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)

This chapter covers

- An overview of the anatomy and physiology of the respiratory system
- Respiratory assessment
- Clinical characteristics and underlying pathophysiology of two common respiratory conditions (asthma and chronic obstructive pulmonary disease)
- Common diagnostic procedures and clinical investigations
- Nursing assessment, care and management of the adult presenting with common respiratory conditions.
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>
<thead>
<tr>
<th>Table 24.1 Clinical features of asthma and COPD (NICE, 2010)</th>
</tr>
</thead>
<tbody>
<tr>
<td>COPD</td>
</tr>
<tr>
<td>Smoker or ex-smoker</td>
</tr>
<tr>
<td>Symptoms under age 35</td>
</tr>
<tr>
<td>Chronic productive cough</td>
</tr>
<tr>
<td>Breathlessness</td>
</tr>
<tr>
<td>Night-time waking with breathlessness and/or wheeze</td>
</tr>
<tr>
<td>Significant diurnal or day-to-day variability of symptoms</td>
</tr>
</tbody>
</table>

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](image)

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 (O₂) is delivered to the tissues (cells) from the atmosphere and carbon dioxide (CO₂) 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).

**Internal respiration**

Deoxygenated blood from the right side of the heart (High in CO₂ and low in O₂)

Capillary from pulmonary arteriole

PO₂ 40 mmHg
PCO₂ 44 mmHg

Capillary

Red blood cell

Oxygenated blood going to the left side of the heart (High in O₂ and low in CO₂)

Capillary to pulmonary venule

**External respiration**

Oxygenated blood going to the left side of the heart (High in O₂ and low in CO₂)

Alveolus

Capillaries

Oxygenated blood going to pulmonary vein

Alveolus

Capillaries

Deoxygenated blood from pulmonary artery

Respiratory bronchiole

Oxenated blood to pulmonary vein

Alveolus

**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).

**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 PaO\(_2\)) or hypercapnic (inadequate ventilation i.e. high 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.

![Figure 24.5 Load, capacity and drive](source: Boore et al. (2016). Illustrated by Shaun Mercier, © SAGE Publications.)

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 breathe 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 chest? 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).
- **‘Rattly’ 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 SpO₂ as the 5th vital sign. However, it is important that those monitoring SpO₂ are aware of the limitations and factors that can affect accuracy: SpO₂ monitoring does not provide data on the patient’s pH, PCO₂, 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 SpO₂ reading may appear higher than it really is, and therefore is falsely reassuring. This is because 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**

![Image of normal and asthmatic airway](source)

**Figure 24.6** Pathology of asthma

*Source: 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 mucus 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).
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, beclometasone
- 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.
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>
<thead>
<tr>
<th>Table 24.2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serial domiciliary peak flow measurements</td>
</tr>
<tr>
<td>Alpha-1 antitrypsin</td>
</tr>
<tr>
<td>Transfer factor for carbon monoxide (Tl CO)</td>
</tr>
<tr>
<td>CT scan of the thorax</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>ECG</td>
</tr>
<tr>
<td>Echocardiogram</td>
</tr>
</tbody>
</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. If you are using the interactive ebook, simply click on the book icon in the margin to go straight to the resource.

Books

Journal Articles

Go to https://study.sagepub.com/essentialadultnursing for further free online journal articles 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.

  Discussion of a study to ascertain aspects of care that could be improved for patients receiving non-invasive ventilation.
  Exploration of the adequate assessment, support and treatment required for severe asthma both in primary care and specialist centres.
  Discussion of a framework for a structured, systematic and detailed assessment in COPD.

Weblinks

Go to https://study.sagepub.com/essentialadultnursing for further 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.

- 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.

ACE YOUR ASSESSMENT

Revise what you have learned by visiting https://study.sagepub.com/essentialadultnursing

- Test yourself with multiple-choice and short-answer questions
- Do the chapter activities in the book and check your answers online

REFERENCES

  Philadelphia: Lippincott Williams and Wilkins.


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


