbonate level and simply reinforces evidence of a metabolic component (Jevon and Ewens, 2007).  |
| ABG: Step 5 |  |
| Combine your findings |  |
|  | • Combine your findings from steps 2/3/4 and identify if there is evidence of:  
|  | o respiratory acidosis;  
|  | o respiratory alkalosis;  
|  | o metabolic acidosis;  
|  | o metabolic alkalosis;  |
The patient who needs respiratory support

- signs that the respiratory system has compensated for a metabolic acidosis by increasing the respiratory rate and reducing the CO₂ level;
- signs that the renal system has compensated for chronic respiratory acidosis by increasing the level of HCO₃⁻.

<table>
<thead>
<tr>
<th>ABG: Step 6</th>
<th>Clinical interpretation and recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Interpret the ABGs in the context of all available patient data.</td>
</tr>
</tbody>
</table>

Table 3.3: A step-by-step approach to assessing arterial blood gas results (ABG)

Activity 3.1  
**Decision making**

Read the scenario below and think about the significance of the arterial blood gas results.

Joseph Baglio (age 68 years) has smoked 40 cigarettes a day since his twenties and has experienced angina on exertion for the last five years although this has been managed by the use of beta blockers and GTN. He was admitted to the medical ward six hours ago following a diagnosis of pneumonia. Following his admission he seemed to be responding well to the oxygen therapy and IV antibiotics when he pressed the buzzer and fell forward clutching his chest. When the nurse arrived she found that Joseph was unresponsive with absent respirations and pulse. A cardiac arrest call was placed and he was resuscitated successfully. His arterial blood gas results 30 minutes after his resuscitation were:

- pH: 7.05
- PaO₂: 8.5 kPa on 60% high-flow oxygen (SpO₂ 82%)
- PaCO₂: 14.1 kPa
- HCO₃⁻: 20.5 mmol/L
- BE: -3.0

Joseph was conscious, flushed and anxious. His vital signs were T: 38.0°C, R: 32/min, P: 95, BP: 110/70 mmHg.

- Using the step-by-step guide in Table 3.3, what can you interpret from the arterial blood gas result?

One hour later Joseph was conscious but confused. His skin was cold and clammy to touch and his vital signs were T: 38.0°C, R: 32/min, P: 94, BP: 110/70 mmHg. He was receiving 60% humidified high-flow oxygen and diagnosed with acute coronary syndrome.

(Continued)
syndrome with evidence of ST elevation myocardial infarction (STEMI). As well as his beta blockers, he has been prescribed statins for reducing cholesterol (he had previously refused to commence statins when prescribed before), clopidogrel to reduce the risk of another thrombotic event and morphine and nitrates for chest pain. Joseph was not considered a suitable candidate for percutaneous coronary angiography because of his pneumonia and was assessed as a candidate for thrombolysis instead but this was also decided against in light of his traumatic resuscitation. He now had two acute morbidities affecting his respiratory and cardiac system. A second arterial blood gas result was:

\[
\begin{align*}
\text{pH: } & 7.20 \\
\text{PaO}_2: & 9.4k \text{ Pa (SpO}_2\text{89\%)} \\
\text{PaCO}_2: & 6.70 \text{ kPa} \\
\text{HCO}_3: & 21.4 \text{ mmol/L}
\end{align*}
\]

- Using the step-by-step guide in Table 3.3, what can you interpret from the arterial blood gas result?
- What are your priorities of care for this patient?

*Answers are given at the end of the chapter.*

With reference to Jenny’s story we can see the importance of using a holistic approach to rapid assessment. There are a number of factors that now become significant when monitoring Jenny’s condition. For example we know that:

- Jenny has now been awake and fighting for breath since 2 a.m. and it is now 4 a.m.;
- she is emotionally distressed after her husband appears to have left her;
- she is still recovering from an acute respiratory infection and is presenting with type I respiratory failure and about to be transferred to high dependency care.

### Case study: Jenny’s transfer to HDU

*Following her assessment in the emergency room, Jenny was considered to be a level 2 patient requiring high dependency care for assessment and monitoring of her respiratory system. Following admission to HDU the results of her assessment were as follows.*

**A:** Responding to commands and maintaining her airway.

**B:** \( \text{SpO}_2: 91\% \)

- 60% high-flow humidified \( \text{O}_2 \)
- \( R: 36/\text{min} \)
- \( \text{ABGs}: \)
- \( \text{pH} 7.36 \)
The patient who needs respiratory support

\[
\begin{align*}
\text{PaO}_2 & \quad 8.8 \text{ kPa} \\
\text{PaCO}_2 & \quad 4.2 \text{ kPa} \\
C: & \quad \text{HR: 128/min} \\
& \quad \text{BP: 130/72 mmHg} \\
D: & \quad \text{Blood glucose 6.7 mmol/L} \\
& \quad \text{Agitated but disorientated} \\
E: & \quad \text{Temp: 37.5°C}
\end{align*}
\]

According to BTS and SIGN (2014), the evidence of a severe hypoxia in the presence of a normalizing PaCO\textsubscript{2} and persistent disorientation following intensive treatment, indicates Jenny is having a life-threatening attack. She has been prescribed a once only dose of intravenous magnesium sulphate in an attempt to produce further bronchodilation (Blitz et al., 2005). When her current situation is assessed in the context of her previous admission to ICU, Jenny is referred to the ICU specialist who suggests that she meets the criteria for non-invasive ventilation (NIV) and that commencement of NIV could prevent her from needing intubation and invasive ventilation (Lim et al., 2012). The plan for Jenny is to commence her on NIV and monitor her ABCDE continuously for signs of improvement or deterioration.

What is NIV and why is it appropriate to use this respiratory support for Jenny?

Pulmonary ventilation, or breathing, is essential for life, and the purpose of NIV is to provide varying levels of positive pressure air flow through a tight-fitting mask in order to improve the patient’s levels of PaO\textsubscript{2} and PaCO\textsubscript{2}. Breathing involves the inhalation of gases in air into the lungs and exhalation of gases from the lungs into the atmosphere. All gases in air collectively exert a pressure known as atmospheric pressure. The gases in the lungs also exert a pressure known as alveolar pressure. In air, gases always flow from an area of high pressure to an area of low pressure. During inspiration the thoracic space expands as a result of contraction of the intercostal muscles and diaphragm. This increase in space reduces the overall alveolar pressure in the lungs and air flows into the airways in order to equalise the pressure. Expiration involves relaxation of the respiratory muscles and natural elastic recoil of the lung tissue so that air flows back into the atmosphere. Normal breathing therefore relies on negative pressure ventilation.

For 120 years the principal method of supporting ventilation for patients with respiratory failure was based on the principle of negative pressure ventilation. For example, the iron lung was used successfully for patients with respiratory failure caused by neuromuscular diseases such as polio. In the 1950s, during the polio epidemic in Europe, the demand for iron lungs outstripped supply and alternative methods for providing respiratory support were attempted (Lassen et al., 1954). This led to the development of mechanical invasive ventilation (MIV), which involved air being forced under pressure into patients’ lungs via a tracheostomy tube or endotracheal tube at a rate
of between 10 and 20 per minute in order to mimic normal respiration. This dramatically reduced the mortality rate of patients suffering from respiratory failure and became the mainstay treatment (Borthwick et al., 2003).

In the last 25 years the use of non-invasive positive pressure ventilation (NIV) techniques that supply air through a tight-fitting face mask rather than a tube have escalated, and this method has now become the first-line therapy for adult patients with:

- sleep apnoea;
- acute exacerbations of COPD;
- pulmonary oedema;
- neuromuscular disease;
- pneumonia;
- weaning from MIV (BTS, 2000; BTS, 2008; NICE, 2010c).

In Jenny’s case the use of NIV to manage an acute severe asthma attack does not have such a strong evidence base (Medoff, 2008). However, BTS and SIGN (2014) recommend that it should be considered as an option to prevent the risk of intubation in patients with acute severe asthma but should be based on skilled clinical assessment and knowledge of the patient’s condition. Jenny’s respiratory function is compromised but not so impaired that she is in imminent danger of complete respiratory collapse. She is able to protect her own airway, has only mild disorientation and there is no evidence of a pneumothorax on chest X-ray (Medoff, 2008). The types of NIV and their use are explained in Table 3.4.

### Activity 3.2

**Reflection**

Reflect back on patients you have nursed and ask yourself the following questions.

- Have I looked after patients with acute respiratory failure either in hospital or the community?
- If so, how did I assess and document the patient care?
- Did the patient need support with oxygen therapy or NIV?
- Did the patient have support from the physiotherapist, dietitian and respiratory nurse?

Hint: This reflection is meant to encourage you to think critically about assessing and managing care and should help you to identify good practice and areas for improvement.

As this answer is based on your own reflection, there is no outline answer at the end of the chapter.

### Contraindications for using NIV

The success of NIV techniques in the support of respiratory function relies on effective patient selection. For Jenny, CPAP (can also be referred to as pressure support when given through some ventilators) was chosen as the optimum treatment regime, but this does not mean that the use of NIV will always lead to a successful outcome for every patient. Patients need to be risk assessed
<table>
<thead>
<tr>
<th>Type of NIV</th>
<th>Benefits</th>
<th>Risks</th>
<th>Patient examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continuous positive airways pressure: CPAP.</td>
<td>• Improves oxygenation in patients with type I respiratory failure.</td>
<td>• There is reduced clearance of CO₂ due to air being trapped in the alveoli. Not suitable for patients with type II respiratory failure where there are increased levels of CO₂.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Reduces the risk of atelactasia.</td>
<td>• The airway is not protected so patients must be able to maintain their own airway.</td>
<td>• Mrs Smith is admitted with severe breathlessness and is producing excessive amounts of pink frothy secretions from her airways. She is diagnosed with acute pulmonary oedema and is commenced on CPAP starting at 5 cm H₂O as part of her ongoing treatment to reduce pulmonary secretions by increasing alveolar pressure to above capillary hydrostatic pressure.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Chao Chan is diagnosed with pneumonia and type I respiratory failure. His PaO₂ is 6.2 kPa and his PaCO₂ is 3.6 kPa. He is commenced on CPAP at 5 and then 10 cm H₂O.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Bryn Jones has been diagnosed with obstructive sleep apnoea. He suffers from morbid obesity, snoring and daytime fatigue. He has now been fitted with a face mask and CPAP machine for home use. The equipment delivers CPAP at 10 cm H₂O, to be used at night while sleeping.</td>
</tr>
</tbody>
</table>
Table 3.4 (Continued)

<table>
<thead>
<tr>
<th>Type of NIV</th>
<th>Benefits</th>
<th>Risks</th>
<th>Patient examples</th>
</tr>
</thead>
</table>
| Bilevel NIV or bilevel positive airways pressure ventilation: BiPAP. | - Improves oxygenation and CO\textsubscript{2} clearance in patients with type II respiratory failure.  
   - IPAP reduces the work of breathing and conserves the use of oxygen by the body.  
   - A lower EPAP pressure reduces air trapping but still allows continuous gas exchange during respiration while preventing atelactasis. | - The airway is not protected so patients must be able to maintain their own airway. | - Henry Jones has pneumonia. His PaO\textsubscript{2} is 6.8 kPa and his PaCO\textsubscript{2} is 6.5 kPa. He is breathless and agitated. He is commenced on BiPAP with an inspiratory pressure of 10 cm H\textsubscript{2}O and an expiratory pressure of 4 cm H\textsubscript{2}O.  
- Gladys Cabrera (62 years) suffers from COPD and she is admitted to hospital with an acute exacerbation of her condition. She was commenced on BiPAP at an inspiration pressure (IPAP) of 12 cm H\textsubscript{2}O and an expired pressure (EPAP) of 5 cm H\textsubscript{2}O with 40% oxygen. She didn’t like the face mask but was prepared to give it a try as long as the nurse reminded her. |
for any contraindications before commencing the therapy and then risk assessed for evidence of any change or deterioration in their condition. This is illustrated in Table 3.5. The contraindications of NIV rarely exist in isolation: often patients will present with one or more of these factors. Knowing the patient and their medical history is an essential part of the rapid decision-making process required when determining a patient’s suitability for NIV and relies on good communication between all the carers involved (RCP et al., 2008). Contraindications include:

- life-threatening hypoxaemia;
- severe confusion/agitation/cognitive impairment;
- unconscious patient;
- airway obstruction due to vomiting or a foreign object;
- facial trauma/burns/surgery;
- **pneumothorax**;
- patient unable to protect their own airway;
- copious amounts of respiratory secretions/sputum;
- recent surgery in the upper gastro-intestinal tract;
- severe co-morbidity;
- haemodynamic instability;
- presence of bowel obstruction.

Table 3.5 offers a summary of the risk assessment and nursing interventions required to care for patients receiving NIV.

<table>
<thead>
<tr>
<th>Risk assessment</th>
<th>Nursing interventions</th>
</tr>
</thead>
</table>
| Contraindications for use of NIV | - Rapid assessment of ABCDE using ‘Look: Listen: Feel: Measure’ is important to measure the risk of contraindications to treatment with NIV. In particular, the risk of pneumothorax should be ruled out by reviewing the patient’s chest X-ray following their admission.  
- A patient may decide to refuse treatment. |
| Preparation of the patient and technology | - If the patient has consented and is able to proceed, ensure the equipment has been prepared and checked to ensure it is in working order.  
- Sit the patient upright and, with their cooperation, attach the face mask. The patient will need a few minutes to get used to the mask. Often NIV is commenced at a low level and increased according to the clinical state of the patient (RCP et al., 2008).  
- Document baseline clinical data.  
- Agree and document a treatment plan for escalating and identifying a ceiling of treatment. |
### Chapter 3

**Table 3.5  (Continued)**

<table>
<thead>
<tr>
<th>Risk assessment</th>
<th>Nursing interventions</th>
</tr>
</thead>
</table>
| Airway and respirations              | • Monitor the patient’s airway and respiratory rate, look for signs of respiratory distress and air entry as illustrated in Figure 2.1, page 36.  
• Monitor SpO$_2$ for evidence of improvement or deterioration.  
• Monitor the patient’s arterial blood gas results after:  
  o one hour: if there is no change in the patient’s condition or a slight improvement, then monitor again in four hours;  
  o one hour: if there is a deterioration in the patient’s condition:  
    o assess patient and check the equipment;  
    o consider either increasing the oxygen or pressures;  
    o consider a change to mechanical ventilation. |
| Haemodynamic state                   | • The increase in pulmonary airway pressure from NIV can cause a rebound reduction in the patient’s blood pressure, particularly with CPAP pressures above 10 cm H$_2$O.  
• Monitor the patient’s blood pressure every five minutes during the first 30 minutes and then at 30 minutes to hourly as the patient’s blood pressure stabilises. Continuous arterial monitoring of blood pressure provides an effective way to monitor BP as well as obtaining arterial samples for blood gas analysis. |
| Mental state and level of consciousness | • Monitor for signs of increased confusion or agitation. Any deterioration in level of consciousness is an indication that the NIV should be discontinued and the treatment plan utilised.  
• Patients on NIV should not normally be sedated as this can compromise their airway and compliance with treatment. |
| Fluid balance and gastro-intestinal function | • There is a risk of fluid retention triggered by the stress response (Chapter 6). Look for evidence of reduced urine output and interstitial oedema.  
• There is a risk of increased air swallowing and gastric distension associated with the air flow. This may be reduced by inserting a nasogastric tube. |
| Psychological distress               | • Patients receiving NIV experience discomfort and distress due to the tight-fitting mask and side effects of the treatment. Communication is difficult with the face mask in place, although this may be resolved for some patients by using a nasal mask. Alternative techniques for delivering the air under pressure include a mouth piece and a helmet.  
• The role of the nurse in providing support and reassurance is essential. Frequent removal of the mask is counterproductive, and it is important to encourage the patient to keep the mask in situ for at least 30 minutes if any benefit is to be achieved. |
If a patient is becoming very distressed, this will impact on their physiological state and is often an indication to discontinue the NIV and refer to the treatment plan (Jarvis, 2006).

Optimum management of patients with acute respiratory failure and NIV is achieved in ICU. However, patients can be nursed in acute wards and accident and emergency provided there is an appropriate skill mix and staff ratios of 1 or 2 patients to 1 nurse.

Table 3.5: Risk assessment and management of patients receiving NIV

Case study: Jenny’s condition changes

Jenny consented to the use of NIV and commenced the support at a low level of positive pressure (5 cm) and this was gradually increased to 10 cm with support and encouragement from the nursing staff. There seemed to be an initial improvement with an increase in SpO₂ (94%). However, two hours after commencing the NIV Jenny’s condition rapidly deteriorated as illustrated in the following assessment.

A: Difficult to rouse but still able to maintain her airway.
B: SpO₂ 91% on O₂ 60% high-flow humidified O₂
   R 39/min, with shallow respirations
   Blood gases showed:
   pH 7.20
   PaO₂ 8.2 kPa
   PaCO₂ 9.8 kPa
   HCO₃ 24 mmol/L
C: HR: 128/min
   BP: 110/72 mmHg
D: Blood glucose 6.7 mmol/L
   GCS had dropped to 9 (eyes opening to pain 2, inappropriate words 3 and flexion to pain 4).
E: Temp: 37.5°C

ABG analysis

- Jenny has persistent hypoxaemia.
- She is acidicotic.
- The high CO₂ indicates a respiratory acidosis.
- No sign of a metabolic acidosis.

(Continued)
• Jenny has hypoxia and a respiratory acidosis.
• Her worsening clinical condition of a high respiratory rate, reduction in her level of consciousness and increasing heart rate, combined with hypoxia and respiratory acidosis, indicates severe type II respiratory failure. Immediate intervention with intubation and mechanical ventilation is now required.

What happens when NIV is not suitable: the case for mechanical invasive ventilation (MIV)

The benefits of supporting patients with respiratory failure with NIV include the following (RCP et al., 2008).
• There is reduced risk of ventilator-acquired pneumonia.
• The patient is fully awake and an active partner in their care.
• The patient may be nursed in an acute care setting.
• The use of NIV may prevent the requirement for invasive respiratory support.

There are, however, a number of reasons why patients may require an escalation of treatment to MIV or direct intervention with MIV without NIV (Brainard and Deutschman, 2010). These include:
• life-threatening hypoxic (PaO₂ below 8.0 kPa) respiratory failure accompanied by patient confusion and/or exhaustion;
• life-threatening hypercarbic (PaCO₂ above 6.0 kPa) respiratory failure accompanied by patient confusion and/or exhaustion;
• impaired consciousness and/or the patient’s inability to protect their airway.

For patients in these situations, clinical assessment, combined with medical and nursing experience, is the most important tool for judging when invasive support with intubation and mechanical ventilation is required. Based on Jenny’s assessment following her deterioration, she meets all of the three criteria above for intubation and mechanical ventilation. Jenny’s sudden deterioration may have been related to the combined effects of physical exhaustion and the latent release of inflammatory mediators triggered by the initial inflammatory response several hours before. This can lead to further bronchospasm, oedema, mucus secretion and obstruction of air flow, an increase in variable and uneven airway obstruction and air trapping in the alveoli and hyperventilation (McCance and Huether, 2014). According to Medoff (2008) and Brenner et al. (2009) it is the combination of progressive airways obstruction and physical exhaustion caused by the increased work of breathing that leads to a reduction in the patient’s respiratory tidal volume, retention of carbon dioxide, respiratory acidosis and deteriorating cardiovascular...
## Reason for MIV

<table>
<thead>
<tr>
<th>Hypoxaemic respiratory failure.</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Pneumonia.</td>
</tr>
<tr>
<td>• Lung consolidation.</td>
</tr>
<tr>
<td>• Atelectasis.</td>
</tr>
<tr>
<td>• Pulmonary oedema.</td>
</tr>
<tr>
<td>• Acute respiratory distress syndrome (ARDS).</td>
</tr>
<tr>
<td>• Pulmonary embolism.</td>
</tr>
<tr>
<td>• Carbon monoxide poisoning.</td>
</tr>
</tbody>
</table>

### Look: Listen: Feel: Measure

- Central cyanosis.
- Altered respiratory pattern.
- Agitation/irritability.
- Confusion.
- Exhaustion.
- Seizures.
- SpO$_2$ $<85\%$.
- PaO$_2$ $<8.0$ kPa.

### Patient examples

<table>
<thead>
<tr>
<th>Chao Chan (Table 3.4) is diagnosed with pneumonia and type I respiratory failure. His PaO$_2$ is 6.2 kPa and his PaCO$_2$ is 3.6 kPa. He was commenced on CPAP at 5 cm H$_2$O then 10 cm H$_2$O. However, after the first hour he was confused and agitated, pulling off his mask and refusing to put it back on. His ABGs were PaO$_2$ 5.7 kPa and PaCO$_2$ 5.0 kPa. It was agreed that treatment should be escalated to MIV.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mariana Banica (27 years) has a severe scoliosis of her spine (the spine is curved from side to side in an S shape). Since childhood she has been prone to respiratory infections due to reduced and uneven lung capacity. Mariana was admitted to ICU after having collapsed at home following a flu-like illness for three days. On admission she was very confused, cyanosed and her breathing was shallow. Her ABGs were pH: 7.19; PaO$_2$: 12.7 kPa; PaCO$_2$: 10.7 kPa; HCO$_3$$: 24.0$ mmol/L; BE: 0.1. Mariana was intubated and commenced on BiPAP at a rate of 15/min, with an IPAP of 20 cm H$_2$O and EPAP of 5 cm H$_2$O.</td>
</tr>
</tbody>
</table>

## Hypercapnic respiratory failure.

<table>
<thead>
<tr>
<th>COPD.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma.</td>
</tr>
<tr>
<td>Airway obstruction/anatomical.</td>
</tr>
<tr>
<td>Deformity.</td>
</tr>
<tr>
<td>Cervical injury above level C4 and/or damage to the brain stem.</td>
</tr>
<tr>
<td>Excessive sedation.</td>
</tr>
<tr>
<td>Guillain-Barré syndrome.</td>
</tr>
<tr>
<td>Cardiac arrest.</td>
</tr>
<tr>
<td>Heart failure.</td>
</tr>
<tr>
<td>Pulmonary embolism.</td>
</tr>
</tbody>
</table>

### Look: Listen: Feel: Measure

- Increased work of breathing.
- Use of accessory muscles.
- Shallow breathing.
- Dyspnoea.
- Agitation/irritability.
- Confusion.
- Exhaustion.
- Seizures.
- Cardiovascular collapse and cardiac arrest.
- PaCO$_2$ $>6.0$ kPa.

### Patient examples

- Mariana Banica (27 years) has a severe scoliosis of her spine (the spine is curved from side to side in an S shape). Since childhood she has been prone to respiratory infections due to reduced and uneven lung capacity. Mariana was admitted to ICU after having collapsed at home following a flu-like illness for three days. On admission she was very confused, cyanosed and her breathing was shallow. Her ABGs were pH: 7.19; PaO$_2$: 12.7 kPa; PaCO$_2$: 10.7 kPa; HCO$_3$$: 24.0$ mmol/L; BE: 0.1. Mariana was intubated and commenced on BiPAP at a rate of 15/min, with an IPAP of 20 cm H$_2$O and EPAP of 5 cm H$_2$O.
Table 3.6: Indications for mechanical invasive ventilation in the critically ill patient

<table>
<thead>
<tr>
<th>Reason for MIV</th>
<th>Look: Listen: Feel: Measure</th>
<th>Patient examples</th>
</tr>
</thead>
</table>
| Impaired consciousness and/or the patient’s inability to protect his/her airway.  
  - Glasgow Coma Scale (GCS) score of <8 indicates the potential for further deterioration in consciousness, reduced ventilation and poor airway protection, for example:  
    - severe brain injury;  
    - prolonged effects of general anaesthetic;  
    - traumatic injury of the face and neck. | Inability to maintain airway. Unconscious. GCS <8. | Pete Williams (19 years) was assaulted on his way home from the pub. A witness said that Pete had been kicked repeatedly on the head while he lay on the floor. In ICU he was agitated and unable to communicate except with grunts. He was opening his eyes and flexing his arms to pain, GCS 7. The computerised tomography scan showed evidence of progressive brain swelling. The management plan for Pete in the first 24 hours was to intubate him with an oral endotracheal tube and provide continuous pressure ventilation (IPAP 30 cm H₂O) with a rate of 15/min in order to protect his airway and maintain PaO₂ > 8.0 kPa and PaCO₂ 4.5–6.0 kPa. Pete developed ventilator-acquired pneumonia on day four and stayed on MIV for seven days. |
and neurological state, as illustrated in Jenny’s case study. Other clinical examples of situations when MIV is required are included in Table 3.6.

Mechanical invasive ventilation in adults can only take place when a patient is intubated with a cuffed endotracheal or tracheostomy tube. The cuff provides a seal around the tube and prevents leaks. The purpose of MIV is to push air under pressure into the patient’s lungs to ensure there is effective movement of oxygen and carbon dioxide in and out of the lungs (pulmonary ventilation). There are increasing numbers of types and modes of MIV, but for the purposes of this chapter we will limit discussion to two core modes: pressure-controlled ventilation and volume-controlled ventilation (Carbery, 2008; Grossbach et al., 2011). In Table 3.7 you will find an explanation of these modes together with the advantages and disadvantages of both.

In Table 3.7 you will find an explanation of these modes together with the advantages and disadvantages of both.

In both pressure-controlled and volume-controlled ventilation the patient’s respiratory rate can be managed in one of three ways.

- The patient breathes spontaneously and controls their own rate.
- The patient’s respiratory rate is set and controlled by the machine.
- The patient’s respiratory rate is supported by a minimum respiratory rate set by the machine and supplemented by the patient’s own respiratory rate.

The option of as much or as little respiratory support through MIV allows the patients to be involved in the process of respiratory support and aids their readiness to wean from MIV as they improve.

Case study: Jenny’s intubation and ventilation with MIV

Jenny now required immediate intubation and mechanical ventilation and she was induced into anaesthesia with ketamine and alfentanil (short-acting anaesthetic agents) and paralysed with suxamethonium (a fast-acting muscle relaxant) to facilitate safe tracheal intubation with an oral endotracheal tube. Because of the combination of risks related to hyperinflation of the lungs, air trapping and increased airways resistance caused by bronchospasm, inflammation and mucus production, it was decided that the best clinical intervention for Jenny was controlled ventilation with SIMV (Table 3.7), with an inspired tidal volume set at 400 ml and a rate of 16 breaths per minute and a plateau airways pressure of 30 cm. The respiratory rate was set to give Jenny a short inspiration time and a prolonged expiratory time to reduce the risks of further air trapping and barotrauma (Brenner et al., 2009). In order to achieve this type of controlled ventilation it was necessary to fully sedate and paralyse Jenny with neuromuscular blockade and this was achieved by the use of propofol (a short-acting anaesthetic) and cisatracurium (a short-acting neuromuscular blocking agent). Jenny’s airway resistance was reduced by suctioning of the airways to remove secretions. A chest X-ray was performed to check the position of the endotracheal tube.
<table>
<thead>
<tr>
<th><strong>MIV mode</strong></th>
<th><strong>Risks</strong></th>
<th><strong>Benefits</strong></th>
</tr>
</thead>
</table>
| **Pressure-controlled/pressure-support ventilation:** air is pushed into the lungs until a preset alveolar pressure is reached. For example:  
- bilevel positive airways pressure (BiPAP) (see NIV).  
- continuous positive airways pressure (CPAP) (see NIV).  
- pressure-support ventilation (PS).  
- positive end expiratory pressure (PEEP). | Ineffective ventilation.  
Hypo ventilation and variable tidal volumes triggered by reduced lung compliance in the presence of acute lung injury, sputum and/or bronchospasm.  
Compliance measures the ‘ease of stretch’ ability in the lungs. The more compliant the lungs are, the less pressure is required to open the airways during MIV. | Reduces the risk of ventilator-associated lung injury. |
| **Volume-controlled ventilation:** a preset volume of air is delivered to the lungs with each breath. For example:  
- synchronised intermittent mandatory ventilation (SIMV). | Ventilator-associated lung injury:  
- barotrauma: over-distension of some alveoli;  
- volutrauma: over-distension of the alveoli caused by large tidal volumes;  
- biotrauma: the release of inflammatory mediators that may increase patient mortality. | The machine delivers a set tidal volume with each breath, thus improving overall ventilation. |
| **Modes that deliver a combination of both.** For example:  
Ensures effective tidal volumes and pulmonary ventilation. | |

*Table 3.7: A comparison of pressure-controlled and volume-controlled ventilation modes*
The patient who needs respiratory support

Research summary: Sedation

The aim of using drugs to sedate patients during MIV is to promote comfort, relieve distress and anxiety, and facilitate effective respiratory function. The majority of drugs used for this purpose, however, can cause side effects, including: depression of the cardiovascular system leading to reduced BP; respiratory depression and delayed weaning from respiratory support; reduced motility of the gastro-intestinal tract with delayed absorption of nutrients and poor quality sleep (Whitehouse et al., 2014). The use of sedation assessment scales and sedation protocols have been recommended as a method for getting the balance right between the advantages and disadvantages of using sedation. The Ramsay scale, Riker Sedation-Agitation scale and Richmond Agitation and Sedation scale are examples of tools adapted for patients on MIV (Ramsay et al., 1974; Riker et al., 2001; Ely et al., 2003). There is limited evidence, however, that such scales and protocols can improve patient outcomes (O’Connor et al., 2010; Williams et al., 2008; Whitehouse et al., 2014). There is evidence, however, that daily sedation interruption combined with patient assessment can improve patient outcome (Chen et al., 2014). There is also evidence that healthcare staff do not always follow sedation recommendations due to lack of awareness, lack of conceptual agreement with the guidance, poor strength of evidence in their use and lack of clarity over who is responsible for prescribing the guidance (Sneyers et al., 2014; Miller et al., 2012). In summary, the use of sedation protocols and daily sedation interruption while seen to be clinically effective continue to be areas that require further research and should always be used in the context of the patient’s clinical condition.

Why are tidal volume, respiratory rate and airway pressure important in promoting optimum ventilation?

The tidal volume (TV) is the volume of air in each breath and can be measured as inspired (ITV) and expired (ETV) tidal volume. The respiratory rate (R) describes the total number of respirations in a minute. If a patient is on MIV this may include set ventilator breaths and the patient’s own breaths. Minute volume is the total volume of air either inspired (IMV) or expired (EMV) in one minute and is equal to tidal volume times respiratory rate (Hall, 2011). Airway pressure is the same as alveolar pressure and is the pressure required or allowed to push air into the patient’s lungs.

When assessing and monitoring a patient receiving MIV, tidal volume, rate, minute volume and airway pressure are some of the important indicators for measuring effective ventilation. For example, increasing ITV, R or IMV can improve the elimination of CO$_2$. If, however, by doing this the inspired airway pressure goes above 30–35 cm H$_2$O, then the patient becomes at risk of acute lung injury. Patients such as Jenny often have high airway resistance and it becomes harder to push air into the lungs. In this situation it is important to reduce the risks of barotrauma and pneumothorax caused by high inflation pressures by balancing the controlled respiratory rate.
Chapter 3

and ITV to ensure inspired airway pressure does not exceed 30–35 cm H\textsubscript{2}O. Promoting effective patient ventilation therefore requires assessment, monitoring, communication and collaboration with the patient, nurse, intensivist (anaesthetist) and physiotherapist to promote optimum lung function, and with the dietitian to promote optimum nutrition to support the patient’s metabolic requirements and promote recovery (Woodrow, 2012). A summary of the risk assessment and management of patients such as Jenny is illustrated in Table 3.8.

<table>
<thead>
<tr>
<th>Risk assessment</th>
<th>Nursing interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Airway</strong></td>
<td>• Look for evidence of distress and agitation such as coughing and biting on the tube, assess the patient’s sedation score and reassure. If the patient continues to be distressed, there is a higher risk of unplanned extubation and/or trauma to the patient’s airways. If necessary, increase the sedation according to the prescribed guideline until the patient is comfortable.</td>
</tr>
<tr>
<td>• Risk of the endotracheal tube/tracheostomy (tube) occluding due to poor humidification, the patient biting down on the tube and/or secretions.</td>
<td>• Humidification of the airways can be achieved by:</td>
</tr>
<tr>
<td>• Risk of airway irritation.</td>
<td>o heat/moisture exchange (HME) filters that are attached to the ventilator circuit close to the endotracheal tube;</td>
</tr>
<tr>
<td>• Risk of the tube becoming dislodged.</td>
<td>o hot water humidifiers (37°C);</td>
</tr>
<tr>
<td>• Risk of unplanned extubation.</td>
<td>o cold water humidifiers.</td>
</tr>
<tr>
<td><strong>Breathing</strong></td>
<td>• Narrowing or occlusion of the patient’s airway can be identified by an increase in the inspired airway pressure and evidence of patient agitation, rattling/bubbling on chest auscultation.</td>
</tr>
<tr>
<td>• Risk of airways becoming partially occluded leading to a rise in airway pressure and ineffective ventilation.</td>
<td>• Endotracheal suction is used to remove secretions in the trachea but should only be performed when there is evidence of the above. Suction can be painful, distressing and increase the risk of infection and trauma to the airways.</td>
</tr>
<tr>
<td>• Risk of air leak due to poor connections.</td>
<td>• A loose connection can be identified by a reduction in inspired airway pressure, tidal volume and reduction in ( \text{SpO}_2 ).</td>
</tr>
<tr>
<td>• Risk of inappropriately set alarm parameters.</td>
<td>• Assess respirations, inspired and expired tidal volumes and airway pressure, ( \text{SpO}_2 ) and ABG analysis if the patient’s condition changes.</td>
</tr>
<tr>
<td>• Risk of ventilator-associated lung injury and ventilator-associated pneumonia (VAP).</td>
<td>• Set alarm limits to between 5 and 10 marks above and below the prescribed range and assess the patient hourly.</td>
</tr>
<tr>
<td></td>
<td>• Adhere to the ventilator bundle (see Concept summary: Care bundles).</td>
</tr>
</tbody>
</table>
Circulation

- Risk of impaired circulation and cardiac function: MIV increases venous return pressure because the right side of the heart has to pump against a higher alveolar pressure, thus raising the patient's CVP. Left ventricular cardiac output is reduced due to more blood staying in the venous circulation. Thus the patient is at risk of hypotension and oedema.
- Risk of liver dysfunction leading to clotting disorders, immunosuppression and reduced albumin production.

Disability

- Inability to communicate verbally due to the endotracheal tube and sedation.
- Risk of pain.
- Risk of poor skin integrity, dry eyes and mouth.
- Risk of anxiety, delirium and/or boredom.

- Assess the patient’s vital signs for evidence of impaired circulation using continuous monitoring: heart rate and rhythm; BP; CVP; chest X-ray; signs of venous thrombosis; urine output, which should be ≥0.5 ml/kg/hr (> about 30 ml/hr).
- Adhere to the ventilator bundle to reduce the risk of VAP.
- Assess the patient for signs of peripheral oedema, bruising.
- Assess blood results including: serum electrolytes; urea and creatinine; liver function tests; clotting.
- Assess and screen for sepsis daily (Chapter 7).

- When appropriate, encourage the patient to use non-verbal means of communication, picture cards and alphabet cards. Use eye contact and explain all procedures before they are attempted.
- Assess the patient’s pain using non-verbal cues and pain scores and manage appropriately.
- Assess the integrity of the patient’s eyes and mouth hourly and manage appropriately according to each patient’s needs.
- Adopt the Institute for Healthcare Improvement (IHI, 2009) care bundle for pressure ulcer prevention: risk assess on admission; reassess daily: inspect skin, manage moisture on the skin, optimise nutrition and hydration, minimise pressure through positioning.
- Help the patient to be orientated to night and day, and assess for signs of delirium (Chapter 8).
- Encourage family-centred care and patient-focused care.
- Encourage the patient to be involved in decisions and, where possible, life outside the unit.

(Continued)
Concept summary: Care bundles

Evidence-based practice is concerned with ensuring that the best available evidence is applied to practice. One method for achieving this is through the use of care bundles. Care bundles are a group of evidence-based interventions that, when combined, provide the most clinically effective method for reducing risk and improving patient outcome (Fulbrook and Mooney, 2003). The ventilator care bundle is an example of how combining selective interventions appears to have reduced the incidence of ventilator-acquired pneumonia (Lawrence and Fulbrook, 2011; Eom et al., 2014). The bundle recommended by the IHI (2014) combines the following five elements.

- Elevation of the head of the bed to 30–45%.
- Periodic interruption of the patient’s sedation and daily assessment of the patient’s readiness for extubation.
- Peptic ulcer disease prophylaxis.
- Venous thromboembolism prophylaxis.
- Daily oral care with chlorhexidine.

Case study: Jenny’s ventilation with MIV

Jenny continued to be ventilated, sedated and paralysed for a further 12 hours until her clinical condition improved and her neuromuscular blocking agent was discontinued. Jenny’s sedation was ceased and she was assessed:

A: Airway maintained through an oral endotracheal tube.
   Responding to commands.

<table>
<thead>
<tr>
<th>Risk assessment</th>
<th>Nursing interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposure and safe environment</td>
<td>• Risk assess and manage the patient with due regard to the ventilator bundle and risk assessment for sepsis.</td>
</tr>
<tr>
<td>• Risk of infection associated with the use of invasive procedures.</td>
<td>• Assess noise levels and reduce noise pollution where possible. Reorientate the patient to their environment and offer reassurance when appropriate.</td>
</tr>
<tr>
<td>• Risk of noise and the environment disturbing sleep and rest.</td>
<td></td>
</tr>
</tbody>
</table>

Table 3.8: Risk assessment and plan of care for a ventilated patient

Table 3.8 (Continued)
The patient who needs respiratory support

Chapter summary

In this chapter you have been introduced to patients who need advanced respiratory support. The technology and assessment strategies for patients in these situations are often complex and the patient’s condition can change suddenly. We have seen how Jenny’s condition initially deteriorated but continuous assessment of her condition alerted staff to changes in her condition and she received intensive care. To complete her story, Jenny’s condition improved sufficiently for her to be transferred to a general ward and she was discharged from hospital five days later. She decided to separate from her husband and has managed to give up smoking and make a new life for herself. She can still remember her time in the intensive care unit and is determined to improve how she manages her asthma in order to reduce the risk of readmission.

(Continued)
The important messages to gain from this chapter are as follows.

- Always begin by assessing the patient’s airway, breathing and circulation, disability and environment, and you will always be able to prioritise care and communicate your concerns.
- Interpretation of the patient’s condition through blood gas analysis means much more if the results are assessed in the context of the patient’s story.

**Activities: brief outline answers**

**Activity 3.1: Decision making (pages 63–4)**

*Using the step-by-step guide in Table 3.3, what can you interpret from the arterial blood gas result?*

These are the first set of ABG results.

- Joseph was showing signs of hypoxaemia on 60% oxygen.
- pH 7.07: shows evidence of acidosis.
- PaCO\(_2\): 14.1 kPa shows evidence of respiratory acidosis.
- HCO\(_3\): 20.5 mmol/L shows evidence of metabolic acidosis also.
- Joseph shows signs of both a respiratory and metabolic acidosis.
- This result in the context of his clinical situation are consistent with a period of inadequate oxygenation and tissue perfusion related to his cardiac arrest. Re-establishment and maintenance of Joseph’s respiration and circulation will provide an opportunity for acid-base balance to be restored.

*Using the step-by-step guide in Table 3.3, what can you interpret from Joseph’s second arterial blood gas result?*

- Joseph’s oxygen levels had improved however his Pa\(_O_2\) and Sp\(_O_2\) are still below the accepted level.
- His pH of 7.20 is still showing signs of acidaemia.
- PaCO\(_2\): 6.70 kPa still shows evidence of respiratory acidosis but has improved from his previous results.
- HCO\(_3\): 22.5 mmol/L shows evidence of a resolving metabolic acidosis compared to the previous result.
- Joseph still shows signs of both a respiratory and metabolic acidosis, however, this is not as severe as his post cardiac arrest results.
- Joseph’s ABGs still indicate evidence of type II respiratory failure which has now been complicated by a diagnosis of a STEMI.

**What are your priorities of care for this patient?**

- Joseph needs to receive support for his respiratory failure and should be assessed to determine the most suitable treatment plan. This may be a combination of nebulised short-acting beta agonist, short-acting muscarinic antagonist and intravenous antibiotics. He should be encouraged to sit up in the most comfortable breathing position and be assessed for NIV. Following an assessment from the critical care outreach team, Joseph was transferred to ICU for NIV and was commenced on bilevel positive airways pressure.
- Your role is to risk assess the patient and reassure him, and if he is commenced on NIV, to support him to promote his comfort.
- Joseph will require continuous assessment of his respiratory and cardiac function with a view to reducing the NIV support over the next few hours if his condition continues to improve.
- He will need continued support and reassurance to maximise the effect of the respiratory support and his cardiovascular status should be monitored to assess for signs of deterioration following his cardiac event.
The patient who needs respiratory support

Further reading


This book offers practical help with learning how to use the technology when involved in the care of level 2 patients.


This document gives you a helpful introduction to some of the drugs used to promote safety and pain relief for patients with mechanical ventilation. It also gives you examples of some of the assessment tools available for monitoring pain and sedation.

Useful websites


The Intensive Care Society site provides access to relevant innovations and standards that relate to the care of patients who are critically ill. The website is multidisciplinary and offers information to patients and relatives in user-friendly guides. A revised edition of sedation guidance is now available on this website.

[www.ihi.org](http://www.ihi.org)

The Institute for Healthcare Improvement website offers evidence-based and practical ways in which to provide safe and effective care for patients with acute and critical care needs.
Hi! I have recently been diagnosed with asthma, which has surprised me as my symptoms were that of a persistent cough and tight chest rather than a wheeze. My symptoms have been somewhat difficult to manage, but are now improving, and I have been reviewed by my GP and attended the asthma clinic on a regular basis.

(Patient voice)

Respiratory assessment may on the surface appear as simple observation that nurses can follow guidelines on. However, on greater analysis many of the components of a respiratory assessment can appear in both health and illness depending on the context, so presence or absence is not necessarily something that can just be simply observed for. Therefore, when student nurses are learning this skill it is useful to reflect on episodes of care with more experienced colleagues.

(Practitioner voice)
INTRODUCTION

When we are healthy, we take our breathing for granted, never fully appreciating that our lungs are essential organs for life. But when our lung health is impaired, nothing else but our breathing really matters. (Forum of International Respiratory Societies, 2013: 4)

Worldwide, the mortality and morbidity associated with lung disease is quite simply staggering, and asthma and COPD are two of five respiratory conditions which contribute to this global burden. It is estimated that 235 million people suffer from asthma and more than 200 million people have COPD (Forum of International Respiratory Societies, 2013: 4). According to the British Lung Foundation (2015), 700,000 hospital admissions are associated with lung disease each year and this accounts for 8% of all hospital admissions. COPD is seen to be one of the leading causes of death within the UK (30,000 deaths/year) and although asthma mortality is considerably lower (1,200 deaths/year) it is the highest in Europe and significant for a controllable disease. The two diseases may cause similar symptoms; however, the table (24.1) below may assist in differentiating between asthma and COPD.

Table 24.1 Clinical features of asthma and COPD (NICE, 2010)

<table>
<thead>
<tr>
<th></th>
<th>COPD</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoker or ex-smoker</td>
<td>Nearly all</td>
<td>Possibly</td>
</tr>
<tr>
<td>Symptoms under age 35</td>
<td>Rare</td>
<td>Often</td>
</tr>
<tr>
<td>Chronic productive cough</td>
<td>Common</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Breathlessness</td>
<td>Persistent and progressive</td>
<td>Variable</td>
</tr>
<tr>
<td>Night-time waking with breathlessness and/or wheeze</td>
<td>Uncommon</td>
<td>Common</td>
</tr>
<tr>
<td>Significant diurnal or day-to-day variability of symptoms</td>
<td>Uncommon</td>
<td>Common</td>
</tr>
</tbody>
</table>

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This chapter covers how to care for an adult with a respiratory condition and its aim is to provide you with a knowledge and understanding of what you should consider when assessing a patient, how to undertake an accurate respiratory assessment and your role as a nurse in the provision of timely and appropriate interventions both in the acute and community setting. Respiratory assessment is fundamental in the provision of effective nursing care and it is essential that you are able to recognise and differentiate between the norm, and the signs and symptoms associated with respiratory deterioration.

Specific focus will be placed on two of the most common respiratory conditions that you may encounter in practice: asthma and COPD. The underlying pathophysiology, clinical characteristics and assessment of these conditions will be discussed in more detail and clinical investigations will be considered. Case studies and scenarios will be used to further support learning and additional resources accompanying this chapter can be found on the companion website.

**ACTIVITY 24.1: CRITICAL THINKING**

Have you had the opportunity within clinical practice to undertake a respiratory assessment? What things do you need to consider before doing so? Reflect on your experience and knowledge gained so far and discuss this with your mentor.
ANATOMY AND PHYSIOLOGY

Anatomically, the respiratory system is made up of the upper and lower respiratory tract. The upper consists of the nose, mouth, pharynx and larynx whereas the lower consists of the trachea, bronchi, respiratory bronchioles, alveolar ducts and alveoli (Tu et al., 2013).

Air entry occurs via the nose through the external nares or nostrils as they are known. When air enters the nasal cavities it is warmed, filtered and humidified by a system of nasal turbinates and a dense capillary network, which ensures further mixing of inspired air as it passes onto the pharynx. The mucous membrane lining the nasal cavity traps debris, and hair-like projections known as cilia propel dust particles towards the pharynx where this is swallowed or expectorated. The pharynx, or throat as it is more commonly known, sits behind the nasal cavity and mouth and extends to a point where the larynx and oesophagus divide. The larynx can be found between the pharynx and trachea and sits anteriorly to the oesophagus. A flap of cartilage known as the epiglottis is attached to the entrance of the larynx and on swallowing, the entrance to the larynx is blocked and food is directed into the oesophagus.

The lungs sit within the thoracic cavity and the heart, major vessels and anatomical structures of the mediastinum can be found between them. A protective membrane lines the wall of the thoracic cavity (parietal pleural membrane) and another surrounds the lungs (visceral pleural membrane). The narrow cavity that exists between the two layers is known as the pleural space and it contains a small amount of fluid which lubricates the two surfaces allowing them to move freely against one another during breathing. The diaphragm separates the thorax and the abdominal cavity and its action is essential to the inflation and deflation of the lungs. On inspiration, the diaphragm contracts and flattens and the ribs are pulled upwards and forward by the contraction of the external intercostal muscles. As the chest dimensions increase, intrathoracic pressure changes and air is drawn into the lungs. On expiration, the diaphragm relaxes and air flows out of the lungs and intrathoracic pressure returns to normal.

The lower respiratory tract is commonly known as the tracheobronchial tree, which subdivides into a series of branches: primary, secondary and tertiary bronchi, which increasingly narrow and shorten. In its simplest sense, this network allows air to be conducted or transported to the alveoli via the trachea, bronchi and bronchioles. As their primary function is that of conduction they are not involved in gas exchange, and this is often referred to as the anatomical dead space (Creed and Spiers, 2010).

Figure 24.1  Organs of the respiratory system

Source: Boore et al. (2016). Illustrated by Shaun Mercier, © SAGE Publications.
CONTROL OF BREATHING

The respiratory centres are found within the medulla oblongata and pons, which form part of the brainstem. They control the rate and depth of breathing and the rate at which this occurs will change according to oxygen demand and the body’s needs.

Gas exchange

Respiration is the process by which oxygen (O$_2$) is delivered to the tissues (cells) from the atmosphere and carbon dioxide (CO$_2$) is then removed. There are four distinct phases involved in this process, although only two relate to the respiratory system:

- pulmonary ventilation
- external respiration
- oxygen transport
- internal respiration

Pulmonary ventilation

Pulmonary ventilation is a mechanical process that relates to the movement of oxygen into and carbon dioxide out of the lungs, and in order for this to occur there needs to be a change in intra-pleural pressure (figure 24.2).

Figure 24.2  Inspiration and expiration

*Source: Boore et al. (2016). Illustrated by Shaun Mercier, © SAGE Publications.*

At rest, atmospheric and intrathoracic pressures are equal but during inspiration the pressure falls (sub-atmospheric) as the thorax expands and the diaphragm contracts, and air flows from an area of high to low pressure until an equilibrium is reached and inspiration ceases. Expiration is a passive
process and is due to the elastic recoil of the lungs which forces air out of the lungs and back into the atmosphere (Peate and Nair, 2011).

![Diagram of internal respiration](image1)

**Internal respiration**
- Inhaled $O_2$
- Exhaled $CO_2$
- Alveolus
- Deoxygenated blood from the right side of the heart (High in $CO_2$ and low in $O_2$)
- Capillary from pulmonary arteriole
- $PO_2$ 40 mmHg
- $PCO_2$ 44 mmHg
- Capillary
- Red blood cell
- Oxygenated blood going to the left side of the heart (High in $O_2$ and low in $CO_2$)

![Diagram of external respiration](image2)

**External respiration**
- Movement of $O_2$ from blood
- Oxygenated blood $→$
- $O_2$
- Capillary
- Tissue cells
- Movement of $CO_2$ from tissue
- $CO_2$
- Deoxygenated blood

**Figure 24.3** Internal and external respiration
Source: Boore et al. (2016). Illustrated by Shaun Mercier, © SAGE Publications.

**External respiration**
An adult lung contains approximately 300 million alveoli (Peate and Nair, 2011; Creed and Spiers, 2010) each of which is 0.2 mm in diameter and which are surrounded by a network of capillaries (Figure 24.4).

![Diagram of alveoli](image3)

**Figure 24.4** Alveoli
Source: Boore et al. (2016). Illustrated by Shaun Mercier, © SAGE Publications.
External respiration (Figure 24.3) is the process by which oxygen diffuses across the alveolar capillary membrane and into the pulmonary circulation. Gaseous exchange and diffusion occur because of the concentration gradient that exists between the alveoli and the pulmonary capillary. Deoxygenated blood returning to the lungs, from the right side of the heart, has a higher content of carbon dioxide and lower content of oxygen than that of alveolar air and oxygen moves accordingly into the blood and carbon dioxide moves into the alveoli. The effectiveness of external respiration is dependent on an adequate supply of both oxygen and blood being delivered to the alveoli capillary membrane (Peate and Nair, 2011; Creed and Spiers, 2010).

Breathlessness and the load capacity drive relationship

In health, the complex mechanisms described above maintain respiration and ventilation over a wide range of activities. However, during illness different factors affect the ability of the lung to function and maintain adequate gas exchange. Imbalances may lead to respiratory failure. According to Hess and Kacmarek (2014) respiratory failure can either be hypoxaemic (failure to oxygenate i.e. low \( \text{PaO}_2 \)) or hypercapnic (inadequate ventilation i.e. high \( \text{PaCO}_2 \)).

The ventilatory system can be described as a ‘pump’ consisting of the diaphragm and chest wall muscles, and the neural control of these muscles. If any of the above components (pump) deteriorate, hypercapnic respiratory failure may result.

According to Moxham and Jolley (2009) the respiratory load is the pressures that need to be generated for the lungs to expand and achieve ventilation. If the patient has an underlying condition such as lungs that are inelastic (fibrosed, pulmonary oedema), or the lungs are hyperinflated, or airways are obstructed by conditions such as asthma or cystic fibrosis, the respiratory muscles have a greater load to overcome. Chest wall deformities, including obesity and ascites, will also increase respiratory load.
If the load (Figure 24.5) on the respiratory muscles is increased, respiratory muscles require additional effort to breathe adequately. Hess and Kacmarek (2014) also describe secretions, mucosal oedema and bronchospasm among factors that cause excessive ventilatory muscle load. Some factors described above are reversible, such as management of secretions and bronchospasm by appropriate therapies, physiotherapy or patient positioning to reduce load on respiratory muscles.

Respiratory capacity is the ability of the respiratory muscle ‘pump’ to function (Moxham and Jolley, 2009). Respiratory muscle function is reduced if the patient’s lungs are hyperinflated, due to respiratory muscle fatigue, which also impairs gas transfer. Hess and Kacmarek (2014) describe several factors that may result in inadequate muscle function. These include electrolyte imbalances, malnutrition, pharmacologic agents, muscle atrophy and fatigue.

Drive can be abnormally high or low. Neural drive can be abnormally high, for example, in patients with COPD, as they have a flattened diaphragm and reduced muscle capacity, which results in dyspnoea. Conversely, neural transmission can be impaired and also cause respiratory failure, for example, in patients with a spinal cord injury. Central neural drive may be impeded by pharmacologic agents (sedatives or narcotics), hypothyroidism or brainstem injury (Hess and Kacmarek, 2014).

**Activity 24.2: Critical Thinking**

Consider a patient that you have been involved in providing care for in relation to their respiratory history and symptoms. Are there any elements that you can optimise with regard to the load/capacity/drive relationship? What strategies would you use?

**Respiratory Assessment**

Higginson and Jones (2009) suggest: inspection, palpation, percussion and auscultation. The ‘Inspection’ stage should include a ‘Look, Listen and Feel’ approach. Palpation, percussion and auscultation are all advanced skills that require specific training.

**Look:** The practitioner can gain a lot of assessment data as they approach the patient and observe them.

**Colour** – What do you notice about the patient’s skin and mucus membrane colour? Is cyanosis present? – this may provide an indication of haemoglobin saturation. However, presence of cyanosis is a late sign that a patient has low oxygen saturations; conversely, cyanosis may be present chronically in those with long-term lung or heart conditions (Jevon and Ewens, 2007). Cyanosis should be assessed centrally – such as lips and buccal mucosa – as peripheral cyanosis may also be due to poor perfusion, not necessarily a respiratory cause.

**Ability to speak** – Can the patient talk to you in full sentences without appearing breathless? Increased effort/ inability to speak, use of short sentences or monosyllables may indicate difficulty in breathing.

**Use of accessory muscles** – Do you notice use of abdominal muscles, sternomastoid and scalene muscles? This is normally only seen in someone with respiratory distress or increased exertion. In health or with normal activity levels, the diaphragm and intercostal muscles facilitate the patient achieving adequate volumes without the use of additional muscles.
Rate, rhythm and depth of breathing – What do you observe about how the patient is breathing? What is the respiratory rate? Above or below the normal range of 12–18 bpm can indicate respiratory difficulty and would normally trigger an Early Warning System (EWS). The rhythm of breathing can be observed: is the patient’s breathing shallow, normal or deep? Shallow or deep breathing needs to be taken in the context of other parameters; for example, deep breathing may be normal during exercise and shallow breathing normal during sleep – but both can also be signs of respiratory distress.

In health, people breath in (Inspiration) and out (Expiration) at a ratio of 1:2 (Higginson and Jones, 2009). Breathing in for longer (with a short expiration) or breathing out for longer (a longer expiration) may indicate respiratory distress or illness.

Chest movement – What do you observe about the how chest is moving? In health, the chest moves symmetrically, therefore asymmetry may be a sign of pathology. Do you see any paradoxical movements such as chest moving opposite the abdomen or sternum moving inwards? These are not seen in health.

According to Welch and Black (2017), not being able to talk in sentences, sweating or cold clammy skin, altered level of consciousness (including restlessness and confusion) may be due to a greatly increased effort in breathing or inadequate respiratory support.

Does the patient have physical signs of a chronic lung condition? These include a ‘barrel shaped chest’, this may be noted as the nurse observes chest movement, the anterior–posterior diameter is enlarged and clubbing of the fingers (Moore and Woodrow, 2009).

Do you notice additional factors that may be observed during respiratory assessment, e.g. productive cough, pursed lip breathing and nasal flaring? Does the patient need to sit upright and lean forward to assist their breathing? Again, these are not present in health.

Listen: Can you hear added sounds as you assess the patient’s breathing? Turbulent airflow causes added sounds. Jevon and Ewens (2007: 37) provide a summary of those sounds that are audible without the aid of auscultation:

- **Stridor**: ‘croaking’ respirations which are louder during inspiration; caused by laryngeal or tracheal obstruction, e.g. foreign body, laryngeal oedema or laryngeal tumour.
- **Wheeze**: noisy musical sound caused by turbulent flow of air through narrowed bronchi or bronchioles, more pronounced on expiration; causes include asthma and chronic obstructive pulmonary disease (COPD).
- **‘Ratty’ chest**: e.g. chest infection, pulmonary oedema and sputum retention.
- **Gurgling**: caused by fluid in the upper airway.
- **Snoring**: snoring sounds may be associated with the tongue blocking the airway in an unconscious patient.

It is of note that not all respiratory difficulties are accompanied by a sound. A completely silent chest/breathing may indicate an obstructed airway or absent movement of air and this is a medical emergency.

Feel: With permission, by placing both hands gently on either side of the patient’s chest the nurse may ascertain additional information, such as sputum retention, surgical emphysema and rise, fall and depth of breathing (Higginson and Jones, 2009). Again, this is an element of respiratory assessment with which senior nurses, physiotherapists or doctors can assist you in interpreting findings.
In addition, the following parameters might be useful to add for completeness:

The British Thoracic Society (BTS) (O’Driscoll et al., 2017) recommends utilising $\text{SpO}_2$ as the 5th vital sign. However, it is important that those monitoring $\text{SpO}_2$ are aware of the limitations and factors that can affect accuracy: $\text{SpO}_2$ monitoring does not provide data on the patient’s $\text{pH}$, $\text{PCO}_2$, or haemoglobin levels. Therefore, a normal saturation level does not mean a patient will not require further tests such as arterial blood gases (ABG).

According to BTS (O’Driscoll et al., 2017), poor peripheral perfusion, skin pigmentation, motion artefact and the site of the probe can all affect accuracy of readings. Probe placement on the ear or finger is therefore preferable to toes. Before placing the saturation probe, always remove nail varnish or false nails if present. If the patient has been exposed to carbon monoxide (from smoke inhalation) or has methaemoglobinemia (after smoking), the $\text{SpO}_2$ reading may appear higher than it really is, and therefore is falsely reassuring. This is because $\text{COHb}$ and methaemoglobin are not distinguishable from oxyhaemoglobin by the oximeter.

Higginson and Jones (2009) indicate respiratory assessment should form part of comprehensive assessment and, therefore, also include temperature, pulse and blood pressure. Other sources of data such as drug charts, fluid balance and medical notes may also add to the overall assessment. Moore and Woodrow (2009) suggest assessing the patient’s cough and any sputum produced. Sputum that is coloured, frothy, copious or thick may indicate pulmonary oedema or lung pathology.

Once a basic respiratory assessment has been undertaken the nurse must escalate concerns to senior staff/medical staff and document findings. Depending on the severity of assessment findings this may mean a call for a medical emergency team, or SBAR call for a raised EWS. It may be necessary for additional investigations to be carried out, e.g. ABG, chest x-ray.

**ASTHMA**

The prevalence of asthma continues to increase worldwide and despite advances in treatment, there has been a concomitant increase in mortality. In the UK, 5.4 million people are currently receiving treatment and of these, 80% are adults. The cause of this increase in prevalence is not well understood, but one hypothesis (hygiene) suggests that the western lifestyle has reduced our exposure to pathogens and helminths in early childhood and that our relationship with cleanliness predisposes us to allergies later in life. Factors that may trigger symptoms and individual response vary, and these may include genetic predisposition, allergens (pollen, dust mites, fungal spores, and dander), environmental factors, diet, stress and exercise (Forum of International Respiratory Societies, 2013).

In 2014, the National Review of Asthma Deaths (NRAD) published a report ‘Why asthma still kills’. Despite advances in modern medicine and the development of evidence-based guidelines, the review found that ‘major preventable factors were identified in two thirds of all asthma deaths’. There is no gold standard definition of asthma; however, the Global Initiative for Asthma (GINA) (2017: 14) suggests that:

> Asthma is a heterogeneous disease usually characterised by chronic airway inflammation, it is defined by a history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and intensity and variable airflow limitation.

Defined as a chronic inflammatory disorder, asthma is characterised by mucosal inflammation, hyperresponsiveness and narrowing of the airways. Episodic and variable in presentation, asthma is a serious condition and can be difficult to treat due to psychosocial issues: patient compliance
with treatment, denial, and failure to monitor own symptoms, which can lead to a failure in management (Creed and Spiers, 2010). Causes of asthma and individual response may vary and it can either be atopic or non-atopic in presentation. **Atopic** asthma usually starts in childhood and can be associated with certain identifiable triggers (e.g. dust, pollen, dander) and family history (Kaufman, 2011) whereas non-atopic asthma tends to have a later onset and may develop in adults as a consequence of infection.

**Pathophysiology**

![Pathology of asthma](Science Photo Library)

Asthma can affect the trachea, bronchi and bronchioles and symptoms are caused by reversible changes in the airways. Narrowing of the bronchial lumen as a result of epithelial damage, overproduction of mucous and oedema cause an increase in airflow resistance (difficulty breathing out of the lungs) within the airways and the patient will often present with signs of dyspnoea and wheezing (Cohen and Hull, 2015). The epithelial layer that lines the trachea, bronchi and bronchioles can become damaged and peel away (Kaufman, 2011) and ‘shedding’ of this protective layer can lead to hyperresponsiveness of the airway. Bronchospasm, which is defined as a sharp contraction of the bronchial smooth muscle, causes the airways to narrow, capillaries to leak and oedema, which impairs mucus clearance and increases mucous production, which can cause ‘plugs’ that may lead to occlusion of the airway (Figure 24.6). Without proper treatment, airway remodelling (structural changes)
can occur within the lower respiratory tract and this can be associated with a progressive loss of lung function and fibrosis.

**WHAT’S THE EVIDENCE? 24.1**

The recently published NICE (2017) guidelines – Asthma: diagnosis, monitoring and chronic asthma management aims to improve patient diagnosis and control of symptoms.

- Reflect upon the care you have provided to a patient living with asthma.
- Look at the guidelines relating to diagnosis and consider how you can apply your findings to practice.

**Structured clinical assessment**

The British Thoracic Society and Scottish Intercollegiate Guidelines Network (SIGN) (2016) and National Institute for Health and Care Excellence (NICE) (2017) have devised a series of guidelines relating to the aetiology of asthma and its management. This stepwise approach provides clinicians with an evidence-based guide on the best assessment strategies and available treatment. According to BTS (O’Driscoll et al., 2017), there is no single diagnostic test and diagnosis should be based on clinical assessment and objective tests that assess variable airflow limitation and the presence of airway inflammation (spirometry, peak flow, fractional exhaled nitric oxide (FeNO), **bronchodilator reversibility test**, testing of atopic status and sputum eosinophils). Undertaking a structured clinical assessment to assess the initial probability of asthma should be based on Figure 24.7 on the next page.

**Lung function tests**

Spirometry is a relatively simple test which assesses lung function and can be used to differentiate between obstructive and restrictive lung disease by measuring forced vital capacity (FVC) and forced expiratory volume (FEV) over a second (Kaufman, 2011). Tests are performed by measuring the volume of air expelled from the lungs following maximum inspiration of air during a single breath: this is done on three consecutive occasions and the highest reading is then recorded (reproducibility of data). The ratio normally declines with age and so actual and predicted measures are used in assessment in spirometry. Poor technique and misinterpretation of results can lead to a wrong diagnosis and inappropriate treatment. It is, therefore, essential that healthcare practitioners have received appropriate training and are competent in the technique.

Peak expiratory flow (PEF) monitoring is another method used to assess lung function and uses a small hand-held device which measures airflow through the airways on expiration. Although dependent on technique and patient effort, PEF assesses the rapidity of the flow rate during a forced expiration (best of three) and can be used to assess the effectiveness of bronchodilators both pre- and post-treatment. Multiple measurements can be taken over several weeks to assess the variability of airflow using electronic meters and patient diaries; however, reliability is based on individual compliance (BTS and SIGN, 2016).
Algorithm A Initial clinical assessment for adults, young people and children with suspected asthma

Adults, young people and children with symptoms of asthma

Take a structured clinical history. Specifically check for:
- wheeze, cough or breathlessness, and any daily or seasonal variation in these symptoms
- any triggers that make symptoms worse
- a personal or family history of atopic disorders

Examine people with suspected asthma to identify expiratory polyphonic wheeze and signs of other causes of respiratory symptoms, but be aware that even if examination results are normal the person may still have asthma

Do not use symptoms alone without an objective test to diagnose asthma
Do not use a history of atopic disorders alone to diagnose asthma

Treat people immediately and perform objective tests if the equipment is available and testing will not compromise treatment

If objective tests cannot be done immediately, carry them out when acute symptoms have been controlled and advise patients to contact their healthcare professional immediately if they become unwell while waiting to have objective tests

Be aware that the results of spirometry and FeNO tests may be affected by treatment with inhaled corticosteroids

Refer people with suspected occupational asthma to an occupational asthma specialist

Children under 5

Treat symptoms based on observation and clinical judgement, and review the child on regular basis.
If they still have symptoms when they reach 5 years, see algorithm B for objective tests

Children and young people aged 5 to 16

See algorithm B for objective tests

Check for possible occupational asthma by asking employed people:
- Are symptoms better on days away from work?
- Are symptoms better when on holiday?
Make sure answers are recorded for later review

Use skin prick tests to aeroallergens or specific IgE tests to identify triggers after a formal diagnosis of asthma has been made

Adults aged 17 and over

Acute symptoms at presentation

Do not offer the following as diagnostic tests for asthma:
- skin prick tests to aeroallergens
- serum total and specific IgE
- peripheral blood eosinophil count
- exercise challenge (to adults aged 17 and over)

See algorithm C for objective tests

Figure 24.7 Asthma management (BTS and SIGN, 2016).

Reproduced from BTS/SIGN British Guideline on the management of asthma, with kind permission of the British Thoracic Society.
ACTIVITY 24.3: CRITICAL THINKING

We have considered spirometry and peak flow but what other objective tests are available in the diagnosis of asthma? What are the benefits of these tests? How will the results of these tests influence patient care?

Consider utilising the NICE 2017 guidelines when formulating your answers to these questions.

Pharmacological management

The ultimate goal of asthma management is disease control so that patients can remain symptom free and be able to lead a normal life (refer to Figure 24.8).

Complete control of asthma is defined as:

- no daytime symptoms
- no night-time awakening due to asthma
- no need for rescue medication
- no asthma attacks
- no limitations on activity including exercise
- normal lung function (in practical terms FEV1 and/or PEF>80% predicted or best)
- minimal side effects from medication.

Asthma management (BTS and Sign, 2016) Reproduced from BTS/SIGN British Guideline on the management of asthma, with kind permission of the British Thoracic Society.

Pharmacologically a step-wise, age-based approach is currently used to guide asthma management (Figure 24.8). Depending on the severity of the disease, treatment should be commenced at the most appropriate level with the aim to achieve early control. Maintaining control can then be achieved by increasing or decreasing the treatment as necessary according to patient response (BTS and SIGN, 2016). Asthma medication can be divided into two groups: ‘preventers’ (reduces inflammation and swelling) and ‘relievers’ (relax airways). Patients may be prescribed one or more of the following:

- Inhaled corticosteroids (ICS): Steroids work by reducing inflammation and swelling in the airways and helping to control symptoms and prevent attacks, e.g. budesonide, beclomethasone
- Leukotriene receptor antagonists (LTRAs): Reduce the body’s response to allergens and help relax the airways, e.g. montelukast
- Short acting beta agonists: Known as rescue medicines as they act within minutes providing quick relief. They help to relieve bronchospasm but do not reduce swelling or inflammation within the airways, e.g. Ventolin, Albuterol
- Long acting beta agonists (LABA): These are used to provide long-term control of asthma symptoms rather than quick relief and they are in combination within inhaled steroids, e.g. Symbicort, Serevent.

ACTIVITY 24.4: REFLECTION

Environmental exposure, pregnancy and obesity can all lead to the exacerbation of asthma. What non-pharmacological strategies are available? Make a list and discuss this further with your mentor.
### Figure 24.8  Asthma management for adults (BTS and Sign, 2016)

Reproduced from BTS/SIGN British Guideline on the management of asthma, with kind permission of the British Thoracic Society.

<table>
<thead>
<tr>
<th>Asthma - suspected</th>
<th>Asthma - diagnosed</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diagnosis and assessment</strong></td>
<td><strong>Evaluation:</strong></td>
</tr>
<tr>
<td></td>
<td>• assess symptoms, measure lung function, check inhaler technique and adherence</td>
</tr>
<tr>
<td></td>
<td>• adjust dose</td>
</tr>
<tr>
<td></td>
<td>• update self-management plan</td>
</tr>
<tr>
<td></td>
<td>• move up and down as appropriate</td>
</tr>
</tbody>
</table>

**Asthma – suspect**

- Consider monitored initiation of treatment with low-dose ICS

**Asthma – diagnosed**

**Short acting $\beta_2$ agonists as required – consider moving up if using three doses a week or more**

**Regular preventer**

- Infrequent, short-lived wheeze
- Low-dose ICS

**Initial add-on therapy**

- Add inhaled LABA to low-dose ICS (normally as a combination inhaler)

**Additional add-on therapies**

- No response to LABA – stop LABA and consider increased dose of ICS  
  - If benefit from LABA but control still inadequate – continue LABA and increase ICS to medium dose  
  - If benefit from LABA but control still inadequate – continue LABA and ICS and consider trial of other therapy – LTRA, SR theophylline, long-acting muscarinic antagonist (LAMA)

**High – dose therapies**

- Consider trials of:  
  - Increasing ICS up to high dose  
  - Addition of a fourth drug, eg LTRA, SR theophylline, beta agonist tablets, LAMA

**Continuous or frequent use of oral steroids**

- Use daily steroid tablet in the lowest dose providing adequate control  
- Maintain high-dose ICS  
- Consider other treatments to minimize use of steroid tablets

**Refer patient for specialist care**
Health promotion

Health promotion and patient education play a key role in asthma management by empowering patients to take control and responsibility for their condition. As nurses, we can play a pivotal role in promoting patient self-management by using different approaches and resources that best support patients:

- personalised asthma action plans
- self-management education
- monitoring inhaler technique and adherence with asthma treatment
- lifestyle changes: exercise, advice on diet and weight loss interventions, cessation of smoking
- non-pharmacological management: breathing exercise programmes, employing methods for reducing dust mites within the home
- online resources/helplines
- support groups.

ACTIVITY 24.5: REFLECTION

Consider some of the current guidelines with regards to health promotion using the following weblinks:

- British Lung Foundation: www.blf.org.uk
- Asthma UK: www.asthma.org.uk

CASE STUDY 24.1: KATHLEEN

Kathleen is a 44-year-old lady who visits her GP with the following symptoms:

- Persistent, non-productive cough
- Tight chest
- Worsening symptoms at night
- Peak flow 300
  - What are your initial thoughts?
  - How would you assess this lady?
  - Is the peak flow normal?

She is initially started on salbutamol and is reviewed weekly; however, her symptoms do not improve. Her GP prescribes Clenil Modulite.

- What is this and why do you think it was prescribed?

Her symptoms continue to worsen and she requires her ‘reliever’ on a regular basis.

- What is the difference between a ‘reliever’ and a ‘preventer’? Provide some examples.

(Continued)
A five-day course of oral prednisolone 30 mg once daily is prescribed and she is sent for a chest x-ray which shows mild hyperinflation.

- Why do you think this is?
- What are the benefits and side effects of this medication?
- What does hyperinflation mean? Relate this to the patient.

Kathleen responds to prednisolone and on review her medication is changed to Symbicort (2 puffs, twice a day). This is a combination inhaler, which can be used as both a ‘reliever’ and ‘preventer’. Kathleen attends the asthma clinic for review every two weeks; however, her symptoms remain poorly controlled. The GP decides to start her on montelukast and refer her to a specialist centre for further review.

- What is montelukast?
- Why do you think this was prescribed?
- What advice would you give to Kathleen?
- What other assessments could be undertaken?
- How would you promote patient self-management?

To support you in formulating your answer, refer to the resources available on the companion website and the national guidelines that have been mentioned within this chapter.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) (2017) and NICE (2010) define COPD as a chronic airflow obstruction resulting from long-term exposure to noxious particles. The exposure results in a chronic inflammatory response which causes damage to the parenchyma of the lung (previously referred to as emphysema) and small airways fibrosis (previously referred to as chronic bronchitis) (GOLD, 2017). Often, the particles arise from cigarette smoke, but may be from occupational exposure, burning fuel such as wood (indoors and outdoors) and air pollution.

Steiner et al. (2015: 805) refers to COPD as a ‘spectrum of lung pathologies associated with systemic co-morbidities and exacerbations’. The heterogeneous nature of COPD can make diagnosis challenging as there is no one specific test (NICE, 2010). A key characteristic of COPD is that the airflow obstruction is not fully reversible; it is a long-term condition that is likely to be progressive.

The physiological changes described above result in deteriorating lung function that often manifests as exercise intolerance. However, MacIntyre (2008) asserts loss of function in patients with chronic lung diseases is multifactorial and the cardiovascular system, skeletal muscle factors, orthopaedic and psychological issues all play a part, which may affect individuals to a greater or lesser degree.

Ventilatory limiting factors

According to MacIntyre (2008), the limiting factor during exercise in health is the cardiovascular system, not the respiratory system. People with COPD develop respiratory limitation due to a load/capacity imbalance, i.e. the mechanical load on the lungs to breathe exceeds the capacity (strength
and endurance) of the respiratory muscles to respond (MacIntyre, 2008). The increased airway resistance for inspiration and expiration and possible reduction in compliance significantly increase the work of breathing. Gas trapping in COPD causes hyperinflation of the lungs (air is trapped in the lungs due to airway narrowing or collapse and poor elastic recoil) with greater effort required by the respiratory muscles (MacIntyre, 2008).

As the lungs are chronically hyperinflated the diaphragm is pushed down and flattened which lessens the efficiency, and inflammatory mediators reduce respiratory muscle strength and endurance (MacIntyre, 2008). According to Hess and Kacmarek (2014), if changes to the diaphragm are profound a paradoxical breathing pattern may occur when the diaphragm contracts. The lateral rib cage moves inward rather than outwards during paradoxical breathing (Hess and Kacmarek, 2014). The primary muscle groups are the accessory muscles (intercostal, scalenes, sternomastoid, pectoralis, parasternal). The capacity to breath and the efficiency with which people are able to breath are compromised by physiological changes within the respiratory system.

### Cardiovascular factors

MacIntyre (2008) describes the cascade of events that may follow chronic respiratory diseases, but impact on the cardiovascular system. In some patients, pulmonary vascular abnormalities may worsen pulmonary hypertension and right ventricular dysfunction, particularly if hypoxaemia is present (MacIntyre, 2008). The loss of function of the right ventricle reduces cardiac output and, therefore, oxygen delivery to the tissues. Deconditioning of cardiac muscle and dyspnoea lead to a spiral of events – inactivity due to dyspnoea lead to further deconditioning of the heart muscle, which causes reduced exercise ability, which causes further deconditioning.

### Skeletal muscle factors

Inflammatory mediators alter protein turnover and the result for COPD patients is a loss of muscle mass significantly adding to loss of function. Another common finding in patients acutely unwell with an exacerbation of COPD is malnutrition. Inadequate nutrition further impairs respiratory muscle function (limiting respiratory muscle capacity). Care must be taken to replace nutrients but not over feed patients as they may lack protein and calories and also have an electrolyte imbalance (Hess and Kacmarek, 2014).

Corticosteroids taken by patients with COPD during exacerbations or long term have a deleterious effect on skeletal muscle protein (MacIntyre, 2008). Acidosis (as might be experienced during an acute episode) also impairs muscle function, therefore the effects are many and varied and all contribute to the spiralling loss of function that is described above.

### Co-morbidities that are commonly found in COPD patients

In addition to the physiological changes to body systems that may result from COPD, there are many patients who experience co-existing diseases that significantly impact on COPD prognosis.

The Global Initiative for Chronic Obstructive Lung Disease (GOLD, 2017) has identified several co-morbidities that are commonly found in patients with COPD: generally the management of these disorders is not altered in those with COPD. According to GOLD (2017), these co-morbidities can occur during any stage of COPD and often have similar symptoms, confounding diagnosis.
Diagnosis

According to Vestbo et al. (2013: 350):

a clinical diagnosis should be considered in any patient who has dyspnoea, chronic cough and/or sputum production, and a history of exposure to risk factors for the disease.

NICE (2010:11) suggests those over 35 with a risk factor and one or more of the following:

- exertional breathlessness
- chronic cough
- regular sputum production
- frequent winter bronchitis
- wheeze.

Due to the common co-morbidities and exhaustive differential diagnosis of COPD symptoms, NICE (2010: 11) also recommend asking about the following factors:

- weight loss
- effort intolerance
- waking at night
- ankle swelling
- fatigue
- occupational hazards
- chest pain*
- haemoptysis*.

* These symptoms are not common in COPD and may suggest an alternative diagnosis.

Diagnosis is complex due to the heterogeneous nature of the illness, which affects people differently. However, the primary symptom is breathlessness; therefore, NICE recommend the use of the Medical Research Council (1959) dyspnoea scale to quantify the amount of exertion required to experience breathlessness.

During the initial assessment patients should have a chest x-ray, full blood count (to assess for anaemia or polycythaemia) and calculation of body mass index (BMI) (NICE, 2010). Other investigations might include (Table 24.2):

<table>
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<tr>
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<tr>
<td>Serial domicillary peak flow measurements</td>
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<tr>
<td>Alpha-1 antitrypsin</td>
</tr>
<tr>
<td>Transfer factor for carbon monoxide (TL CO)</td>
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<tr>
<td>CT scan of the thorax</td>
</tr>
<tr>
<td>ECG</td>
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<tr>
<td>Echocardiogram</td>
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</table>
Once a diagnosis is confirmed the next task is to establish the severity of the disease and the impact on the patient’s life. The disease has many underlying causes and sequelae (as previously described); therefore, a careful medical and social history are required to assess the impact of the disease and options for reducing ongoing exposure risks such as smoking cessation.

**Assessment of severity**

Once the diagnosis of COPD has been made it is vital that assessment of severity of illness and the impact on the individual is appraised. Due to the variable nature of both cause and effect this may require assessment of the following:

- Symptoms – this may include a questionnaire to establish breathlessness, wellbeing and impact of disease on daily life.
- Severity of airflow limitation – spirometry can enable a grading system such as NICE (2010) or GOLD (2017) to be utilised to classify severity of illness.
- Exacerbation risk – exacerbation of COPD is when symptoms have worsened to require a change in medication, and management may be community based or require hospitalisation. Exacerbations are classified as mild – a change to inhaled medication, moderate – where an oral antibiotic and/or oral steroid is required, or severe – support for respiratory symptoms requires hospital admission (GOLD, 2017).

**Treatment/Therapeutic options**

The goals of therapy are to reduce symptoms and frequency at which exacerbations occur and to improve lifestyle and exercise tolerance (GOLD, 2017). However, it is of note that none of the currently available therapies avert the long-term deterioration in lung function.

GOLD (2017) recommend that patients with COPD are offered vaccination for influenza and pneumococcus. Rehabilitation should also be offered as it may significantly improve people’s ability to engage with activities of daily living (GOLD, 2017).

A key goal suggested by both NICE (2010) and GOLD (2017) is smoking cessation; this should be offered to all patients with COPD or, if relevant, reduction of exposure to other pollutants.

Therapeutic options are also broadly categorised as management of stable disease and management of exacerbations. However, therapy is individualised and will vary depending on severity of disease.
Management of stable disease

Inhaled therapy – This is likely to include a short acting bronchodilator to relieve breathlessness (NICE, 2010), short acting or long acting muscarinic antagonists may be required if bronchodilators are not sufficient (NICE, 2010), and inhaled corticosteroids may also be an option for some patients.

These medications may be used in combination. However, it is important the correct delivery device is chosen. Patients need to master inhaler technique in order to deliver the drug effectively; alternative devices such as spacers or nebulisers may be more appropriate.

**WHAT’S THE EVIDENCE? 24.2**

Reflect upon the care you have provided to a patient living with COPD.

- Look at the guidelines relating to medication and consider how you can apply your findings to practice.

You may wish to utilise the GOLD (2017) guidelines when formulating your answer.

Oral therapy – NICE (2010) provide a summary of oral medication use in COPD patients as part of the overall management guideline. Oral therapy may include oral corticosteroids (this is only for exceptional circumstances and the dose should be kept to a minimum). Oral theophylline is a possible therapeutic agent; however, drug interactions may occur and the plasma level requires to be monitored. Oral mucolytic may be considered for patients with a chronic production of sputum, but are not routinely used.

The use of inhaled and oral medication in combination may be required depending on patient response to therapy.

**WHAT’S THE EVIDENCE? 24.3**

It may be appropriate for people to receive long-term oxygen therapy; however, there is a risk of respiratory depression. Refer to the BTS (Hardinge et al., 2015) guidelines for home oxygen use in adults and discuss your findings with your mentor.

**Non-invasive ventilation (NIV)** – If persistent abnormalities exist in arterial blood gas analysis, patients may be referred to specialist centres for assessment for long-term NIV that they manage at home.

This disease is extremely variable between individuals and patient-specific therapy or referral to a specialist is likely. Another consideration is management of co-morbidities. Specialist teams should be established for COPD due to the multifactorial nature and complex management required.

GOLD (2017) suggest patients benefit from pulmonary rehabilitation programmes with improved exercise tolerance and a reduction in breathlessness and fatigue.
Management of exacerbations

Periodically, people may experience an acute worsening of their symptoms and this is described as an exacerbation of COPD (or infective exacerbation of COPD). The deterioration is over and above normal fluctuations of symptoms and often requires a change in medication or support, and is commonly precipitated by respiratory tract infection (GOLD, 2017). An important part of the management of COPD is to prevent occurrence of exacerbations. Exacerbations are significant as they are associated with an acceleration of lung function decline, significant mortality, and long recovery periods that may involve admission to hospital (GOLD, 2017). Patients may be managed in the community or hospital following an assessment of the severity of symptoms and likely need for more advanced respiratory support than they are receiving at home. According to NICE (2010), diagnosis of an exacerbation is made clinically and does not rely on investigation findings. Each individual has a different baseline therefore it is a change from their norm.

Exacerbations of COPD are likely to require pharmacological management. Short acting bronchodilators are commonly used to relieve symptoms of breathlessness. Oxygen should be provided and adjusted to achieve a pulse oximetry target range. Arterial blood gases should be measured on admission and repeated to monitor response to treatment. Antibiotics may be prescribed but only if there is a clinical indication such as purulent sputum or chest x-ray changes. If patients do not respond to optimal medical therapy, non-invasive and possibly invasive ventilation may be required in appropriate settings.

Prior to hospital discharge a full assessment of care requirements should take place, including the possible need for long-term oxygen therapy. Follow-up in the community should be provided.

Oxygen therapy

Oxygen is an important element of care of patients with a wide range of respiratory conditions. As provision of healthcare develops and initiatives such as Hospital at Home increase, oxygen therapy is increasingly being delivered in community settings.

A subset of patients with COPD may be suitable for management using long term oxygen therapy (LTOT) at home. BTS (Hardinge et al., 2015) define LTOT as ‘oxygen used for at least 15 hours a day in chronically hypoxaemic patients’. They require monitoring of this therapy using pulse oximetry. Kelly (2013) states that some community teams also have portable blood gas analysers, sampling ear lobe capillaries to obtain readings. The pH and PCO₂ readings are comparable with arterial samples; however, PO₂ readings will not reflect arterial measurements. Therefore, SpO₂ readings are taken in conjunction to ensure patients are well oxygenated.

Ambulatory oxygen therapy (AOT) may also benefit people who already have LTOT and desaturate during exercise; they would need an assessment to determine suitability.

However, despite the benefits of oxygen therapy there are some important considerations for patients with COPD. NICE (2010) advise patients who have LTOT need to be warned of the dangers of fire and explosion. Oxygen prescription and administration needs to meet the standards required for any medication. Oxygen must be prescribed to meet a target oxygen saturation range recorded on a prescription chart. The current target saturations for patients with hypercapnic respiratory failure are likely to be 88–92%, whereas the target for most acutely ill patients requiring oxygen therapy is 94–98% (O’Driscoll et al., 2017).

Delivery devices are often nasal cannulae or face masks incorporating a venturi system. Nasal cannulae deliver a variable amount of oxygen depending on how much air the patient is also breathing. For example, if the flow is set at 2 l/min and the patient is not entraining much additional air as they breathe they will receive a higher amount of oxygen, or conversely less oxygen if they increase the amount of air they entrain as they breathe. However, nasal cannulae are more easily tolerated. There
is less variability in concentration of oxygen received by the patient with a venturi system as the air is blended as it is entrained through the device to give a more reliable amount.

Kelly (2013) described the emerging evidence base for the dangers associated with the use of oxygen therapy as even short episodes of high flow oxygen during ambulance transfers and during emergency treatment may be detrimental (Kelly, 2013). Careful titration of oxygen using a saturation target is required: using criteria such as apparent breathlessness is not appropriate. It may be appropriate to provide patients at risk of hypercapnic respiratory failure with an alert card they can give healthcare professionals.

CONCLUSION

Worldwide, the burden of respiratory disease is ever increasing, as is the need for safe and effective care. As nurses, we must attain the necessary knowledge and skills to care for this patient population and in doing so we will be able to provide appropriate individualised care across a variety of practice settings.

CHAPTER SUMMARY

- Respiratory disease remains a major cause of premature death worldwide, and asthma and COPD are two of five respiratory conditions that contribute to this global burden.
- By having an understanding of the respiratory system, you will be able to relate your assessment findings to your patient and their underlying pathophysiology.
- Respiratory assessment is an invaluable skill that can be used to assess and care for patients whatever the practice setting.
- By learning to undertake an accurate respiratory assessment you will be able to guide patient management and the provision of timely and effective respiratory care.
- Nurses have a vital role to play in health promotion and lifestyle changes, which can help to improve a patient’s quality of life.

GO FURTHER

Go to https://study.sagepub.com/essentialadultnursing for a further case study related to this chapter.

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Weblinks

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- www.brit-thoracic.org.uk/ The website of the British Thoracic Society (BTS) which develops standards to improve care for people who have respiratory diseases: Better Lung Health for All.
- http://goldcopd.org/ Website of the Global Initiative for Chronic Obstructive Lung Disease (GOLD) which works with healthcare professionals to improve prevention and treatment of COPD internationally.
- www.blf.org.uk/ Public-facing website of the British Lung Foundation which is the only UK charity dedicated to lung health.

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REFERENCES


GOLD (2017) Available at: http://goldcopd.org/


NICE (National Institute for Health and Care Excellence) (2010) Chronic obstructive pulmonary disease in over 16s: Diagnosis and management. Available at: https://www.nice.org.uk/guidance/cg101

NICE (National Institute for Health and Care Excellence) (2017) Asthma: Diagnosis, monitoring and chronic asthma management. Available at: https://www.nice.org.uk/guidance/ng80


Chapter aims

- Describe the origins of reflection in nursing and the reasons for which nurses have not widely adopted critical thinking techniques.
- Outline a number of innovative approaches that seek to promote reflective practice in nursing, in a practical, realistic and creative way.

Introduction

In Chapter 2, the personal impact of nursing in contemporary healthcare and the effect of change were considered. Models of reflection have been used since the 1970s as a means of encouraging thoughtful analysis of nursing practice with only modest success. While there have been many positive examples of reflective approaches being adopted – such as the use of clinical supervision in mental health and health coaching in adult nursing – perhaps the problems associated with broader use has been the rigid, structured ‘model’ approach to promoting and teaching the skills of reflection and the difficulty in then using these models in a busy practice environment. That said, the value of nurses developing their reflective skills akin to Schön’s reflective practitioners ‘in action’ has been shown to correlate with the delivery of high-quality care, evidence-based practice and personal stress reduction.
The Origins of Reflection in Nursing and the Reasons Why Nurses Have Not Widely Adopted Critical Thinking Techniques

What do we mean by ‘reflective practice’ in nursing?

Boud et al. define ‘reflection’ as:

Reflection is an important human activity in which people recapture their experience, think about it, mull over and evaluate it. It is this working with experience that is important in learning. (1985: 43)

Bulman and Schutz describe ‘reflective practice’ as:

‘Reviewing experience from practice so that it may be described, analysed, evaluated and consequently used to inform and change future practice. (2013: 6)

Thus, it can be seen that ‘reflective practice’ can result in critical thinking about one’s clinical practice, skills and competency and the practice of other team members, the application of research and evidence-based practice and the delivery of high-quality care. Reflective practice is also concerned with trying to analyse and understand clinical problems that occur or, conversely, evaluating situations that achieved positive care outcomes.

Approaches to reflection can be informal, but it is the more formal models of reflection that have tended to be used within nursing, and specifically pre-registration nursing courses. The importance of reflection is reinforced within these initial education programmes, with students being expected to select and utilise a model of reflection (from the many options) in order to compile portfolios of their learning and skill attainment, use reflective diaries or employ a model for personal reflection within a written assignment. The difficulty with this strategy is that reflection tends to become an enforced approach that is then often not used after the student qualifies as a registered nurse. Nevertheless, continuing learning is a fundamental aspect of professional development and the contribution of reflection to this process is essential.

The models of reflection used in nursing

A basic search engine exploration using a ‘model + reflection + nursing’ format shows Johns’ model of reflection to be the most widely documented framework used within nursing by a considerable margin. This is followed by Gibbs’ framework, with Schön’s work close behind. Kolb’s (1984) reflective cycle is in fourth place, although it is still utilised quite extensively (Table 6.1).

This was initially proffered as a learning theory but has become more widely known as a ‘model of reflection’. It uses cue questions to structure and help analyse an experience. This then enables us to break down our experience and reflect on the process and outcome. Johns’ (ibid.) work uses Carper’s (1978) four patterns of ‘knowing’ in nursing: empirical, personal, ethical and aesthetic, adding a fifth pattern of ‘reflexivity’. This cyclical process follows five stages:

1. *Bringing the mind home*: Creating space for reflection.
2. *Description*: Summarising the incident.
3. *Reflection*: Thinking about the situation and what occurred.
4. *Alternatives*: Generating different ways of interpreting the incident.
5. *Changes*: Summarising the most workable potential solutions.

Johns’ model is often utilised for written reflections on nursing practice in portfolios and as a structure for student assignments. The model’s popularity seems to stem from the straightforward stages but, while being relatively easy to use, the potential number of suggested questions often has to be reduced so as to ensure that the reflective process is realistic. However, a central concept of the framework (‘bringing the mind home’) is often overlooked and, yet, it has potentially high relevance to more informal approaches to reflection. This first stage emphasises the importance of generating the ‘mental space’ for personal learning by creating time for reflection through such techniques as imagery and mindfulness.


This also evolved as a learning theory but was then utilised in a structured way as a debriefing framework or cycle. It is a frequently used model within pre-registration
nursing courses and is often utilised to structure reflection in professional practice and for critical incident analysis. The framework is particularly useful in helping students reflect on the merits of an experience and their learning styles, and so is often considered easier to use than Johns’ model. The model uses a similar series of steps:

- **Description**: What happened?
- **Feelings**: What were you thinking and feeling?
- **Evaluation**: What was good and bad about the experience?
- **Analysis**: What sense can you make of the situation?
- **Conclusion**: What else could you have done?
- **Action plan**: If it arose again, what would you do?

Schön’s (1983) ‘Reflection-in-action’

In Schön’s (1983) influential book, *The Reflective Practitioner*, the philosopher comments that practice presents problems that are inherently ‘messy, indeterminate situations’, which are characterised by ‘uncertainty, instability, uniqueness and value conflict’ (ibid.: 20). Here, Schön was referring to the process of reflection in professional education, and so his ideas became hugely significant in many professional curricula. He considered experiential learning to be the cornerstone of effective education, and so this may well explain why his work has become so influential in nursing. Schön’s approach celebrates the intuitive and artistic styles that can be brought to uncertain situations. These can be seen in the two central components of the model:

- **Reflection-in-action**: Thinking about one’s practice while doing it.
- **Reflection-on-action**: Thinking about an experience after the event.

Arguably, it is Schön’s concept of ‘Reflection-in-action’ that is critically relevant to nursing. Schön viewed this as an essential characteristic of ‘expert practitioners’ who can test out their ideas while directly engaged in an experience: ‘Our thinking serves to reshape what we are doing while we are doing it’ (1987: 26).

Schön (1987: 13) further describes the process of reflection in action as:

*The art of problem framing*

Surprise triggers reflection, directed both to the surprising outcome and to the knowing-in-action that led to it. It is as though the performer asked him/herself, what is this? And at the same time, what understandings and strategies of mine have led me to produce this?

*The art of improvisation*

The performer restructures his/her understanding of the situation-his/her framing of the problem s/he has been trying to solve, his/her picture of what is going on, or the strategy of action he has been employing.
The art of implementation

On the basis of this restructuring, he/she invents a new strategy of action. S/he tries out the new action s/he has invented, running an on-the-spot experiment whose results s/he interprets, in turn, as a ‘solution,’ an outcome on the whole satisfactory, or else as a new surprise that calls for a new round of reflection and experiment.

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Activity 6.1 Examining Professional Practice Using Schön

Identify an example of health-related ‘good practice’ that you have seen (as a colleague or a patient). This can be anything that you thought was good, well done, impressive, helpful, etc. Thinking about Schön’s work, reflect on your ‘story’ and consider:

• How the person responsible for the ‘good practice’ identified that there was a problem to be solved (the art of problem framing).
• How he/she tried different approaches to solving the problem (the art of improvisation).
• How he/she put into practice their chosen action that led to the good outcome (the art of implementation).

What common themes developed from your reflection? Do you think that these themes would be the same across all professional groups? Perhaps share your observations with a work colleague?

Why nurses do not always use structured reflective techniques in their practice

Given the strong evidence base, it is surprising to note the lack of consistent application of approaches to critical reflection within nursing to date. Why might this be the case? A poll conducted by the author of a group of qualified adult and mental health nurses attending a mentorship seminar elicited the following reasons:

• Reflection has become too formalised.
• Pressure of time owing to the intensity of clinical work.
• The excessive requirements of documenting care events.
• The emotions attached to a situation require quiet reflection rather than a structured approach.
• It is inconvenient to assign time to write up reflections.
• It is preferable to talk and unburden to a colleague.
• Some issues are too personal to document.
• Reflecting in a planned way does not match real-life reflective thinking.
• There is a need to consider individualised approaches to reflection.

The above reasons correlate with the evaluative work undertaken by Barksby et al. (2015), who cite problems with the practical use of staged models and apprehensions regarding who might see the reflective writing. Oelofsen (2012) also underlines the vast number of reflective models available and the need for simplicity of same in practice. While Jasper (2013) explains that, although the techniques to support reflection in nursing are widely known, initiating action needs to become more of a consistent response. Significantly, de Vries and Timmins (2015) claim that ineffective reflective practice can even contribute to poor quality of care. To help counteract this, de Vries and Timmins (ibid.) further call for greater understanding of the impact of the processes associated with reflection such as removing obstacles. Once the barriers have been identified, it then becomes necessary to remove these before reflection can be undertaken (Caldwell and Grobbel, 2013). With this point in mind, the procedure for dealing with barriers as advocated by Boud et al. (1985) is useful:

1. Acknowledge barriers.
2. Name the barriers.
3. Ask how the barriers operate and their origin.
4. Work with the barriers.

Activity 6.2 Examining Professional Practice Using Schön (cont.)

Consider repeating the poll that I conducted with mentors, with nurses from your clinical team, and then try to think creatively about how you and your clinical team can work on removing these barriers.

Innovative Approaches That Seek to Promote Reflective Practice in a Practical, Realistic and Creative Way

Caldwell and Grobbel (2013) conducted a literature review on the importance of reflection in nursing. The outcome of this review identified four themes:

• Development of practice – Reflection has the potential to enhance nursing practice.
• Emotional impact – Reflection provides a safe opportunity for nurses to explore their feelings and emotions.
• **Mentor support** – With support from mentors, students can partake in expressive reflection.
• **Barriers** – Acknowledging the barriers will assist in making the required changes for reflection in practice.

Thus, it is evident that reflection can help to reduce anxiety or feelings attached to stressful events or challenging situations encountered in clinical situations. With the potential benefits of reflection in mind, the following questions were posed to a group of final-year work-based learning student nurses in a supervision session:

• How and when do you reflect?
• Do you prefer formal or informal techniques?
• What advice would you give to a new nurse about reflection?

Reflective writing, using some of the techniques outlined in this chapter, are built into the work-based learning pre-registration nursing course. The feedback received seemed to demonstrate that reflection was relatively widely used. The emotional labour of nursing was clearly identifiable and the importance of critical thinking as a means of helping to cope with such stressful situations was evident. It was further apparent that a range of methods was utilised with many being informal. Yet, formal methods were also seen as necessary, with reflection being recommended as an essential learning tool. The responses further provided some suggested options for reflection such as note-taking, supervision, group debriefings, reflecting on events, returning to personal reflections, peer reflection and contemplative activities. The requirement for a supportive structure was also mentioned and as was the need for colleagues to give and be receptive to constructive feedback.

Comparable feedback on critical thinking was documented by Tashiro et al. (2013), who conducted a thematic analysis and then described the attributes, antecedents and consequences of reflection in nursing. One of the aims of this review was to help nurses enhance their reflective skills. The authors describe how, through reflection, nurses develop self-awareness that then expands care skills and promotes excellence and professionalism. Communication with service users and team members is also enhanced, and self-directed learning improved.

Similarly, a particularly useful way of dealing with some of the pressures implicit in clinical nursing practice is striving to attain the high level of ‘reflection-in-action’ embodied in Schöns’s ‘reflective practitioner’ framework. Schöns’s ‘excellent practitioner’ is perceived by nursing students (as cited to the chapter author) as a nurse who has maintained their enthusiasm and motivation to provide high-quality care and who utilises both structured reflection approaches and ‘reflection-in-action’.

With this observation in mind, and given the strong evidence base for the use of reflection in nursing, one way of dealing with the pressure of working in contemporary healthcare could be by using the strengths of models of reflection, self-reflection and reflection-in-action in a more integrated, realistic, usable, creative and practical way. How might such an aim be achieved? The following suggestions outline some options that could be tested by the practitioner in the clinical setting. Many of these are based on ideas put forward by registered adult and mental health nurses and so are currently being utilised in their clinical roles.
The opportunity for a carefully considered personal review and application of some of these approaches seem to have been presented by the reflection strategy that is now an expectation of the revalidation process by the Nursing and Midwifery Council (NMC). The approach adopted for revalidation appears to exemplify the very structured procedures used to date by being based on evidencing five reflective pieces of work. While ‘PREP’ (Post-Registration Education & Practice) and the ‘ENB (English National Board) portfolio’ did not have a good record of success, the new NMC method provides an opportunity to embed reflective practice in nursing practice. Nurses are often now overheard saying, ‘Note that for your portfolio,’ and potential topics are also frequently explored in mentor updates as a basis for triennial review. Similarly, the NMC (2010) standards for pre-registration nursing education require nurses to be self-aware, evaluate their care and, importantly, learn by reflection as part of their personal and professional development. The Code ‘will be central in the revalidation process as a focus for professional reflection’. (NMC, 2016: 6).

Using mnemonics and abridged models of reflection

Given the relative success of the use of mnemonics in clinical practice, such as the VERA communication framework in dementia care (Hawkes et al., 2015) and the adoption of the ‘PICO’ concept in research question formulation, the embracing of reflective techniques could be similarly aided by simplicity combined with approaches to reflection that are easy to learn and apply. Indeed, the limitations of staged models of reflection are acknowledged by Barksby et al. (2015), who instead advocate the use of the mnemonic ‘Reflect’ as a new model of reflection on action for clinical practice. Use of the mnemonic makes it easier than more traditional models of reflection (Table 6.2).

Similarly, Oelofsen (2012) proposes a ‘reflective cycle’ that has three simple stages:

- **Step 1: Curiosity** – This step involves noticing things, asking questions and questioning assumptions.
- **Step 2: Looking closer** – This step involves actively engaging with the questions from step 1.
- **Step 3: Transformation** – This phase is all about turning sense-making into action.

This framework was developed by the author when working with clinicians in different care contexts and from an interprofessional perspective. This approach can be used in facilitated groups or by individual practitioners in order to promote valuable reflective opportunities.

Another reflective technique – based on an abridged form of a model that could be utilised as a template – that provides simplicity and might be comparatively easy to use is the ‘What?’ ‘So what?’ and ‘Now what?’ process developed by Driscoll in 1994 (Table 6.3).
Table 6.2 ‘Reflect’ as a new model of reflection on action for clinical practice

<table>
<thead>
<tr>
<th>STAGE</th>
<th>(The REFLECT model comprises seven stages)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>R</strong> – RECALL the events (Stage 1)</td>
<td>Give a brief overview of the situation upon which you are reflecting. This should consist of the facts – a description of what happened</td>
</tr>
<tr>
<td><strong>E</strong> – EXAMINE your responses (Stage 2)</td>
<td>Discuss your thoughts and actions at the time of the incident upon which you are reflecting</td>
</tr>
<tr>
<td><strong>F</strong> – Acknowledge FEELINGS (Stage 3)</td>
<td>Highlight any feelings you experienced at the time of the situation upon which you are reflecting</td>
</tr>
<tr>
<td><strong>L</strong> – LEARN from the experience (Stage 4)</td>
<td>Highlight what you have learned from the situation</td>
</tr>
<tr>
<td><strong>E</strong> – EXPLORE options (Stage 5)</td>
<td>Discuss options for the future if you were to encounter a similar situation</td>
</tr>
<tr>
<td><strong>C</strong> – CREATE a plan of action (Stage 6)</td>
<td>Create a plan for the future - this can be for future theoretical learning or action</td>
</tr>
<tr>
<td><strong>T</strong> – Set TIMESCALE (Stage 7)</td>
<td>Set a time by which the plan outlined in Stage 6 will be complete</td>
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By remembering the key questions in this way, this strategy promotes critical thinking in action on care situations aligned with the potential to make brief notes as soon as the opportunity becomes available. This approach can be useful as a structure for serious incident debriefing, biographical work, learning seminars or for clinical supervision.

Table 6.3 ‘What?’, ‘So what?’ and ‘Now what?’ process, developed by Driscoll (1994)

<table>
<thead>
<tr>
<th>Reflective Log</th>
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<tr>
<td><strong>What?</strong></td>
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<td><strong>So what?</strong></td>
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<td><strong>Now what?</strong></td>
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Re-conceptualising reflective practice by applying ‘black box’ thinking to clinical nursing and service improvement

The importance of ‘black box’ thinking, based on the systematic method of investigating airline disasters, has recently been documented in a book by Syed (2016). The ‘black box’ refers to the plane’s data recorder that is used after a crash in order to help investigate the cause. Syed (ibid.) states that aviation has an excellent safety record because, rather than being concealed, mistakes become learning opportunities. He explains that the concept is not concerned with literally creating a black box but, rather, with the ‘willingness and tenacity to investigate the lessons that often exist when we fail, but which we rarely exploit’ (ibid.: 165). He further describes how it is also about ‘creating systems and cultures that enable organisations to learn from errors, rather than being threatened by them’ (ibid.: 116). To emphasise his concept and the importance of resilience, determination and conceptual thinking, Syed describes how Sir James Dyson developed 5127 prototypes before his vacuum cleaner was finally ready to go on sale! He also explains how an anaesthetist worked with a nurse to overrule a surgeon who was incorrectly convinced that a patient was not experiencing an allergic reaction to latex and how junior Korean pilots were reluctant for cultural reasons to challenge more senior pilots, hence creating opportunities for error. As Syed explains, ‘when we are confronted with evidence that challenges our deeply held beliefs we are more likely to reframe the evidence than we are to alter our beliefs’ (ibid.: 375) (Table 6.4).

Table 6.4  The four key concepts that underpin ‘black box’ thinking

| The use of factors that give marginal gain | Marginal gain is not concerned with implementing minor changes but in deconstructing a major problem into smaller components to determine what is effective and what does not work |
| Learning from mistakes | Developing procedures and cultures that facilitate organisational learning as opposed to concealment |
| Learning from successful organisations and individuals | The ability to be open to ideas and critical feedback and to be willing to learn from or share the outcomes of successful projects |
| Avoiding closed loops | Closed loops mean that mistakes are overlooked or misconstrued, whereas an open-loop system ensures action and progress |

The notion of marginal gain

The ‘notion of marginal gain’ was employed to good effect in the Rio Olympics in 2016 in order to achieve the highest number of medals ever recorded by Team GB. The subtle or marginal gain techniques used included the use of the best available
equipment and ergonomically designed apparel. Could such relatively minor changes have had a significant impact on athlete performance? It would appear so. It seems to the author that ‘black box’ thinking may well have potential relevance to healthcare and, hence, could be used for service improvement by following a similar methodical, yet reflective, structure. This method is not entirely dissimilar to established approaches to service improvement, whereby staff (often students) are encouraged to look for and highlight subtle changes in the care environment that could potentially have a significant impact on quality of care. Such changes or approaches to reflection have, for example, resulted in the more productive use of hand gel, which has reduced staff sickness rates, the provision of a clock in an operating theatre in order to ensure that hand-scrubbing conforms to procedures and Wi-Fi availability to promote the more efficient use of care plans in handovers.

The inability to learn from mistakes

The inability to learn from mistakes is based on cognitive dissonance, whereby, if errors occur, then these become difficult to admit to and so are often disregarded or subconsciously re-evaluated. Indeed, de Vries and Timmins (2015) have explained how poor practice, which they called ‘care erosion’, can result from a lack of effective reflective practice. To help overcome this phenomenon, they applied cognitive dissonance theory to several care situations. This theory explains how, when individuals become aware of inconsistencies, they experience discomfort (dissonance) between their thinking and behaviour. People are then motivated to react to remove the dissonance. De Vries and Timmins go on to explain how three focal points to address care erosion emerge from the application of dissonance theory:

- **Improve (or restore) the effectiveness of critical reflection** – By ‘reflecting in a methodical way, aimed at practice improvement’.
- **Promote and maintain strong values and standards** – ‘The nurse needs to become aware of the practical impact of strong values and their expression in practice and their potential to cause dissonance.’
- **Promote optimal care and awareness of signs of care erosion** – ‘Care erosion can be avoided if early signs are addressed before contagion and conformity create its slippery slope.’ (Ibid.: 7)

The ‘ability to learn from successful individuals’ is exemplified in the following case study, combined with the potential of the application of black box thinking to nursing.

Avoiding closed loops

Much of the published literature on communication in nursing is about using closed-loop systems so as to prevent unnecessary risks or misunderstanding. Therefore, a warning that consideration of the use of open-loop systems might cause
Case Study 6.1 Jamie’s Story

Jamie was working as a newly qualified nurse in a community dementia team. The team already offered people recently diagnosed with dementia the opportunity to compile their life story. Because of the author’s request to think about his reflective practice, ‘J’ noted that the uptake of life-story work by people with dementia had declined over the past year. ‘J’ spoke to clinicians experienced in life-story work and read publications by nurses who had compiled their life story and then shared this with relatives and service users before inviting them to develop their biographies. The impact of this was to demonstrate a commitment to the concept of life-story work and to show how powerful it could be. It also promoted a positive therapeutic relationship with the person and their family and an increase in the number of individuals undertaking life-story work. While not apparently a reflective approach, the careful use of evidence and consultation to re-evaluate a care strategy enabled an improvement in life-story uptake and potentially enhanced the quality of care.

cognitive dissonance! Syed (2016) explains that an open-loop system is concerned with implementing a strategy, then testing if it has been successful, working on any presenting problems, thereby enhancing the strategy.

Perhaps consider this idea in respect of your own health/social care organisation or NHS Trust and reflect on the structures that exist to support nurses to speak out about patient safeguarding concerns. Such a requirement is part of your professional code of conduct. Does your organisation facilitate such a culture? Are staff supported to raise concerns? Perhaps make notes on any examples of good practice that you come up with or, equally, any areas that require improvement.

Clearly, the notion of ‘black box’ thinking has resonated with the author of this chapter. I am of the view that the fundamental concepts outlined above have applicability to nursing and should be utilised and tested in the clinical setting. That said, often, the sheer pace of and pressures in practice, mitigate against taking time for even the briefest critical reviews of care situations. However, this chapter has established that the use of the ‘black box’ thinking phenomenon could help resolve some quality of care problems. One potential approach that could also be tested in such situations is an idea closely allied to the notion of marginal gain, namely ‘one-minute’ interaction analysis.

Activity 6.3 Reflective Exercise

Perhaps test this idea out. Take just a minute or two to step back from the intensity of your clinical role and observe an interaction between a colleague and a patient in your practice environment. What communication techniques is your colleague employing? Is the person being listened to in a meaningful way? What non-verbal signals are being given?
Creating opportunities for ‘bringing the mind home’

It seems to the author that removing oneself from stressful situations and taking the time to contemplate is essential. Is it similar to Johns’ notion of bringing the mind home by making space for reflection? While compiling this chapter, I spoke to a number of qualified nurses about how they create opportunities for reflection and have recorded below the ideas that they found to be both useful and efficient. Essentially, these strategies are concerned with opportunities for reflection in action, however, as has been seen, establishing a time for reflection on action can be equally important.

Activity 6.4 Reflective Exercise

Ask some of your practice colleagues if they reflect and, if they do so, how they go about it. Emphasise that this exercise isn’t concerned with trying to catch them out but with documenting the creative ways in which reflection can take place. The approaches used by your colleagues may include making notes and thinking about everyday practice activities in a different way.

Make a bullet point list of the reflective techniques that they use. Are any of them useful for your own practice?

A popular method seems to be keeping a notebook to hand and then later using these records to make an entry into a personal journal. Both can take advantage of the abridged headings from established or new models of reflection. Some nurses have extended this idea by using a Dictaphone or audio device to record personal thoughts for a later discussion or portfolio entry, but, again, you may need to check local information governance procedures if you plan to use this approach. The logical progression from a journal is to consider the use of tablet computers, apps or other software, interactive whiteboards, work-supplied mobile phones or laptops. Again, information-governance procedures will need to be checked. These means are already being used to record care events, so why not use your mobile device to note your reflections? Meanwhile, Knight (2015) explains how a facilitated reflective practice group, based on containment principles, enables participants to express their thoughts in a form that is more meaningful and endurable. The structure of these groups is provided by agreeing on ground rules, promoting positive conversation and by broadly using the framework of a reflective model.

Some adult nursing colleagues cited the use of ‘bedside handovers’ as events for reflective practice and learning opportunities. Additionally, many mental health nurses spoke of the usefulness of clinical supervision as occasions for reflection. Most talked about the importance of individual supervision but a number mentioned group supervision because this was more realistic regarding resources. The use of recording
in the ‘first person’ in care plans was cited as a means of promoting care ownership by service users. In turn, this enables empowerment and contributes to recovery. Structured storytelling has been used to promote engagement. Brief seminars on clinical issues based on Driscoll’s work was also mentioned and so, too, was case formulation, which is a process that promotes reflective practice and critical discussion in mental health, which then increases clinician understanding of and empathy towards service users. The starting point for this is often the consideration of the person’s life story. Case Study 6.2 gives an example of how case formulation might be used to support reflective practice and so contribute to the professional revalidation requirements.

**Case Study 6.2  Parveen’s Story**

Parveen, an experienced charge nurse working in an acute mental health inpatient unit, found the pressure of working in such an environment demanding, particularly regarding finding the time to document care events and risk management. Parveen was aware of the value of reflection in terms of enhancing her use of evidence-based practice and the need for professional development. She suggested that a way of still meeting her personal aims of using an integrative approach to reflection was to utilise a case-formulation style within scheduled ward handover meetings. This consisted of using a personal biographical approach as a precursor to discussing service user needs. The outcome of this process was detailed written entries in care records, which then promoted a more meaningful and individualised dialogue with service users. This also provided the basis for entry into Parveen’s NMC revalidation portfolio.

**Conclusion**

This chapter has acknowledged the pressures inherent in contemporary clinical practice but has emphasised the positive personal and care outcomes of making time for reflection. The focus has been on describing a number of creative approaches to reflection that aimed to provide a chapter that you could ‘dip in and out of’. Many of these ideas have been suggested by clinicians, while some have been published in academic papers. The strategies for reflection include the use of mnemonics or abridged models of reflection-on-action, re-conceptualising reflection by utilising the principles of ‘black box’ thinking, marginal gain and cognitive dissonance theory and by outlining several approaches that create opportunities for ‘bringing the mind home’.

The author hopes that you will test out some of these ideas and incorporate them into your practice. It may be that you develop your own eclectic paradigm of reflection by taking the best parts of each approach or strategy to fit with the uniqueness of your experiences. Good luck with the methods that you use. Please bear in mind that reflection does not have to be on negative or difficult situations, reflect on the positive things, too.
References

Nursing and Midwifery Council (NMC) (2010) Standards for Pre-registration Nursing Education. London: NMC.
Nursing and Mindfulness
Caroline Barratt and Tess Wagstaffe

We have a finite amount of energy to spend every day before becoming exhausted. Mindfulness helps you use your energy wisely, spending it on situations, people and causes that bring you the most joy, meaning and peace. (Hanh and Cheung, 2011: 7)

Chapter aims

- Introduce mindfulness.
- Discuss how mindfulness can benefit nurses.
- Understand how nurses have integrated mindfulness into their professional practice through case studies.
- Develop mindfulness techniques.
- Explore common questions about mindfulness.

Introduction

The inclusion of a chapter on mindfulness practice in this book for nurses reflects the personal experiences of the authors of the benefits of mindfulness practice, in both their professional and personal lives, as well as reflecting a growing evidence base that suggest the potential of mindfulness to improve the well-being of health professionals. However, it is not just about using mindfulness practice to cope with difficulty. We also explore the potential of mindfulness to help us build relationships, with ourselves, patients and colleagues; support the creation of positive working environments; improve patient safety; and putting nurses back in touch with why they engage in the work they do and the joy therein.
This chapter includes some activities that you may wish to engage in. Mindfulness doesn’t really mean anything while it remains an abstract or intellectual concept. It is something that needs to be experienced. Please take care of yourself throughout your exploration of mindfulness. It is possible to experience strong and difficult emotions as well as calmness and positive states of mind, so it is important to take into account your current state of mind and capacity for engaging in these exercises. If you do experience difficulties or feel uncertain, we would recommend stopping any further mindfulness practices and seeking the guidance of an experienced mindfulness teacher.

What is Mindfulness?

Interest in mindfulness training to support the well-being and professional practice of health professionals has grown significantly in the last 20 years. The evidence about how mindfulness practice can impact health professionals suggests that the development of mindfulness can help to protect healthcare professionals, including students, from burnout and stress (Burton et al., 2016; Craigie et al., 2016; Horner et al., 2014; Shapiro et al., 2005; Gockel et al., 2013; Newsome et al., 2012; Gauthier et al., 2015; Cohen-Katz et al., 2005). For students training to be healthcare professionals, mindfulness practice has also been associated with improved patient care (Shapiro et al., 1998; Shields, 2011; Warnecke et al., 2011). A critical interpretative synthesis of findings from qualitative research on the impact of mindfulness training on nurses and midwives concluded that mindfulness created a:

quiet mental space giving them agency and perspective and leading to improved caring, including a more patient-centred focus and increased presence and listening. Mindfulness appears to alter the way nurses and midwives operate within a stressful work environment, thereby changing the way the environment is experienced by themselves and, potentially, the people in their care. (Hunter, 2016: 918)

Other nursing literature, such as Watson’s Human Caring Theory (Sitzman and Watson, 2013) and Parse’s concept of ‘true presence’ (Palmieri and Kiteley, 2012), has also drawn on the concept of mindfulness as a mechanism of improving connection and quality of care.

There is, however, some criticism of the evidence base for the effectiveness of mindfulness, particularly due to methodological issues such small sample sizes, lack of control groups and failure to investigate side effects, which means that the benefits of mindfulness may have been overemphasised and the risks overlooked (Farias and Wikholm, 2016; Lomas et al., 2015).

What it means to be ‘mindful’ or ‘practice mindfulness’ is currently a matter of much debate. The complex historical and spiritual roots of the term in Buddhist philosophy and the variety of definitions for ‘mindfulness’ make its translation in
Westernised mindfulness teaching difficult (Stanley, 2013). Kabat-Zinn is often credited with the initial work in which the concept of mindfulness was integrated into a training course to improve well-being at the University of Massachusetts Medical School. That course is now known as Mindfulness-Based Stress Reduction (MBSR). Kabat-Zinn defines mindfulness as: ‘paying attention in a particular way; on purpose, in the present moment, and non-judgmentally’ (1994: 4). We can therefore start thinking about mindfulness as getting out of ‘autopilot’, dominated by constant thoughts and judgements, and become more aware and present in our moment-to-moment experience. Kabat Zinn describes the reason for doing this:

when you begin paying attention to what your mind is doing, you will probably find that there is a great deal of mental and emotional activity going on beneath the surface. These incessant thoughts and feelings can drain a lot of your energy. They can be obstacles to experiencing even brief moments of stillness and contentment. (2004: 25)

Our minds are rarely still. Not only are we thinking about what we are doing at the time but we are also thinking about what happened that morning, the argument we just had with a colleague or what we will make for dinner in the evening. Not only does our busy mind create suffering but it also prevents us from noticing and being present within the joyful and happy moments in life. This quote from Hanh helps to convey what is meant by ‘being mindful’ during a particular activity and how it can alter the experience of being alive. Here, he focuses on washing-up:

If while washing dishes, we think only of the cup of tea that awaits us, thus hurrying to get the dishes out of the way as if they were a nuisance, then we are not ‘washing the dishes to wash the dishes’. What’s more, we are not alive during the time we are washing the dishes. In fact we are completely incapable of realising the miracle of life while standing at the sink. If we can’t wash the dishes, the chances are we won’t be able to drink our tea either. While drinking the cup of tea, we will only be thinking of other things, barely aware of the cup in our hands. Thus we are sucked away into the future and we are incapable of actually living one minute of life. (1987: 4)

In terms of nursing practice, when we are attending to a person’s personal care or assessing a person’s mental or physical health whilst being preoccupied with an endless list of other tasks, we may not only be missing out on gathering information, listening attentively or focusing on the task in hand, but we are also at risk of missing out on moments of connectedness with our patients. These are moments in which we can perhaps reconnect with why we do what we do and, in turn, find satisfaction and pleasure within our role as a nurse. Therefore, developing mindfulness is not just about becoming more present in and aware of our suffering, it has the potential to wake us up in all aspects of our lives and thankfully that includes moments of joy, love and connection in both our work and personal lives.
Activity 5.1 The Pause

A first step in developing mindfulness is learning to stop and notice our current experience. It can be useful to regularly take a pause in our daily life as a way of learning to check in and notice how we are. This exercise can be used as many times as you would like throughout the day:

1. Stop what you are doing. You can remain seated or standing.
2. If appropriate for where you are, you can close your eyes.
3. Bring your attention to your breathing and follow the breath cycle for 10–15 breaths. There is no need to breathe deeply or in any particular way.
4. When you are ready, open your eyes.

Try this at different times during the day. How does it affect you?

Writing from a Buddhist perspective, Subhuti describes that, in general, the term mindfulness ‘may refer either to a particular quality of consciousness or to the effort to create that quality of consciousness in oneself … But, whether we are thinking of the product or the process, mindfulness clearly has to do with the highest possible lucidity and clarity of mind’ (2015: 190). He describes how Buddhist discourse emphasises three aspects of mindfulness: attentiveness, awareness and vigilance. Subhuti describes ‘attentiveness’ as becoming more aware of sensory experience – the direct experience of a given moment as opposed to being caught up in a mental dialogue about it. The ability to attend to experience in this way can be developed through training the mind in the ability to focus and concentrate. Modern definitions of mindfulness, such as Kabat-Zinn’s described in the introduction, tend to focus on the attentiveness aspect of mindfulness.

However, attentiveness to experience practiced in isolation may lead to a practice in which the practitioner is detached from the context in which the experience is arising. Without a broader ‘awareness’, there is a lack of clarity about where we are, our motivations for doing what we are doing and why we are participating in that activity. This is important for you as a nurse, for whom becoming narrowly focused and losing a sense of the wider context is clearly potentially problematic for patient care. Developing awareness supports our attentiveness by supporting a broader sense of what is going on within our experience and how it relates to others. In caring for others, this is particularly important. It is important that we are attentive to our own sensory experience but without a broader awareness – which also acknowledges the experience of the person we are caring for within the context around us – we will not be effective.

Lastly, Subhuti (2015) refers to the importance of ‘vigilance’, which he describes as the ethical quality that is informing our actions and state of mind. Is the way in which we are behaving or thinking indicative of kindness and compassion, the values that we wish to model? Or has a more negative mental state crept in to cause us to act in ways that don’t align to the values that we hold?
So, mindfulness is not necessarily just about ‘bare attention’ and non-judgement in the present moment. That is a very good place for us to start in terms of developing our mindfulness practice but, in itself, it only presents part of the picture. Whilst your own experience of caring for another is important, clearly it is important to hold that within a broader awareness of the situation and the needs of your patients and colleagues, you must not become isolated or disconnected from them.

Activity 5.2 Becoming Aware of Distraction

To be more present, it can be helpful to develop awareness of how distracted we often are and so this is the focus of this second activity.

When caring for patients, start to become aware of where your attention is and to what extent you are ‘present’ with the person you are caring for. At the end of your shift, note down what distracts you and pulls your attention away from providing care. You may wish to consider:

- Sensory experience, such as smells, sounds, pain and hunger.
- Emotions, such as emotions arising that make it difficult to focus and be present.
- Thoughts, such as mental chatter about issues that you are not immediately concerned with, planning for what is next or judgements about the situation.

You may find it useful to ask a colleague to do the activity, too, so that you can then discuss what you notice and explore the implications for practice.

Source: Adapted from Barratt (2017)

Attitudinal Foundations of Mindfulness and their Role in Nursing

Another way to explore mindfulness is to consider what Kabat-Zinn refers to as the ‘Seven Attitudinal Foundations’ (2004) (Figure 5.1). These foundations describe the qualities that are helpful in the cultivation of mindful awareness.

Beginner’s mind – In order to become more present, we need to start to become aware of the assumptions and beliefs that prevent us from seeing things as they are. ‘Beginner’s mind’ is the willingness to see things as though we have not seen them before. It is the cultivation of curiosity and interest even in situations that are familiar to us, so that we are attentive to what is happening rather than relying on mental constructions of what we think is happening based on previous experience. Whilst as nurses we are aware of the importance of our knowledge of previous health history, or perhaps previous risk assessments, it is important to combine this with paying attention to the situation as it presents itself at the time. Brandon explained...
trust

Attitudinal factors of mindfulness

Figure 5.1 Seven attitudinal foundations of mindfulness (adapted from Kabat-Zinn, 2004)

how there are times when our minds are ‘wandering so much that here no room for anything that is being said. One is just there physically’ (Brandon, 1990: 6), and assumptions can lead us almost to make decisions before we have completed the new assessment and risk, missing new signs or symptoms. Developing a beginner’s mind can help overcome these risks.

Non-striving – In everyday life, we engage in tasks with the hope of seeing a particular outcome. We administer medication with the hope of making patients better, we sit and talk with a patient in the hope that they will feel brighter, or comforted or engage a patient in therapy in the hope that their depressed mood will change or they will stop self-harming. However, in developing mindfulness, this attitude is not helpful as it suggests that in order for things to be ‘ok’, something needs to happen, something needs to change. This thought prevents us from being present with whatever it is that is happening. For nurses, a perceived lack of therapeutic success can lead to feelings of frustration, burnout and disengagement from our patients. This can also prevent us from engaging or recognising the positive contributions that we make to patient care that do not result in a ‘cure’ or an outcome that we judge to be successful. Rogers asserts that when we are open to different outcomes, we can ‘realize the vital strength of the capacity and potentiality of the individual for constructive action’ (Rogers, 1951: 48).

Letting go – As we become more mindful and more sensitive to our thoughts and feelings, we may find that there are thoughts, beliefs or feelings that we cling to.
Wanting things to be a certain way or wanting other people to act in particular ways can be a cause of tension and stress. Mindfulness practices ask that we are open to however things are and to do what we need to do in order to let go. Whilst at work, it can be easy to get caught up in a general sense of stress in the workplace or to start ruminating on an issue with a colleague or patient. By bringing ourselves into the present moment, we are more able to let this go and focus on what is in front of us rather than clinging to thoughts about the way something should or shouldn’t be.

Acceptance – When difficult things happen in our life or at work, for example the death of a patient, we make a clinical error or disagree with a colleague, and we tend to go through a process of denial and anger before starting to develop some level of acceptance. Arguing with the way things are is ultimately fruitless and can prolong our suffering. Very often, we cannot change what has happened and, if there is the possibility of changing things, then we are often unable to see it until we can accept what is going on and work with it in a more creative way.

Non-judging – Intrinsic to the development of mindfulness is becoming aware of the constant stream of judgement that our minds engage in and becoming able to stand back from that. The ability to do this means that we are not caught up in our mental judgements about the situation we are in. This increases the possibility of being present in our experience and responding appropriately to it rather than reacting based on unconscious habits that have developed in response to our judgements. Within nursing, we are constantly required to think critically and continuously reflect on our practice, which has the potential to manifest itself in being judgemental towards ourselves in a negative and unhelpful way that can greatly impact on our well-being. This will be explored further in Chapter 7, titled ‘Self-compassion’. We may also engage in judgement of others, which can often colour our interactions with them, causing us to label them as ‘problematic’ even before we have greeted them on that occasion.

Patience – Kabat-Zinn describes patience as a ‘form of wisdom’, the cultivation of which shows that we ‘accept that the fact that sometimes things must unfold in their own time’ (2004: 34). As we become more aware of our minds and bodies and the suffering we can experience in relation to them, learning to hold that with gentleness and patience is important. Patience gives us the room to allow what is there to be there and not to be in a hurry to change it or ourselves in a forced, strident way. Trying to integrate mindfulness into nursing practice is a challenge, so we need to ensure that we are patient with ourselves as we try to develop new ways of working and being.

Trust – As we become more present and aware, we become more able to listen to our feelings and intuition. This enables us to identify the most skilful responses to the situations in which we find ourselves and we start to trust ourselves more and more. We start to notice the judgements and assumptions of others and are less blown about by those with strong opinions, and with awareness we are able to bring ourselves back to our experience and use that as the basis for our decision-making. This is likely to influence our self-confidence as a nurse and, paradoxically, as we start to trust ourselves more, and become less defensive, being open to the views of others becomes less threatening.
Developing mindfulness

There are many ways to develop mindfulness and it is important that you don’t feel forced to engage in any one particular practice, but explore what suits you. Mindfulness practices fall into two rough groups: formal and informal.

‘Formal practices’ are those that require you to take time out from your normal activities to engage in them. This may include meditation practices such as the Breath Awareness Practice, which involves focusing on the breath usually whilst seated and with the eyes closed; or Body Scan, which involves focusing on different parts of the body in a sequential process, this is usually done lying down with the eyes closed. The time taken can vary considerably from five minutes to an hour.

‘Informal practices’, such as ‘The Pause’ in Activity 5.1 or ‘Becoming aware of distraction’ in Activity 5.2 can be done within your everyday life, providing an opportunity to become aware and present within the flow of the day. The most effective way of developing mindfulness is by combining formal and informal practices and engaging in them regularly. Reading about mindfulness is not sufficient. Conceptual and experiential understanding are not the same, so engaging in practice is an important element of coming to appreciate what is meant by ‘mindfulness’.

There are now many resources on mindfulness available online as well as in books. The References section at the end of the chapter includes some helpful resources in this regard. However, we have found that engaging with an experienced mindfulness teacher who has a committed mindfulness practice themselves can be very valuable in developing a mindfulness practice. Opportunities for training are often available in the workplace and, if you are not aware of any it, may be worth enquiring. You may also wish to consider taking a course such as Mindfulness-Based Stress Reduction (MBSR), which gives an experiential introduction to mindfulness.

Although mindfulness meditation is usually well tolerated, it has been associated with side effects such as anxiety and psychosis (Farias and Wikholm, 2016) and, as yet, insufficient research has been carried out to establish why this is the case or who is particularly at risk (Dobkin et al., 2012). We have chosen the exercises and suggestions in this chapter with your safety and well-being in mind, avoiding extended periods of meditation, but be attentive to your own needs and limits. If you have suffered from recent trauma, bereavement or are suffering an acute period of mental illness, then be cautious, particularly around periods of silent mindfulness meditation, and seek out an experienced and suitably qualified mindfulness teacher if, after having read this chapter, you wish to explore things in more depth.

Mindfulness, nursing and resilience

Hopefully, from the discussion of what mindfulness is, you are starting to get a sense of why mindfulness is relevant to you. However, we now move on to draw out some of the most salient points about why mindfulness practice may be particularly useful
for nurses. We provide specific examples so that you can start to explore in your own mind and experience the relevance to your professional practice.

Noticing Thoughts and How They Impact Experience

I’m not saying that thinking is bad. Like everything else, it’s useful in moderation. A good servant, but a bad master. (Watts, 2013)

As we have already said, the aim of mindfulness is not to stop thinking. Thinking is a useful tool—‘a good servant’, as Watts suggests above. As nurses, the wealth of knowledge that you have built up through your training and experience is not forgotten or put aside during mindful nursing practice. Thought is an essential tool in your decision-making, a way of drawing on the knowledge that you have.

However, mindfulness can help us change our relationship to thought. It is possible for us to get stuck in unhelpful cycles of thought about our experience that can make us feel worse and prevent us from seeing what is going on. It is also possible for our underlying beliefs and assumptions to affect how we perceive our experience and influence how we react in any given situation. Becoming aware of what is influencing our perception allows us to step to one side and take a fresh look. Developing mindfulness means that we develop the ability to be less caught up in the mental dialogues about what is going on in our lives, and the wrong or right of it, and become better able to identify what our actual experience of it is. This does not mean that mindfulness practice will instantly make us feel better, or that difficult emotions will never affect us, but it does mean that we can have greater insight and increase the possibility that we can intervene and not get trapped in unconscious patterns of thoughts that cause us distress.

Thinking, stress and suffering

As we have already discussed, our minds tend to jump around from thought to thought, but our minds can also get stuck on a particular thought pattern that might be unhelpful. For example, when I am tired and feeling overwhelmed at work, I only notice tasks that I feel I could have completed better, or remember conversations with patients or relatives that I feel did not go as well as I would have liked. I have noticed that it is this thought pattern and this constant anticipation of feeling overwhelmed that is causing much of the stress and poor confidence.

Even when I sit down to relax, I feel stressed and anxious because my mind is still caught up. This can also happen when I get in from work and I continue to ruminate over how I could have performed better, within my role, or how difficult it feels at times. This means that the anxiety gets prolonged and eats into the apparently relaxing and pleasant aspects of our lives.
Activity 5.3  Identifying negative thoughts

Identifying what negative thoughts and stories our mind gets stuck on can be very helpful and is the focus of this activity. If, however, you find the task upsetting or that the focus makes you more stressed, perhaps you can try the activity at a different time with the support of a friend or colleague. Make sure that you take care of yourself.

Over the next week, pay attention to stressful or difficult thoughts or trains of thought that get repeated in your mind even when you have no wish to think about those issues. You may wish to make a note of:

1. What are these thoughts about? For example, work, family, money, etc.?
2. When are these thoughts most prevalent?
3. What is your experience of your physical body whilst thinking about it? For example, is there pain or tension in the body? How is your heart rate and breathing?
4. What emotions are present?
5. What effect does bringing awareness to these thoughts have on your experience of them?

Through mindfulness practice, we can become aware of the things that pull us away from where we actually want our mind to be. Often, these distractions are fuelled by our fear of suffering. This includes the whole spectrum of human difficulties, from the small frustrations of writing to the painful experiences of illness and grief. Thomas Merton wrote:

The more you try to avoid suffering, the more you suffer, because smaller and more insignificant things begin to torture you, in proportion to your fear of being hurt. The one who does most to avoid suffering is, in the end, the one who suffers most. (1948: 91)

Nurses are constantly exposed to the most challenging aspects of human life – those events that cause the most pain and suffering. It is not surprising then that nurses often experience stress and burnout. Being able to recognise that we are in pain or experiencing difficulty and being able to then respond appropriately is fundamental in self-care and is something that can arise through the development of mindfulness. This will also be explored in Chapter 7.

Using mindfulness in our working with others

Central to the idea of mindfulness is the idea of being present, aware and open to our present moment experience. In particular, this relates to developing our ability
to control where we place our attention, to become less vulnerable to our ‘monkey minds’ and take ourselves out of ‘autopilot mode’, which can dominate our lives. Halifax (2014) developed the GRACE model of interacting with patients that encourages a mindful approach by helping to focus our intention as we first step into a relationship with the patient, encouraging meaningful and helpful interaction and then bringing the encounter to an end. Halifax describes how the GRACE model can support compassionate care:

Clinicians often do not take a ‘reflective pause’ but jump into immediately assessing the patient before getting attentionally and ethically grounded, seeing their biases, then sensing into the patient’s experience before making a clinical assessment. The GRACE process can guide a nurse into that moment (or moments) of reflection that can provide the base for healthy, grounded and principled compassion. (Ibid.: 123)

The stages of the GRACE model are shown in Figure 5.2 and are discussed below. In the first stage of gathering attention, Halifax suggests that we take just a moment to notice where our attention is, perhaps becoming aware of the in-breath or sense of feet on the floor to help us become present.

In the second stage, she suggests we bring to mind our intention, get in touch with a sense of why we are about to engage with that person and the values that we seek to embody while doing it. We do not often think about the values that bring us to the work that we do and, yet, for many nurses, it was a strong sense of vocation and a desire to care that brought them into the profession. Bringing this to mind during the workday may be a source of support for us as it helps reconnect us to our sense of purpose, which is often lost in the busyness of everyday healthcare delivery. It should be noted that neither of these stages needs to take any more than a few

![Figure 5.2 GRACE model of compassion care (adapted from Halifax, 2014: 123)](image)
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seconds – Halifax is not suggesting that we take a break every time we move on to a new person!

In the third stage, we attune first to ourselves, how we are feeling, the thoughts that are arising as we start to move towards engaging with this person. Having become aware of these, we become more conscious of what we are bringing with us into the interaction and how what we are feeling and experiencing might affect our perception of the person for whom we are caring. Then, we attune to the other person, having noticed our own biases and subjectivity and using that as the basis for empathy, we orientate our attention to the other person.

In the fourth stage, we consider what will serve, explicitly bringing to mind relevant experience and knowledge that is relevant to identify what needs to be done. Halifax described how this process of discernment ‘requires attentional and affective balance, a deep sense of moral grounding and an ethical imperative, as well as an unbiased attunement into the patient’s experience and needs’ (2014: 124), which is what is established in the first three stages of the model.

In fifth stage, we engage with the patient, carrying out the work that needs to be done before finding a way to end the encounter. Halifax places strong emphasis on setting up before engaging with the patient by first getting in touch with yourself, becoming aware and present. Equally, she then emphasises the importance of needing to consciously end the interaction, to: ‘acknowledge internally and often interpersonally what has transpired’. Failure to do this may make it difficult to move on to the next task because, mentally, you may remain caught up in previous encounters that, although no longer actually happening, remain live either emotionally or in our thoughts or both.

It is important to note that although we have discussed this model in relation to working with patients, it is equally applicable in any encounter that we have. Its value is particularly powerful in interactions that may be problematic such as with a difficult colleague or manager. Grounding ourselves before engaging, especially at times where we feel an emotional charge or particularly vulnerable, can help to create space for skilful responses as opposed to reactions based on habit.

Finding the joy: Becoming mindful of what we give and what we receive

As we said earlier, becoming mindful is not just about recognising the difficult or challenging aspects of our lives. We can also become more aware of moments of joy, happiness and beauty, more able to feel them deeply and become better at noticing them within our own experience. In a qualitative study of the impact of mindfulness training on nurses by Cohen-Katz et al. (2005: 84), they cited a nurse who had been on an MBSR course, which includes a task in which they are asked to observe and record pleasant events over the week, like saying:

When looking for a pleasant event, I kept looking for something really big. Then I realised that the simple interactions with others in my life are the ones that are the most pleasurable – really noticing how it feels when my husband's arms are around me in bed or hugging my child and stroking her hair …
We can train ourselves to start to notice pleasant experiences when they arise but this is challenging if we have become used to living in states of stress and anxiety. The phenomenon of confirmation bias describes how people pay more attention, and give more weight, to information that confirms their current position or beliefs (Wason, 1960). Whilst a little oversimplistic, an example of this is if we have decided that, today, ‘I got out of the wrong side of bed,’ we will naturally be drawn to noticing the aspects of our experience that confirm this – the person who pulls out on us on the motor way; the negative comment from a colleague; the milk running out – as opposed to those aspects of your experience that may present a different case: your partner making you a cup of tea; the flowers in your front garden; the person who let you out at that busy junction. This is not to say that there aren’t difficult circumstances in our lives and jobs that are demanding and draining but, as we learn to pay attention, we start to notice the variability of our experience and the aspects of our experience that don’t fit with our dominant narratives.

This is just one very particular example about a given day but imagine these patterns writ large over the whole course of our lives? How much are we missing? In order to work that out, we have to stop and notice – which is the first step in mindfulness practice. Activity 5.4 provides a way of starting to reconnect with the positive aspects of our experience.

Activity 5.4  Shifting Our Focus to Change Our Experience

Where we choose to place our attention has a huge impact on how we experience the world. As human beings, we are wired to notice threat and, as such, tend to focus on negative experiences. Have you noticed how despite all the people you might help in one day, all of them will be forgotten as you focus on the one person whom you couldn’t help or the mistake that you made? This task has two elements. At the end of each day, sit down and write:

1. At least three things that you have done to help others, where you are able to appreciate the contribution that you made to their lives (personally or professionally).

   They do not have to be big things! Making someone a cup of tea, giving your time to chat with a friend, holding a patient’s hand to ease their fear, remembering to say ‘Happy Birthday!’ to a colleague … these are worthy of recognition and celebration.

2. At least three things that you are grateful for that day.

   Again, they do not have to be big things! Perhaps seeing the sunshine as you walked from your car into work, a patient who said ‘Thank you,’ a nice meal.

These are exercises that help to retune our attention so that we start to become increasingly conscious of the positive aspects of our lives.
Nursing and Mindfulness: Frequently Asked Questions

In this section, we take three key questions that are often raised about mindfulness and nursing as a way of exploring how mindfulness is relevant to nurses and within nursing practice:

1. At times, when I am feeling really stressed and anxious, I find the mindfulness meditation exercises difficult as the ‘space’ becomes filled with worry and preoccupation. I have at times tried the practice before work but, due to the worry, this has been difficult. What would you recommend?

This is a very common experience even amongst experienced mindfulness practitioners. First, it is important to note that the practice of mindfulness meditation does not require us to stop thinking, have a ‘clear mind’ or be relaxed. This is a common unhelpful misconception. During meditation, the practice is simply to be aware of what is – including the thoughts that arise and fade out during practice as well as the tension that may be present in our bodies. Striving to become calmer or to stop thinking is likely to make the practice more difficult, as you may get more tense when you notice that your experience is not as you wish it to be (you may find it helpful to reread the ‘Attitudinal Foundations of Mindfulness and Their Role in Nursing’ section above). For example, when engaging in a breath awareness practice, which involves focusing on the experience of the breath, when you realise that you have become distracted from the breath, then you repeatedly bring your attention back to the breath as best you can. Sometimes, it may feel like you do this hundreds of times a minute! Or, perhaps, if you have one dominant story running through your mind, you may completely forget the practice for many minutes at a time. But this does not mean that you are ‘bad at mindfulness’ or ‘can’t meditate’, it is simply how the practice is today. So, even when we feel like the practice is ‘going badly’, the invitation is to keep practicing with gentleness, patience and a beginner’s mind. Your experience of the practice can change dramatically even within a short time.

Having said this, however, I appreciate how challenging it can be to meditate when your mind is all over the place and I have at times cut meditations short for this reason. When starting out, be realistic about how long you want the mindfulness practice to last. You may find it easier to build up to longer periods of time. Be conscious of what is going on in your life and be kind to yourself regarding whether to engage in a practice, whilst also noticing our tendency to be lazy or put things off. When things are particularly stressful in our lives is when mindfulness becomes most beneficial, yet it can also be the time when practice is the most challenging. Continuing to practice regularly when things are relatively calm is a good way of developing our skills and confidence when conditions are more supportive of our practice:

2. I feel that being hyper-vigilant and forever on alert is an essential part of my role and I am not sure how I can be mindful or in the moment when it is important that I am accountable and responsible for the health and safety of all of my patients.
It is possible to be mindful in any situation that we find ourselves in and being mindful does not mean that we are not able to meet our responsibilities or that we forget everything. It is not about shutting our minds down or disconnecting from what’s going on. However, as we become more mindful, there may be a shift in how we relate to the pressure placed on us. When we consciously bring awareness to where we are and what is going on and stop being so caught up in our thoughts about what’s going on, which may well be adding to our sense of anxiety, we may deal more effectively with what is in front of us.

Imagine that you have to carry out a procedure with a patient and you know that you are pushed for time, constantly ruminating on thoughts such as ‘I must finish this soon as I need to write up those notes and speak to that colleague,’ ‘I don’t have enough time to do this properly,’ ‘I am not good at doing this,’ ‘What if I get it wrong?’ will not mean that you are more likely to do the procedure well. Constantly thinking about the mistakes that we fear we might make does not protect us from making them – it actually draws our attention away from where it needs to be, on the patient, and it also fuels a negative thought cycle which may contribute to decreased confidence and burnout. Another important aspect is that if we are present, we are better able to identify creative solutions to the issues that cause stress and we become better able to identify and meet our own needs. This may include looking after ourselves better as well as identifying steps to reduce stress in the workplace. It may, of course, also include leaving a job role that becomes too overwhelming for us.

However, we don’t expect you to blindly believe us. It is important that if you are curious about the benefits of mindfulness practice that you then find ways of engaging in mindfulness practice – it is not about transforming everything you do overnight but about starting slowly and taking small steps during your day so as to become present and aware.

3. I would like to feel that I can end my day but I often take home my concerns about clinical situations at work or issues with colleagues. I would like to feel that I could leave some of this worry at work. It can feel as though, emotionally, I am on call 24/7 and, sometimes, it is almost a relief to return to work to allay these anxieties.

Earlier in the chapter, we introduced the work of Halifax (2014), who developed the GRACE model of compassionate care-giving. She focuses on endings in recognition of the importance of consciously ending interactions with patients. This can also be applied, and perhaps is even more important, when marking the end of a shift so that you are able to consciously move into other roles in your life and put down the role of being a nurse. Any mindfulness practice aims to develop our capacity to be present, which means that when we are at work, we can be fully present at work, and when we are at home, we can fully engage at home. However, there are particular ways of using mindfulness practice to end your workday:

- When you get into your car at the end of the day, put you key into the ignition and, before you turn it, take several breaths and check in with yourself.
On the drive home, spend some time without the radio on and be aware of the experience of driving, particularly bring attention to what you are seeing, noticing how this familiar route looks at that time.

If you use public transport and a phone and headphones, use that time to do a short guided mindfulness practice.

Place a small notepad in your car or bag so that if anything work-related occurs to you on the way home, you can write it down before arriving home.

On arriving home, take a pause before entering the house, consciously acknowledging that you are now home and that you can choose to be present here.

I find it hard to give undivided attention to my patients as I feel that I am always preoccupied with the next ‘task’ or being aware that there is a job that I have not completed. I like to think or hope that my patients are not aware of my inattention but I am sure that there are times when they are.

Being distracted this way is very common in today’s society, which places a lot of value on being busy. That is not to say that your job is not busy! But even within the busyness, we can learn to pay closer attention to what we are doing at any given moment. Sometimes, we might confuse being busy with being efficient but, from my experience, these are not always the same thing. Research suggests that we are actually very poor multitaskers and that mistakes occur due to distraction. Healthcare settings can be chaotic; disruptions and interruptions are significant contributory factors to errors such as drug-administration errors (Kreckler et al., 2008). Perhaps there are times where our perceived hyper-vigilance is an illusion and, in fact, we are in a state of inattention. Practically, we need to consider how to focus and explore ways of ending the previous task before consciously moving our attention to the task in hand (reread the above answer for suggestions on how this might be achieved). It is also important to bear in mind that mindful presence does not need to take up more time and that it may help us to be more efficient and effective, thereby avoiding mistakes and saving time in the long run.

With regards to whether or not patients notice if you are distracted, just consider your own experience for a moment: Can you recall a time when you were trying to talk to someone and they are clearly not engaged with you? It can be quite hurtful and alienating, particularly if we are feeling vulnerable at the time, so it is unlikely that patients are not aware. Furthermore, it is not only the patients who suffer from your lack of presence, it also detracts from your experience as a nurse. Ultimately, you are there to care and, although your role has many different facets, if you are not actually present in those moments of care delivery, you also miss out on the joy and connection that can arise from them which make the work of nursing worthwhile. There are probably not many nurses who would describe writing notes or care plans as one of the more rewarding parts of their role. But, as we said above: when you are writing the notes, be present to writing notes; when you are caring for a patient, be present with the patient; when you are talking with a colleague, talk with a colleague.

When these tasks are interrupted – as they inevitably will be because you are more aware – you can actively choose how to deal with the interruption rather than just reacting. Is it urgent? Does it require your immediate attention? In which case, you
choose to shift your attention to the new task. Or can the interruption wait? Be clear with the person who has interrupted you about how much time you need before you can deal with them. Responding in this way means that you don’t get pushed and pulled like a bottle on the waves of the sea. However, if you do find yourself getting completely caught up in events, be kind to yourself as you notice this and take a few conscious breaths or feel your feet on the floor as a way of becoming present and reengaging in a more conscious way.

Conclusion

Mindfulness is currently a bit of a buzzword and is often portrayed as something of a cure-all. Although we don’t agree with all the ‘hype’, we do feel that it is useful and, when developed through regular practice, is supportive of nurse well-being as well as effective and compassionate patient care. A last point that we would like to make is that, although much of the discussion in this chapter has been about acceptance and letting go, mindfulness is not about becoming passive or submissive, gallantly coping with whatever is thrown at you. Attention is rightly being drawn to the current stresses in the healthcare working environments and the risks posed by nursing burnout and distress to the quality of care provision and the harm to nurses themselves (see McPherson et al., 2016). Action needs to be taken in order to ensure that the rights of nurses are protected and that working conditions for nurses are improved. However, paradoxically, acceptance of the way things are in the moment can free up energy and facilitate creative action to effect change. Developing a mindfulness practice is therefore not about sticking your head in the sand or being walked all over in the name of ‘acceptance’ and ‘letting go’. It is about being present with what is, both within ourselves and in the world, and then taking action in accordance with our values. Sometimes, this may include advocating for change in the workplace, standing up for a patient or perhaps a career change. But, whatever it is that we do, it is done with awareness, informed by our values and aspirations and not as a knee-jerk reaction, based on old habits and preconceived ideas.

References


This chapter addresses the following competencies:

**Domain 1: Professional values**

*Competencies:*

7. All nurses must be responsible and accountable for keeping their knowledge and skills up to date through continuing professional development. They must aim to improve their performance and enhance the safety and quality of care through evaluation, supervision and appraisal.

8. All nurses must practice independently, recognising the limits of their competence and knowledge. They must reflect on these limits and seek advice from, or refer to, other professionals where necessary.

**Domain 4: Leadership, management and team working**

*Competencies:*

4. All nurses must be self-aware and recognise how their own values, principles and assumptions may affect their practice. They must maintain their own personal and professional development, learning from experience, through supervision, feedback, reflection and evaluation.

6. All nurses must work independently as well as in teams. They must be able to take the lead in co-ordinating, delegating and supervising care safely, managing risk and remaining accountable for the care given.
Introduction

The purpose of this chapter is to recognise and understand the importance of support to develop into an autonomous practitioner during your transition from student nurse to newly registered nurse. This chapter will help you to identify what support systems
are currently available and how this can affect your job performance. The chapter will also help you to identify when and how you should commence clinical decision making in order to develop your autonomy. The chapter will begin by introducing you to imposter syndrome and the impact this can have on your transition. It then moves on to explore transition support and how this will change from mentorship to preceptorship or another form. You will then have an opportunity to explore what supernumerary status means as a newly registered nurse and how feedback should be provided. Finally, the chapter will identify what other forms of support are available and how this will help you to develop your autonomy and clinical decision-making skills.

Scenario: Marek

Marek is a newly registered nurse who obtained a first staff nurse post in a local NHS Trust. Marek was appointed to a preceptor, Gayle, on the first week of employment.

Although Marek felt competent and knowledgeable as a third-year student, the position of a registered nurse with the increased responsibility and accountability led to feelings of self-doubt and low confidence.

On commencing employment Marek found that Gayle and the other staff appeared too busy to help answer any questions raised. As time passed Marek became increasingly anxious and did not fully understand the role of the staff nurse and what was expected. This led to feelings of being an imposter. This job did not match expectations and Marek began to have sleepless nights, phoned in sick and started to consider whether this was the right profession.

Finally, Marek decided to approach Gayle, an experienced registered nurse, to express these concerns. Gayle was surprised and had thought that Marek was coping. Once this misunderstanding was addressed, although Gayle was often busy with workload responsibilities, Marek’s questions were always answered, and time was made to discuss caseload and offer guidance. Gayle would often work alongside Marek and offer feedback, encouragement and support on any progress, this helped to improve Marek’s confidence.

Marek discovered that the Trust also offered occasional study days for newly registered nurses. When attending study days Marek developed existing skills as well as skills required of a registered nurse. Marek met with other newly registered nurses, shared stories, experiences and gained valuable support.

This scenario is intended to highlight the potential for newly registered nurses to leave the profession due to their expectations not matching experiences in practice and the lack of personal resilience and coping strategies; this is demonstrated in figures collated by the NMC where during 2016–17 more than 29,000 registered nurses allowed their registration to lapse.

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This chapter aims to help you to identify and utilise the support and resources that will be available to you when you make the transition from student to newly registered nurse, and to recognise some of the potential problems that you may face. It is anticipated that once you are familiar with the concept of preceptorship, the role of your preceptor/support person and your own responsibilities as a preceptee/newly registered nurse you will be able to build your own network of support, advice and feedback in your chosen clinical area.

Imposter syndrome and transition

It may seem surprising that many high achieving individuals feel like a fraud when they first register as a professional and take up their first post. They think they haven’t the skills and knowledge others believe they have. Feeling like this is more common than you think and most people at some point in their life will feel like a phoney. However, when feeling like a fraud gets out of control it can develop into something called imposter syndrome (Kearns, 2016) and this can affect how you think and behave.

Imagine what it will be like on your first day as a newly registered nurse in your first post, wearing the uniform of a newly registered nurse. You are charged with the responsibilities that come with the role and no doubt you will have had both positive and negative feelings; being a newly registered nurse can be both exciting and unnerving.

Don’t worry you are not alone!

As you have read in Chapter 1, the transition that student nurses go through to becoming newly registered nurses can be both exciting and stressful. Kramer (1974) described how newly registered nurses experienced ‘reality shock’. This reality shock occurs with the transition from education to the clinical setting where there are different priorities and pressures. Seminal research by Duchscher (2009) referred to the ‘transition shock’ that nurses experience as they realise that they are professionally accountable for their actions and need to rapidly become acquainted with increased autonomy and local responsibilities.

Indeed, the transitional experiences of newly registered nurses are also consistent with those experienced by other health professionals and you may even feel as though you are an imposter like Marek in the scenario (Mandy and Tinley, 2004; Morley, 2009; Kearns, 2016).

The adjustment from education to full-time practice and the nurse’s ability to integrate themselves in their new environment will hasten the transition and lessen the shock. From the moment nurses are registered, they are autonomous, accountable practitioners (NMC, 2015). It is clear then that those feelings of stress and fear felt during this time are often linked to high expectations of yourself and how you will meet your own
Transition support

and others’ expectations. There is then a need for a newly registered nurse to form functional relationships with colleagues, to be integrated into the ward team and subsequently to develop into the role.

The following case study from an interview of a GP by Hugh Kearns demonstrates the impact of transition.

**Case study**

*Entering general practice training as a junior registrar was a completely different story. With just you, the patient, and a supervising specialist GP watching your progress, you are completely exposed. And this, coupled with the fact that junior registrars are going to make mistakes, made for a very humbling experience. I couldn’t count the number of times I reached the conclusion that being a doctor was just not for me. I regularly thought about my ‘fall back’ options, going back to research, perhaps teaching, or maybe stacking shelves at the supermarket.*

**Activity 7.1 Reflection**

On a piece of paper write down how you are feeling now about your transition, reflecting on the case study above. Now go to your SWOT analysis formulated from Chapter 2 and reflect on what you have found.

*As this activity is based upon your own reflection, there is no outline answer at the end of the chapter.*

By getting you to reflect on the GP’s story and your SWOT analysis above we wanted you to be able to distinguish whether you were a real imposter. We hope you have gathered that you are not, as a real imposter is a person who pretends to be someone else in order to deceive others, especially for fraudulent gain; whereas once you see your SWOT it will help you to realise how competent you are. What you may be sensing are imposter feelings; that is, feelings that you are a fraud, and when you explore the facts you are not. If you continue to feel like an imposter a lot of the time despite evidence to the contrary such as Marek in the scenario and this affects how you think and behave, you may have developed imposter syndrome. In this instance, you need to seek help. Table 7.1 lists imposter breaking strategies by Thinkwell that you may wish to consider.

You will note that reservations and worries can hold us back, so the best strategy when you are beginning to feel like an imposter is to act. For example, if we refer to Marek in


<table>
<thead>
<tr>
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<th>Transition support</th>
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<tbody>
<tr>
<td>1</td>
<td>Realise that imposter feelings are normal</td>
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<td></td>
<td>Most people have imposter feelings from time to time. It’s normal to question yourself, to ask how you’re going. Then you need to look at the evidence.</td>
</tr>
<tr>
<td>2</td>
<td>Know your imposter moments</td>
</tr>
<tr>
<td></td>
<td>There will be times when you are more likely to experience imposter feelings. If you know your imposter moments, then you can prepare yourself.</td>
</tr>
<tr>
<td>3</td>
<td>Objective standards of success</td>
</tr>
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<td></td>
<td>Before you start on a project or task, write down what you would consider a success. This will stop you changing the goalposts later.</td>
</tr>
<tr>
<td>4</td>
<td>Setting realistic standards</td>
</tr>
<tr>
<td></td>
<td>Set goals and standards that you can achieve. If you set outrageous standards, it makes failure more likely and you might avoid starting at all.</td>
</tr>
<tr>
<td>5</td>
<td>Prepare for mistakes</td>
</tr>
<tr>
<td></td>
<td>Mistakes can stir up imposter feelings. Since mistakes are inevitable, it is a good idea to prepare yourself. Expect to feel annoyed but then decide what you will do.</td>
</tr>
<tr>
<td>6</td>
<td>Mind your language</td>
</tr>
<tr>
<td></td>
<td>Stick to the facts. Was it just good luck or did you work hard? Did others do all the work or did you contribute too?</td>
</tr>
<tr>
<td>7</td>
<td>Get external evidence</td>
</tr>
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<td></td>
<td>Rather than just relying on your opinions, seek out evidence, ask others, get facts.</td>
</tr>
<tr>
<td>8</td>
<td>Do some behavioural experiments</td>
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<tr>
<td></td>
<td>Try things out to see whether your assumptions are true, for example when in practice ask for feedback on the care you give or clinical decisions you make.</td>
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<tr>
<td>9</td>
<td>Create a fact file</td>
</tr>
<tr>
<td></td>
<td>Write down the facts in a fact file. Use this when an imposter moment strikes.</td>
</tr>
<tr>
<td>10</td>
<td>Create a brag file</td>
</tr>
<tr>
<td></td>
<td>This will help you keep a record of your achievements and positive feedback.</td>
</tr>
<tr>
<td>11</td>
<td>Remember that you are in charge</td>
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<tr>
<td></td>
<td>Even though they may be compelling, remember feelings are not facts.</td>
</tr>
</tbody>
</table>

Table 7.1  Imposter breaking strategies

the scenario, Marek should have understood that feeling like an imposter was normal. Using reflection to help, Marek could have identified that feeling anxious prior to each shift because of fear of not knowing how to respond to the demands of patients and members of the interprofessional team were ‘imposter moments’. By using personal development planning with support from a preceptor, Marek could identify long- and short-term goals in relation to fear of not knowing how to respond to the demands of others and used objective feedback to measure success.
Transition support

Setting realistic goals, with actions and resources that address who to ask and how to ask for feedback that reflects both personal progression and what to do when things go wrong. Marek could assess professional development and ability to cope. This would be from factual information, rather than a personal, one-sided emotional response. The benefits of a PDP also allows Marek to create a ‘brag and fact’ file that visibly demonstrates achievements during the period of preceptorship. Please refer back to Chapter 3 for further information.

The physical presence of a supporting individual (moving from mentorship to preceptorship/transition support)

<table>
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<th>Concept summary</th>
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What is a preceptorship/transitional support programme? How is it different to mentorship?

To support the transition from student nurse to newly registered nurse many healthcare settings have adopted a Staff Nurse Preceptorship Programme. The NMC (2006) defines preceptorship as a period to guide and support all newly qualified practitioners to make the transition from student to develop their practice further.

Preceptorship is not a new concept, the need for support was formally recognised in the UK in 1986 and professional bodies at this time recommended a period of learning after registration followed by a lifelong programme of continuing education. The drivers to implement supportive structures for newly registered nurses were based on two main features: to alleviate the transitional challenges of new practitioners to reduce the number of newly registered nurses leaving nursing as soon as they qualify, and a concern about the fitness to practice of newly registered practitioners.

Since 1986 there have been key documents that have ensured preceptorship has remained a recommendation for sound professional practice. The table below provides three key external drivers that promote the need for preceptorship. However, as all guidance on preceptorship is optional and not mandatory, some employers may offer a preceptorship programme, whereas others offer other forms of transition support.
In 2010 the Department of Health launched a ‘preceptorship framework for newly registered nurses, midwives and allied health professionals’ to set clear standards for preceptorship.

Preceptorship is therefore a system put in place to support the transition phase for newly registered nurses as they continue their professional development, building confidence and further developing competence to practise and provide structure and direction. Preceptorship continues to feature as a priority as the Shape of Caring Review (Willis Commission, 2012) has recommended 1-year preceptorship with an employer following registration. Preceptorship is an integral part of enabling a newly registered nurse to practise safely unsupervised. As such it is a very important part of the development and transition route to independent practice; the programme may feature completion of mandatory workbooks, reflections and study days (to name some activities). Failure to advance at the two progression points within the first six to twelve months of a preceptorship programme could compromise a nurse’s career or registration.

At the interim and end of the preceptorship period reviews should be held. The discussion at the reviews should not come as a surprise to the preceptee as feedback should be consistent throughout the programme with regular feedback on progress. If the preceptee has not provided sufficient evidence that they have met the required standards, the line manager as well as the preceptor will record which of the standards or performance criteria have not yet been achieved and provide detailed feedback to the preceptee. This will be recorded both in the preceptorship and appraisal documents. At this point it is the line manager who will decide locally whether human resources advice and support should be sought. At this time consideration will be given to either extend the preceptorship period or follow the trust/organisations competency policy; this may include contacting the Nursing and Midwifery Council under Fitness to Practice if the incidents or ill health issues are serious and compromise patient safety.

<table>
<thead>
<tr>
<th>Organisation</th>
<th>Driver</th>
</tr>
</thead>
<tbody>
<tr>
<td>Care Quality Commission</td>
<td>Competent</td>
</tr>
<tr>
<td>Staff – Standard 14</td>
<td>Registration requirements states we must take all reasonable steps to ensure that workers are appropriately supported to enable them to deliver care safely and to an appropriate standard</td>
</tr>
<tr>
<td>Department of Health</td>
<td>Developing the Healthcare Workforce; DoH preceptorship framework (for newly registered nurses, midwives and allied health professionals March 2010)</td>
</tr>
<tr>
<td>Nursing and Midwifery Council</td>
<td>Recommendation 21 of the NMC’s ‘Fitness to Practice’</td>
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</table>

Table 7.2 Key external drivers
When you are considering which staff nurse post to apply for or attending job interviews it is wise to discover whether your prospective employers have a preceptorship programme in place. Ask how long it lasts and what it will include, consider whether this programme will be of benefit to you, will it address your development needs. Cross-reference the programme against your SWOT analysis formulated in Chapter 2. For further information on applying for a staff nurse job see Chapter 8.

It is important that the programme you select is suitable for your needs as preceptorship programmes can be varied. Robinson and Griffiths (2008) and Chapman (2013) called for preceptorship programmes that fit the needs of the individual and should be a way to build confidence and further develop competence and not as a way to meet any shortfall in pre-registration education (DoH, 2010).

The need for a preceptorship/support programme is acknowledged in policy, though details of what is needed are sometimes unclear. Literature demonstrates a variance in length and content of both preceptorship and support programmes and authors have found difficulties in identifying individual learning needs of newly registered nurses (Evans, Boxer and Sanber, 2008; Darvill, 2013, Strauss et al., 2015).

You may find that support programmes on offer vary in length, content and structure, it is a good idea to discuss the details of any support programme during your interview, that way you may find your ‘right fit’. Some nurses may prefer a more structured programme such as their experience during nurse education, which focuses on attaining specific clinical competencies; however, some nurses may prefer the programme to focus on other important aspects of preceptorship such as peer support/networking and socialisation. A recommendation made by the National Nursing Research Unit (Robinson and Griffiths, 2008) concludes that any formal Structured Nurse Preceptorship programme should be speciality specific, and tailored toward the individual nurse’s needs.

The support programme you choose should provide a supportive function, as if it became a task it could add more pressure to an already stressful time and serve to have the opposite effect to the supportive, developmental programme it set out to be. Some degree of formal outcomes such as developing your competencies may, however, be beneficial in developing the skills pertinent to your new role. Therefore, the programme you choose should be a balanced period of support and needs to be specific, individualised and not overly onerous. The Department of Health (DoH) is specific when it states that the programme should be seen as a way to build confidence and further develop competence and not as a way to meet any shortfall in pre-registration education (DoH, 2010, p10). It is now time for you to think: What do I want from my support programme? You may wish to read the systematic review that is included in the further reading section of this chapter.

Now that you have surveyed and reflected on your needs during your transition it is important to explore self-confidence. According to the literature, self-confidence could be your perception of your ability to interact with patients, families and colleagues.
Activity 7.2 Reflection

Now that you have read more about preceptorship/transition support what do you think you would want/need to be included in your preceptorship/support?

Review your Personal Development Portfolio, Practice Assessment Document, or a Band 5 Job Description & Specification. Undertake a SWOT analysis (as demonstrated in Chapter 2) which will help you to identify actual/potential areas for development that should be included in your transition support programme.

While writing your list you may have used the word ‘competent’ and the word ‘confidence’. What do you understand by the term confidence and the term competence? Write down your answer.

Consider how the preceptorship/transition support programme can develop your self-confidence and your clinical competence.

You will find an outline answer at the end of the chapter.

and safely carry out your new role in the clinical setting. Competence however, predicates the application of your knowledge and skills in responding appropriately to the dynamic patient-care environment (Roach, 2002). You could say that when you develop and safely demonstrate your competence you will then increase your confidence. The preceptor or support individual is charged with the role of guiding newly registered nurses and helping them to apply theory in practice; when you work with your preceptor/support person and demonstrate to them your competence, then this will help you to develop your confidence.

Supernumerary period

Supernumerary status means that you are additional to the clinical workforce and will spend time as such. A preceptorship/transition programme may allow a newly registered nurse to have time in the clinical area as a supernumerary member of staff. This will enable you to spend periods working with your preceptor/support person to learn from them and not as a member of staff with an allocated work load. This status would allow you attend study days where you will learn and develop alongside a group of your peers away from the clinical area, you may also be expected to complete specific outcomes and competencies that your employer has identified to help you to develop basic knowledge, skills and attitudes to perform your new role. This will enable you to build on the knowledge, skills and competences acquired as students in your chosen area of practice, laying a solid foundation for lifelong learning. The length of time your supernumerary status lasts can be anything from 15 days to a month. However, this is
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dependent on many variables such as appraisal of your current knowledge and skills and how quickly you adapt to the new clinical role.

Role of the preceptor/support person

A preceptor/support person has been defined as a registered practitioner who has been given formal responsibility to support a newly registered practitioner through preceptorship (DoH, 2010, p6).

As a student nurse, you would have worked closely with a mentor during each clinical placement. Working with a preceptor/support person should allow you to receive both support and education. There are however some small differences in the two roles. A mentor is required to undergo training and holds a qualification to perform the role. Currently there is no specific preparation for the role of preceptor/support person. If you are familiar with the differences this may help you to get the best from your preceptor.

Although your mentor would have been responsible for verifying your competence as a student the preceptor/support person will be there to help you consolidate your learning and support you through the transitional process to become an autonomous practitioner. Working alongside your preceptor/support person you will observe how they demonstrate their professional attributes, such as communication skills, problem solving, prioritising and decision making; you could look upon your preceptor/support person as a role model.

You may have noted that the ability to give constructive feedback is the first attribute a preceptor/support person should possess. It is therefore important to explore what feedback, the types of feedback, how to receive it and what to do with it.

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Scenario: Marek and Gayle

If we look once again at how Marek and Gayle developed their preceptor/preceptee relationship we can appreciate that each preceptee will have individual development needs and will require varying levels of support to help consolidate their learning through the transitional process toward becoming an autonomous practitioner. By working alongside Gayle, Marek was able to observe how Gayle demonstrates professional attributes such as communication skills, problem solving, prioritising and decision making; you could look upon the preceptor/support person as a role model.

Gayle also provided support by simply taking time to talk with Marek, answer questions and offer guidance and feedback, this resulted in Marek feeling confident in the new role to seek out further development opportunities independently.
Feedback – why is this important?

Let’s go back to the initial scenario of Marek: in order for Marek to advance in knowledge and skill development, Marek required constructive feedback from Gayle. As a student, Marek would be aware of the concept of assessment and feedback in relation to meeting the requirements in both theory and practice across the duration of their pre-registration nursing programme. Marek would then find it difficult if feedback was not forthcoming in the new role as a registered nurse.

A dictionary definition of feedback is information about reactions to a person’s performance of a task, which is used as a basis for improvement. However, as a newly registered nurse, feedback is considered detailed information about the assessment between a trainee’s observed performance and a standard; given with the goal to advance the trainee’s performance (Van der Ridder et al., 2008). In Marek’s case feedback was important, in order to assess Marek’s competence against the job roles and responsibilities of a registered nurse on a general medical ward.

Constructive feedback is the method of offering feedback about knowledge, skills and attitudes that are below the required level of competence and ability with the aim to improve it. It can involve informing the newly registered nurse of the standard required and/or providing them with suggestions about how to meet them.

Unconstructive feedback, however, is the process of providing feedback to a newly registered nurse deprived of any intention of improving their knowledge, skills or attitudes. This type of feedback is negative, often destructive, and should be avoided.

Constructive feedback should be:

- based on observed skills and behaviour;
- given on a regular basis;
- both verbal and written;
- full of probing questions about the newly registered nurse’s own assessment of their knowledge, skills and values;
- related to current skill and knowledge level and desired goals;
- clear and focused;
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- positive and promote a change in performance and meeting of learning objectives/skills;
- given in sizeable chunks so any changes can be addressed in a systematic way, too much information and the newly registered nurse may feel overwhelmed;
- socialising the newly registered nurse to the profession;
- specific with information about desired improvements or corrective changes alongside a supporting rationale;
- based around further actions for the newly registered nurse to work towards as part of either an action plan or appraisal process. For example: being provided with opportunities to develop your knowledge, skills and experiences; being allocated workload based on previous experience and capability level; being given the autonomy to work independently to gain confidence through experience;
- encouraging reflective questioning in order to develop the newly registered nurse’s critical thinking and decision-making skills that can help them to analyse current knowledge, skills, attitudes;
- given in private whenever possible;
- a two-way process so the newly registered nurse can share their views on the feedback they have just received (Duffy, 2013).

As you may have noticed, feedback is a complex process; therefore any information provided needs to be meaningful and clearly linked to competencies set out in the transition/preceptorship programme.

The Preceptorship Framework for Newly Registered Nurses, Midwives and Allied Health Professionals (DoH, 2010) suggests that all newly registered staff joining an organisation should have at least two development reviews within the first 12 months of employment. The purpose of these assessments is to establish the progress a preceptee is making towards criteria and competencies defined by the line manager and linked to indicators such as those in the KSF (DoH, 2004). This allows for the objective measurement and feedback of the preceptee’s knowledge, skills, and attitudes by the preceptor; for example, using an assessment of a preceptee against KSF Core Dimension 1: Communication. In this instance the appraisal by the preceptor would be based upon the nature and extent of the communicating in the preceptee’s everyday job. Exploring manner, tone and words used when communicating. Preceptors may use the acronym DOVE: documents, observations, verbal and electronic (NHS Scotland, 2010) to provide them with evidence to measure how the newly registered nurse has met the indicator or competence as found in Table 7.3.

While positive, negative and constructive feedback can enhance learning, unconstructive feedback may have a detrimental effect on both personal and professional development. Providing no feedback can result in a false level of security for the newly registered nurse. They may think they are doing well and have an enhanced sense of confidence and are not having any of their knowledge, skills and attitudes observed and reflected upon. This can subsequently affect patient care as unsupported newly registered nurses often hesitate to ask questions or seek advice as they feel they are not
Transition support

Table 7.3 Examples of evidence used for a development review

<table>
<thead>
<tr>
<th>Core dimension/ Clinical competence</th>
<th>Examples of how the dimension or competence will be met</th>
<th>Date met</th>
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</table>
| KSF core dimension 1: Communication | *Documents:* forms and documents, for example, risk assessments, care plans, treatment records, completed order records, letters and thank you notes from patients, reflective accounts.  
*Observations:* carrying out tasks, talking with colleagues, patients and others, reporting incidents.  
*Verbal:* question and answer sessions on current policies and procedures, discussions on scenarios.  
*Electronic:* e-learning achievements and presentations. |          |

coping or not able. This can end in errors and incidents where the quality of patient care is affected. Unconstructive feedback, however, usually lacks detail, offers no recommendations for how knowledge, skills and attitudes can be improved, and uses rude words or ones with negative connotations. It is often intended to offend and can include undeserved, personal attacks, leaving the newly registered nurse defensive.

Receiving feedback

It is important to consider the skills needed to receive feedback whether that be good or not so good. Listening carefully and being open to what is being said, making sure you have fully understood this before deciding on how you will respond. To ensure you have fully understood the feedback ask specific questions to avoid any misunderstanding and to clarify the points being made. Try to frame questions to get as much information as possible to ensure improvement in the future; e.g. when I did this I should have … is this correct? Not all feedback will be positive, therefore the newly registered nurse needs to be aware of the emotional effect that feedback may generate, particularly if this is not as positive as expected. There needs to be some self-awareness and self-control if feedback causes an emotional response, therefore some understanding of emotional intelligence is essential. For example, you can respond to feedback in four main ways:

Defensive: where you see the feedback as a personal attack aimed at your personal identity, and your emotions respond as though it was a threat to your existence. Being defensive means the feedback is often ignored, denied and creates anger and retaliation. By reacting this way, you will not learn anything and severely affect the preceptor–preceptee relationship.

Dispirited: where you take on board every piece of feedback without checking to see whether this is factually correct and supported. Responding to feedback in this way
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creates a strong emotional response, viewed as a personal attack and demoralising. This then leads to a refusal to learn or to change one’s behaviour.

Dismissive: when feedback is not taken seriously, an assumption is made that the feedback given is wrong, or the person giving feedback is not to be trusted. It does not create an emotional response, but there is no engagement with the opportunity to learn from the feedback given.

Open: reacting to feedback in an open way allows you to reflect on your recollection of the behaviour or actions, check on the facts and take the criticism or praise on board.

Remember that your first response to feedback may change when you have had the opportunity to examine it in a more detached way later. By being open to feedback you can assess whether the facts are correct and make allowances for the skills of the person who delivered it. In the next activity we would like you to reflect on some feedback you have received as a student and how with time you may have changed your response to it.

Activity 7.4  Reflection

Think back to when you received constructive feedback in the past, for example from a lecturer on a piece of theoretical assessment you had submitted, or feedback from your mentors/personal tutor about your knowledge and skills. Which of the four main ways did you initially respond to receiving that feedback? Now that time has passed, review the facts and the skills of the person who delivered it. Has your response changed?

As this activity is based on individual experiences there is no outline answer at the end of the chapter.

The ‘feedback sandwich’

Feedback is more likely to be accepted and acted upon if it is seen to be ‘balanced’ in that it is neither overly critical nor positive but provides a clear indication of the good and not so good. The ‘sandwich’ presents any negative aspects of feedback between two positives that offers a more balanced approach to the feedback process (Dohrenwend, 2002). For example; your written plans of care have improved and are more specific and focussed than when you started … your numeracy skills still need some work,
specifically around intravenous fluid rates as you continue to have problems with this. I can really tell that you care about developing yourself as a nurse.

Networks and resources

In Chapter 4 you explored accessing support to maintain your personal health and wellbeing from the family and friends activities feature as a support mechanism to help ensure you kept physically and mentally well. In the workplace, however, there are other networks and resources that as a newly registered nurse you can call upon to support you during your transition.

Other members of the nursing team (registered and support workers) in the workplace can be a valuable resource to assist in easing your transition. Develop what is called your ‘social capital’ (Melling, 2011) by taking opportunities to build good social relationships. Watch other staff members closely, pay attention to how they work and complete tasks. Some will be excellent role models of how you should conduct yourself while at work and the skills required to provide a quality service. Ask for their advice and help when you are unsure and remember to thank them when they have helped, to show your appreciation.

Other members of the interprofessional team are another helpful resource. Often when newly registered nurses qualify, members of other professions are new registrants also. Take time to get to know who the members of the interprofessional team are, seek their help when you know your limitations, ask them to show you or teach you aspects of their role that may be of benefit to your own knowledge, skills and attitudes. Again, always remember to thank others for their help and support.

Clinical nurse educators can assist your transition with helping to provide practical and skills-oriented training under the supervision of a skilled practitioner.

Study days are encouraged and often a requirement as part of your transitional period. This is for you to develop the skills and knowledge necessary to competently carry out your role, demonstrate that you remain fit to practice and have the necessary skills to provide patients with the highest level of care. Study days, conferences and seminars serve to inform your professional knowledge, by sharing research and best practice – crucial to learning and building up an evidence base from which to draw upon. During your attendance at study days you will also meet other newly registered nurses and staff who can increase your circle of support networks.

Peer support groups are often organised as part of preceptorship/transition support programmes. Support from these groups includes emotional support, new insights and rewards. Peer groups also give encouragement and optimism when you become stressed by the emotional labour of nursing and some of the clinical decisions you have made.
Autonomous decisions about clinical judgements, choices and actions

According to the Royal College of Nursing (2014, p3):

_Nursing is the use of clinical judgement in the provision of care to enable people to improve, maintain, or recover health, to cope with health problems, and to achieve the best possible quality of life, whatever their disease or disability, until death._

To carry this out a nurse must develop and demonstrate autonomy and control over their nursing practice. This in turn has been linked with increased job satisfaction and improved patient outcomes. As a newly registered nurse, a programme of support is needed so that you may develop these skills required to become an autonomous practitioner and make clinical judgements about patient care safely and with support from your preceptor and other registered nurses within the clinical area.

Autonomy and accountability are two major issues that newly registered nurses worry about. However, in order to make effective clinical decisions, which in nursing occur several times a day, newly registered nurses should use information they have gathered using tools of assessment, theoretical knowledge, general awareness and experience to inform the process. Good clinical decision making requires an amalgamation of skills that include: pattern recognition from learning experiences, critical thinking, communication skills using active listening, evidence based practices, team work, sharing and discussion of your decisions with others and reflection.

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**Concept summary**

According to Weston (2010) autonomy represents the ability to act according to one’s knowledge and judgement, delivering care within one’s scope of practice as outlined in current professional, regulatory, and organisational rules. The Nursing and Midwifery Council (2010), as part of the standards for pre-registration education, state that a competency required prior to entry on the professional register is that nurses _must practise autonomously and be responsible and accountable for safe, compassionate, person-centred, evidence-based nursing that respects and maintains dignity and human rights._

Strategies that will help you to become autonomous/independent will include: your preceptor describing expected behaviours and providing opportunity to practise behaviours; senior staff recognising and rewarding your positive behaviours; you role modelling behaviours of autonomy and independence observed; and your support person/preceptor providing constructive feedback when you do not demonstrate positive behaviours.
NHS Scotland (2010) recognised four issues that can have an impact on clinical decision making; these include: knowing the evidence in order to be able to deal with the current patient or situation; knowing yourself and how your and others’ attitudes, values, beliefs and behaviour can impact the care delivered; knowing the patient and their experience, knowledge and current situation in regard to their illness; knowing the environment in order for you to take a considered approach to the decision-making process which may mean bearing in mind team dynamics, ward culture and personalities. For example, you have been asked to carry out a dressing change on a patient with a surgical wound. Using the four issues approach, your ability to care for the patient and their wound would depend upon:

- your knowledge of anatomy and physiology of the skin and the process of wound healing;
- your knowledge and skills of completing wound assessments, wound cleansing techniques and choosing the right dressing;
- your reaction to the wound appearance, odour and/or leakage;
- where you will undertake the dressing change on the ward and do you need assistance;
- who can help and what time you will change the dressing.

A prescriptive method that nurses use to help with clinical decision making is a four-stage process of assessment, planning, implementation and evaluation (Yura and Walsh, 1973). To complete these stages effectively you must consider all assessments and their results using look, listen and feel, then make judgements on the data collected, what is happening, decide what to do, include colleagues in your discussions and evaluate the outcome of the decision. It is important during your preceptorship that you consider activities that will help you to achieve these skills. If you wish to read more about the topic of clinical decision making there are other books in the transforming nursing practice series addressing this topic in some depth such as Standing (2017). A useful way to start to address your skills in clinical decision making is to write a list.

Activity 7.5 Decision making

Considering your new job as a newly registered nurse what activities could you ask to be involved in to develop your autonomy and enhance your decision-making skills.

*You will find an outline answer at the end of the chapter.*

Now you have reflected on the activities you could ask to be involved in to develop autonomy and enhance your decision making, the next section explores what support you could ask for once your preceptorship/transition support ends.
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Clinical supervision

Once the period of preceptorship/transition support has ended, you may feel as though there is no further support for your development. However, many organisations offer clinical supervision. This is defined by the Royal College of Nursing (2002, p1) as:

*an activity that brings skilled supervisors and practitioners together in order to reflect upon their practice. It is a time for you, as a nurse or midwife, to think about your knowledge and skills and how they may be developed to improve care.*

Chapter summary

By reading this chapter we hope that you will be able to comprehend what transition support now means. By completing the reading and activities you should now be able to recognise what to expect in relation to transition support when you commence employment in your new role as a registered nurse. You should also now be able to identify the need for supernumerary status at the start of your job and describe the different types of feedback and recognise the impact this has on job performance. Finally, identifying other networks and resources that can assist with transition support and recognising when you are ready to start to make autonomous decisions about clinical judgements, choices and actions.

Activities: brief outline answers

Activity 7.2 Reflection (page 143)

Competence: the delivery of safe care to a required standard.

Confidence: confidence is an internal feeling of self-assurance and comfort. Confidence as a nurse comes from experience and exposure to as many different patient scenarios and clinical situations as possible.

Activity 7.3 Reflection (page 145)

Here is a list of attributes of a preceptor developed by the Department of Health. Which of these attributes did you consider?

- the ability to give constructive feedback;
- setting goals and assessing competency;
- facilitating problem solving;
- active listening skills;
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- understanding, demonstrating and evidencing reflective-practice ability in the working environment;
- demonstrating good time management and leadership skills;
- prioritising care;
- demonstrating appropriate clinical decision making and evidence-based practice;
- recognising their own limitations and those of others;
- knowing what resources are available and how to refer to a preceptee appropriately, if the preceptee needs additional support;
- being an effective role model and demonstrating professional values, attitude and behaviours;
- demonstrating a clear understanding of the regulatory impact of the care that they deliver and the ability to pass on this knowledge.

Activity 7.5 Decision making (page 151)

Activities that can enable your development of autonomy and clinical decision making include: observing and then participating in ward rounds; observing senior nurses and role models; coaching from your preceptor; attending and participating in staff meetings, case conferences, and best interest’s meetings etc. Then, as you become integrated into the ward team, being put forward for modules of study, study days, becoming a link nurse etc.

Further reading


This essential book covers the issues, themes and principles that nurses practising today should be familiar with. Often aimed at student nurses this book also provides the registered nurse with an easily accessible reference to the issues key to your career. Similar books are also available for other fields of nursing.


This is a useful guide to keep close at hand. It is an essential reference for nurses, not only on the ward but also in every field of practice where patient care is given.


This is a systematic review of the literature that enables you to know what a good preceptorship programme should look like.

Useful websites

**Thinkwell** – a website produced by researchers and practitioners in cognitive behavioural therapy who use the latest psychological and educational research to assist high achievers to achieve maximum productivity.