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

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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.
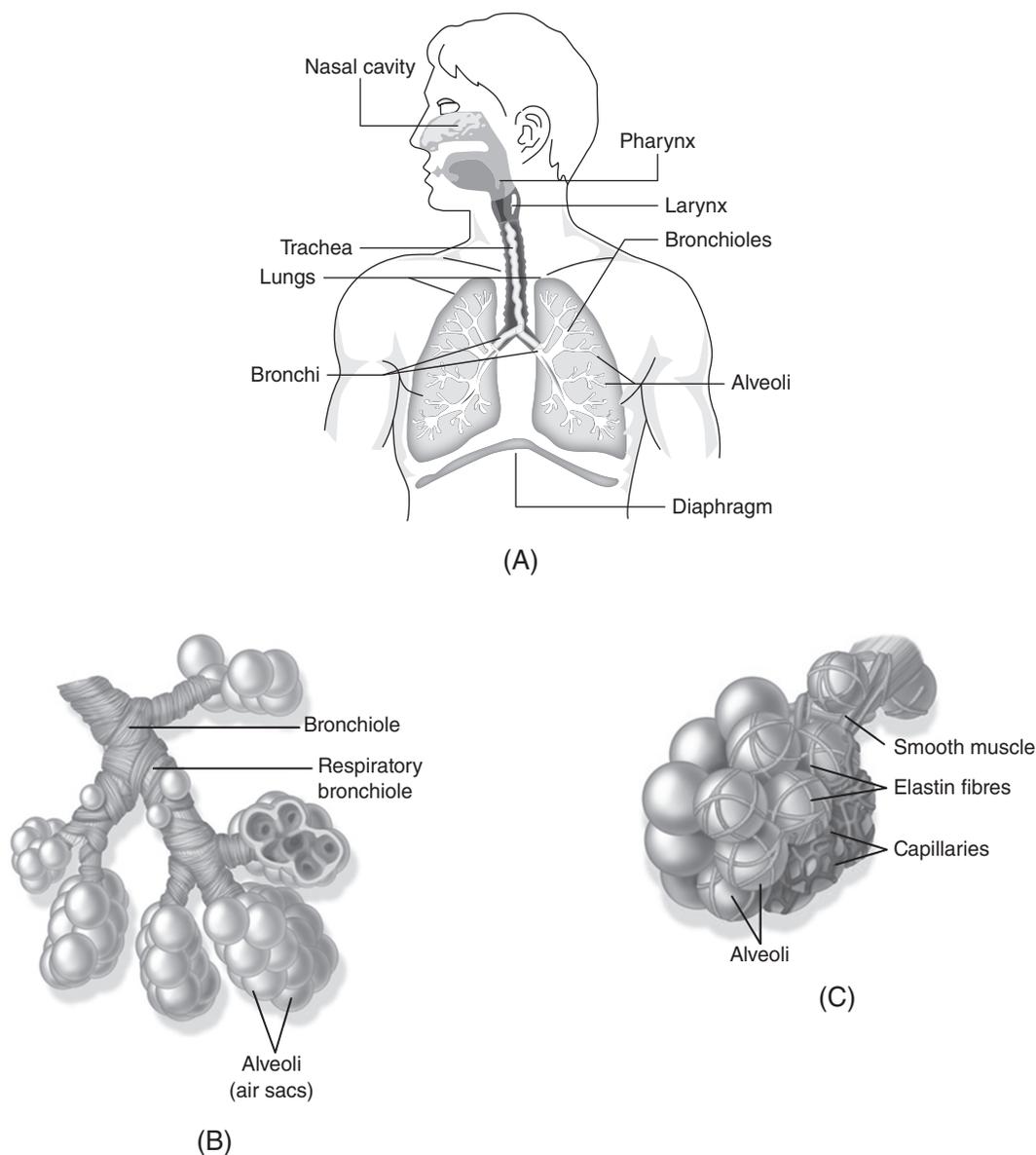

(A)

(B)

(C)

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).
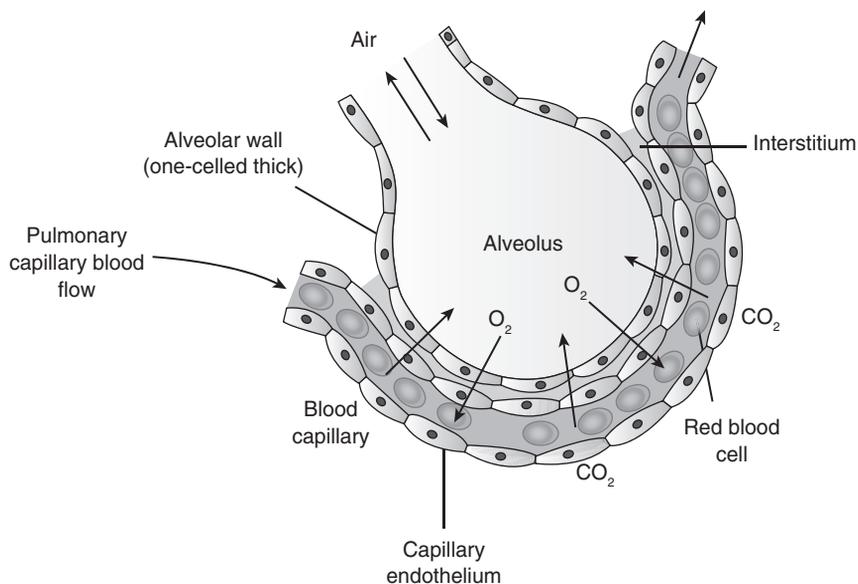

Figure 9.2 A single alveolus showing the structure of the respiratory membrane across which oxygen and carbon dioxide pass by diffusion

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 β_2-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.

Stimulus
Environmental allergens, e.g. pollen, house dust, animal hair, latex
Respiratory tract infections
Exercise
Occupational sensitisers, e.g. isocyanates (from polyurethane varnishes)
Cold air
Ingestion of NSAIDs
Emotional stress
Exposure to bronchial irritants, such as cigarette smoke, perfume

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.

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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 β_2-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.
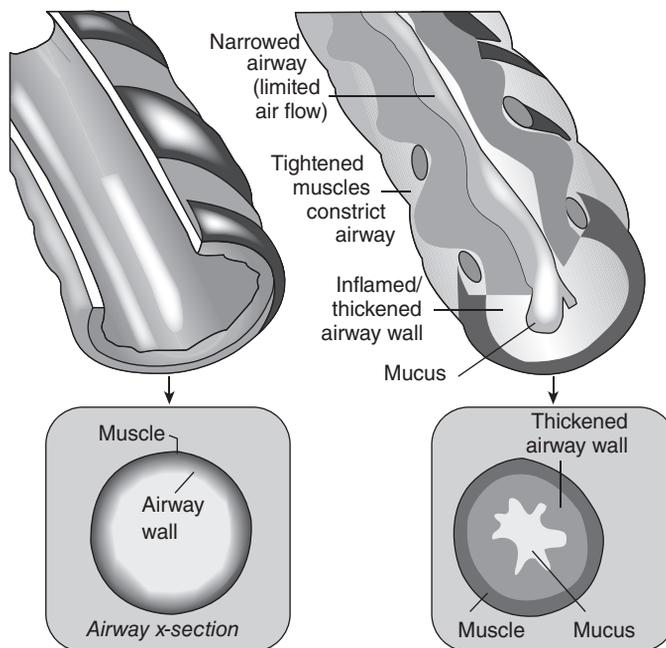
Figure 9.3   Changing occurring in the airways during an asthma attack

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**). 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).
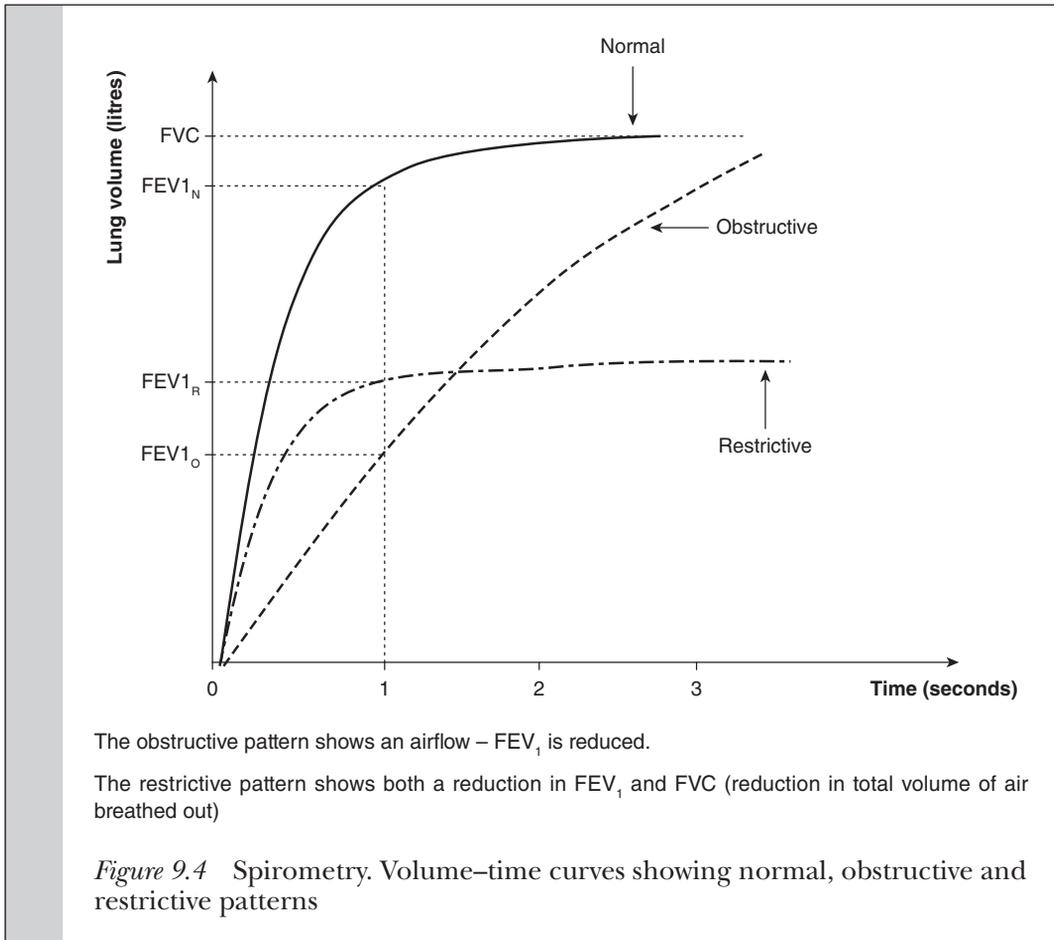
With COPD or other obstructive disease, FEV_1 is reduced due to airway obstruction. FVC on the other hand may be relatively normal, indicating that lung volume has not changed significantly. Restrictive diseases also produce a lowered FEV_1. However, the FVC is reduced as well indicating significant loss of lung volume. Due to this, the ratio FEV_1/FCV 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

Airflow obstruction	% of predicted FEV_1 (NICE 2018b)
Mild	≥ 80%
Moderate	50–79%
Severe	30–49%
Very severe	< 30%

The obstructive pattern shows an airflow – FEV_1 is reduced.

The restrictive pattern shows both a reduction in FEV_1 and FVC (reduction in total volume of air breathed out)

Figure 9.4   Spirometry. Volume–time curves showing normal, obstructive and restrictive patterns

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 α_1-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.
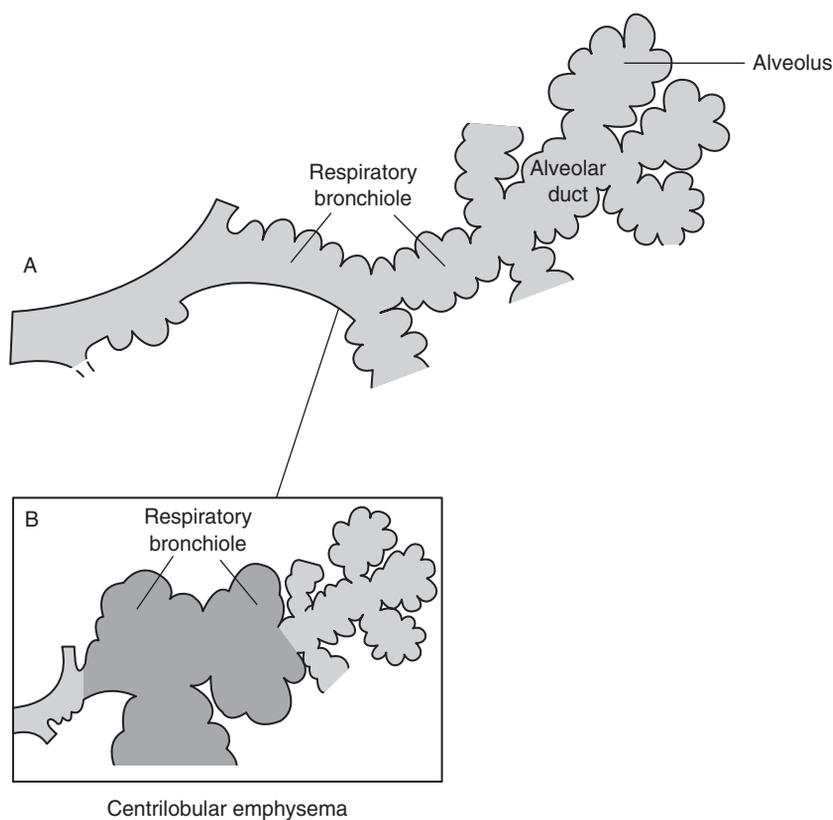

Figure 9.5 Damage to the alveoli shown in emphysema. (A) Shows the normal structure of the respiratory bronchiole and alveoli. (B) Centrilobular damage to the wall of respiratory bronchioles causing enlargement of the airspace

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.
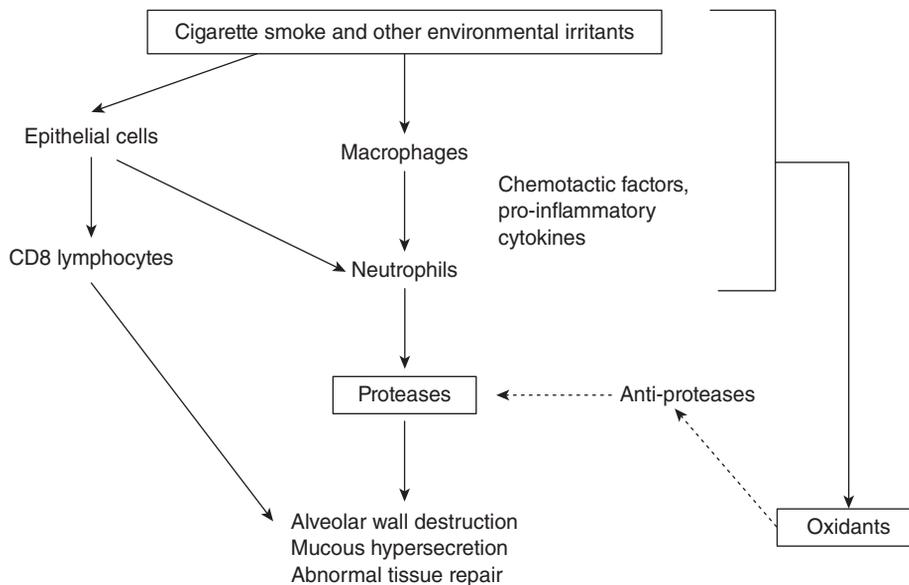
Summary of events leading to alveolar wall destruction

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

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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 FEV_1 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 β_2-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.

β_2-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 β_2-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 – α_1, α_2, β_1, β_2 – 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.

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Target	Effect of sympathetic stimulation (adrenaline/noradrenaline) (type of adrenergic receptor is given in brackets)	Effect of parasympathetic stimulation (acetylcholine) of muscarinic receptors
Lungs – bronchial smooth muscle	Relaxation causing bronchodilation (β_2)	Bronchoconstriction
Stomach and intestines – smooth muscle of wall	Decrease in motility and tone (α and β_2)	Increase in motility and tone
Heart – (cardiac) muscle	Increased rate and force of contraction (β_1)	Reduced rate and force of contraction
Arterioles – smooth muscle	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 (α and β)	No known effect
Liver	Synthesis and release of glucose (from glycogenolysis and gluconeogenesis) (Chapter 12) (α and β_2)	No known effect
Salivary glands	Little or no increase in saliva production	Increased secretion of a watery mucus
Eye – radial muscle of iris	Contraction leading to pupil dilation (α_1)	No known effect
Eye – circular muscle of iris	No known effect	Contraction leading to pupil constriction

Table 9.2   Effects of sympathetic and parasympathetic stimulation of various body organs and tissues

Side-effects and considerations for practice

β_2-agonists used in asthma, such as salbutamol and terbutaline, work directly to stimulate β_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 β receptor. For example, salbutamol also activates cardiac muscle β_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 β_2 and β_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. β_2-agonists must therefore be used with care with other medications, such as diuretics, which also cause hypokalaemia.

Using β_2-agonists with β-blockers (Chapter 8) can cause problems. β-blockers are β_2-antagonists and therefore block the action of β_2-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 β_2-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 β_2-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 β_2-agonists are used for immediate relief of symptoms. Long-acting β_2-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 β_2-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 β_2-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 β_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.

No preventer needed	• Short-acting beta agonist	Move down the table as needed if symptoms deteriorate ↓
Regular preventer therapy	• Add low-dose of inhaled corticosteroid (ICS)	
Initial add-on therapy	• Add long-acting beta-2 agonist (LABA)	
Additional add on therapy	• No response to LABA – stop LABA	Move up the table to maintain lowest controlling step↑
	• Partial response to LABA – increase ICS to medium dose OR consider additional treatment (LAMA, leukotriene antagonist or slow release theophylline)	
High-dose therapies	• Consider increase of ICS to high dose	
	• Consider adding a fourth drug	
Continuous frequent use of oral steroids	• Maintain high dose ICS	
	• Add lowest possible dose of oral steroid	
	• Consider other treatments to minimise steroid use	

Figure 9.7 Stepwise approach for selecting asthma treatment in adults (BTS and SIGN, 2016)

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 β_2-agonist (usually salbutamol), oxygen and an oral or IV corticosteroid. The short acting anti-muscurinic 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

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 (β_2-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 breath-lessness and reduce hyperventilation. The use of bronchodilators will not return the airways to normal and may not improve the FEV_1 greatly. However the quality of life of many patients is often improved with these drugs.

Short-acting β_2-agonist (SABA) and long-acting β_2-agonist (LABA)

These bronchodilating drugs have already been introduced under asthma. Drugs such as salbutamol (a short-acting β_2-agonist) and salmeterol (a long-acting β_2-agonist) are used in COPD to relax smooth muscle in the lungs and cause bronchodilation.

Short-acting muscarinic antagonists (SAMA) and long-acting muscurinic antagonists (LAMA)

If we revisit Table 9.2 we can see that blocking (or antagonising) muscarinic recep-tors 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 recep-tors leading to bronchodilation. Theoretically these drugs could be used in the routine treatment of asthma but in practice they are less effective than β_2-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 β_2-agonists.

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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 β_2-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 β_2-agonist (SABA)
   b)   Long-acting β_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) β_2 receptors on the heart muscle are blocked by salbutamol
   b) β_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. β_2-agonists such as salbutamol act on β_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.

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 (corticosteroid) 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) β_2 receptors on the heart muscle are stimulated by salbutamol

Further reading

BTS/SIGN (2016) *British Guideline on the Management of Asthma*. Available at: **www.sign.ac.uk/sign-153-british-guideline-on-the-management-of-asthma.html**/

This gives more information on the treatment of asthma including algorithms used to choose therapy.

Hickey, AJ (2014) Understanding the impact of inhaler technique on asthma and COPD. *Nurse Prescribing*, 12 (10): 492–6.

This article gives useful information on inhaler technique.

Kumar, P and Clark, M (eds) (2012) *Kumar and Clark's Clinical Medicine* (8th edition). Philadelphia: Elsevier Saunders.

A comprehensive textbook on pathology. Chapter 13 gives a detailed examination of the pathology of asthma and COPD.

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.