Respiratory assessment in adults

Date of acceptance: April 19 2007.

Summary
Respiratory disorders are among the most common reasons for admission to critical care units in the UK. However, anecdotal evidence suggests that nursing assessment of patients’ respiratory function is not performed well because it is not considered a priority and the implications of respiratory dysfunction are underestimated. It is essential that nurses are able to recognise and assess symptoms of respiratory dysfunction to provide early, effective and appropriate interventions, thus improving patient outcomes. This article highlights the role of the nurse in respiratory assessment and discusses the implications of clinical findings.

Author
Tina Moore is senior lecturer, School of Health and Social Sciences, Middlesex University, Middlesex. Email: t.moore@mdx.ac.uk

Keywords
Patient assessment; Respiratory system and disorders

These keywords are based on the subject headings from the British Nursing Index. This article has been subject to double-blind review. For author and research article guidelines visit the Nursing Standard home page at www.nursing-standard.co.uk. For related articles visit our online archive and search using the keywords.

Aim and intended learning outcomes
The aim of this article is to enable readers to understand respiratory physiology and assessment, focusing on the signs and symptoms of respiratory dysfunction and appropriate interventions.

After reading this article you should be able to:

- Understand respiratory physiology,
- Assess respiratory status appropriately and correctly,
- Identify signs of hypoxaemia and hypercapnia,
- Demonstrate knowledge and understanding of type I and II respiratory failure.

- Initiate appropriate nursing interventions for a patient experiencing respiratory difficulties.

Introduction
Deterioration of respiratory function is one of the major causes of critical illness in the UK (Department of Health 2000). The primary purpose of respiratory assessment is to determine the adequacy of gas exchange, that is, oxygenation of the tissues and excretion of carbon dioxide. By undertaking a full and systematic assessment of the patient’s respiratory status, nursing staff are in a prime position to act on findings and ensure that appropriate medical and/or nursing interventions are initiated. A glossary is provided in Box 1.

Respiratory physiology
The main function of the respiratory system is to provide life-sustaining oxygen to all cells in the body and to remove carbon dioxide, a byproduct of cellular metabolism. The respiratory system consists of the upper airway, including the nasal passages, sinuses, pharynx and larynx, and the lower airway includes the trachea, bronchi, lung, bronchioles and alveoli.

The control of ventilation occurs through voluntary and involuntary mechanisms. Voluntary control of the muscles of respiration is regulated through the central nervous system (CNS). The CNS enables individuals to maintain conscious control over their breathing rate. Involuntary ventilation is dependent on the respiratory centre,
comprising the medulla oblongata and pons. The respiratory centre transmits impulses to the respiratory muscles, causing them to contract and relax. Normally, carbon dioxide levels influence the respiratory centre. When PaCO₂ levels, that is, partial pressure of carbon dioxide in arterial blood, in the blood rise, the respiratory centre is stimulated to increase the rate and depth of breathing, resulting in increased excretion of carbon dioxide. Low PaCO₂ levels in the blood eventually inhibit stimulation of the respiratory centre. This results in an initial increase in the respiratory rate, which then becomes slow and shallow to retain carbon dioxide in an attempt to achieve homeostasis.

Patients with chronic obstructive pulmonary disease (COPD) have experienced long-standing lung damage resulting in an alteration in gas exchange. Here, the central chemoreceptors become tolerant of high levels of carbon dioxide, resulting in reliance on hypoxia to stimulate the respiratory (hypoxic) drive. If patients with COPD are given too much oxygen the hypoxic drive will be lost causing respiratory failure and possibly respiratory arrest. The transfer of oxygen from the atmosphere to the tissues is a four-stage process. **Diffusion of oxygen into the alveoli** Diffusion of oxygen is dependent on a normal airway diameter, adequate respiratory rate and depth and a functioning nervous supply. Airway passages can narrow in the presence of sputum, vomit, trauma, pulmonary oedema and irritants such as smoke. Chemoreceptors located in the circulatory system and brain stem sense the effectiveness of ventilation by monitoring the pH status of the cerebrospinal fluid, PaO₂, that is, partial pressure of oxygen in arterial blood and PaCO₂. Chemoreceptors respond to hypercapnia, acidosis and hypoxia by sending impulses to the medulla oblongata to alter the rate of ventilation. There are two main types of chemoreceptor:

- Central chemoreceptors located in the medulla oblongata.
- Peripheral chemoreceptors located in the carotid and aortic bodies, which are more sensitive to decreases in oxygen levels in the blood.

Stretch receptors are located in the bronchial smooth muscle. They are stimulated by lung hyperinflation. Impulses are sent to the respiratory centre to limit further inflation, avoiding over distension of the lung, and to increase expiratory time. When the patient hypoventilates he or she should be encouraged to take deep breaths because a small increase in lung size may stimulate the stretch receptors to cause further inspiration, thus increasing lung expansion.

**Transfer of oxygen across the alveolar capillary membrane** Gas exchange in the lungs occurs across the alveolar capillary membrane, which has a vast surface area, a thin membrane and a constant supply of both air and blood. These are ideal conditions for oxygen diffusion and transfer. Gases move from an area of high pressure in the alveoli, to an area of low pressure in the capillaries, until equilibrium is achieved. Surfactant is secreted by the alveolar cells and maintains its integrity by covering the inner surface of the alveoli and lowering alveolar surface tension at the end of expiration, thus preventing atelectasis and enabling greater transfer of oxygen. The rate at which oxygen diffuses across the alveolar capillary membrane is dependent on conditions in the alveoli, partial pressure of oxygen molecules and the adequacy of pulmonary circulation.

**Transport of oxygen via haemoglobin** Oxygen is transported within the circulation in two interrelated ways. Approximately 3% of oxygen is dissolved in plasma and the remaining 97% is transported by binding with haemoglobin. As oxygen diffuses across the alveolar capillary membrane, it dissolves in the plasma where it exerts pressure. As the partial pressure of oxygen increases in the plasma, oxygen moves into the erythrocytes and binds with haemoglobin until saturated. Measurement of haemoglobin concentration is important when assessing individuals with respiratory dysfunction. This is because a decrease in haemoglobin concentration below the normal value of blood reduces oxygen content. Increases in haemoglobin concentration may increase oxygen content, minimising the effect of impaired gas exchange. An adequate plasma level of PaO₂ is essential for the remaining oxygen to bind with haemoglobin to aid tissue perfusion; respiratory dysfunction impairs this process.

**Movement of oxygen from the haemoglobin to the tissues** Oxygen enters the tissues by diffusing down the concentration gradient from high concentrations in the alveoli to lower concentrations in the capillaries. This process is influenced by haemoglobin level, oedema, fibroses and destruction of the alveoli (Pierce 2007). Inadequate alveolar ventilation may cause a decrease in the normal pH level. PaCO₂ is increased, CO₂ diffuses across the blood-brain barrier until PaCO₂ in the blood and cerebrospinal

---

** BOX 1 **

** Glossary **

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acidaemia</td>
<td>state in which the pH of the blood falls below 7.35 (normal = 7.35-7.45).</td>
</tr>
<tr>
<td>Atelectasis</td>
<td>collapse of lung tissue with consequent reduction in gas exchange.</td>
</tr>
<tr>
<td>Hypercapnia</td>
<td>increased amount of carbon dioxide in arterial blood.</td>
</tr>
<tr>
<td>Hypoxaemia</td>
<td>insufficient oxygen content in arterial blood.</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>diminished amount of oxygen in the tissues.</td>
</tr>
<tr>
<td>Orthopnoea</td>
<td>difficulty in breathing unless in an upright position.</td>
</tr>
<tr>
<td>Platypnoea</td>
<td>shortness of breath when sitting upright.</td>
</tr>
</tbody>
</table>

---
learning zone respiratory focus

fluid (CSF) reach equilibrium. As the central chemoreceptors sense the resulting decrease in pH they stimulate the respiratory centre to increase the depth and rate of ventilation. Increased ventilation causes PaCO₂ of arterial blood to decrease below that of the CSF. As a result PaCO₂ diffuses out of the CSF, returning its pH to normal (Huether and McCance 2006). In patients with COPD these receptors become insensitive to small changes in PaCO₂ and as a result regulate ventilation poorly.

Time out 2

1. After you have assessed a patient’s respiratory status, write down what data you have collected.
2. On your next shift count how many patients have been diagnosed as having an identified respiratory problem or classified as being acutely or critically ill. How many of these patients have had a comprehensive respiratory assessment?
3. Describe the type of data you have observed during respiratory assessment of such patients.

Hypoxaemia

The respiratory assessment may indicate that the patient has hypoxaemia. Hypoxaemia is the reduced oxygenation of arterial blood cells. Air and blood both arrive at the alveoli, the aim is that all the circulatory blood volume should be available for gas exchange. Adequate gas exchange requires ventilation and perfusion of blood flow to be matched. The relationship between ventilation and perfusion in the lungs is measured by calculating the difference between the alveolar and arterial partial pressure of oxygen (Huether and McCance 2006). At rest alveolar ventilation equals 4L/minute and perfusion equals 5L/minute. The ventilation to perfusion ratio is 4:5 = 0.8. In a ‘perfect lung’ gas exchange will be evenly distributed or perfectly matched. In other words, all alveoli receive an equal share of alveolar ventilation and the pulmonary capillaries receive an equal share of cardiac output. Abnormal ventilation to perfusion ratios are the most common cause of hypoxaemia (Huether and McCance 2006). These can be caused by either inadequate ventilation of well-perfused areas of the alveoli or good ventilation with poor perfusion, as occurs in pulmonary embolism.

Causes of hypoxaemia include:

- Reduced oxygen content of inspired gas, most commonly associated with a drop in atmospheric pressure, for example, high altitudes.
- Hypoventilation of the alveoli resulting in hypercapnia, which can occur in unconscious patients or those with COPD. A reduced amount of oxygen enters the alveoli, for example, when a patient takes a shallow breath.
- Thickened alveolar capillary membrane or decreased surface area for diffusion resulting in impaired diffusion of oxygen.
- Low cardiac output or complete vessel occlusion.
- Histotoxic or cytotoxic: histotoxic relates to substances that cause tissue poisoning and cytotoxins are substances that are toxic and hazardous to the cells.
- Atelectasis resulting in partial or complete collapse of the alveoli.

Nursing staff may use arterial blood gas (ABG) measurement to assess for hypoxaemia. This involves obtaining a sample of arterial blood either through the ‘stab’ method, usually from the radial or femoral artery, or through an established indwelling arterial catheter. The latter should be used for frequent sampling. The use of arterial catheters is not recommended in general ward settings because of the complications of disconnection and accidental intra-arterial injection, which can be life threatening.

Respiratory assessment

The purpose of respiratory assessment is to determine the adequacy of gas exchange, that is, oxygenation of the tissues and excretion of carbon dioxide. Wherever possible the same nurse should be involved in the assessment and/or monitoring of the patient’s respiratory status for the duration of the shift. This should enable consistency of assessment and the identification of subtle as well as overt changes in respiratory function. Depending on the severity of respiratory impairment, history taking may be limited and observational skills may need to be used (Moore 2004).

Factors that may influence the patient’s respiratory function include:

- Pregnancy – fluid retention is caused by increasing oestrogen levels resulting in oedema. Progesterone levels rise six-fold during pregnancy (Lumb 2005) and have a significant effect on the control of respiratory function and ABGs. Enlargement of the uterus in the third trimester may cause the diaphragm to become misplaced, affecting lung expansion.
- Obesity – the poor positioning of obese patients in bed may impede lung expansion.
- Circulatory problems – pulmonary oedema and anaemia may impede gas exchange.
- Environmental influences – such as exposure to the cold, may cause shivering, thus the nurse...
will not be able to conduct the assessment properly, distorting findings.

- Trauma – particularly of the chest. A patient with chest pain will be unwilling to take deep breaths. If he or she has fractured ribs the lung may be deflated and cause hypoinflation of the alveoli.
- Known allergies – may cause anaphylaxis, which could cause swelling of the upper airways and subsequent difficulty in breathing.
- Pathophysiological problems – in particular, those which can cause abdominal distension, for example, bowel obstruction and ascites. The lungs are unable to inflate fully as a result of distortion of the diaphragm.

When conducting respiratory assessment, the patient should be positioned upright, if possible. This position not only makes lung expansion easier, but also enables access to the anterior and posterior thorax. Alternative positions may distort findings and should be acknowledged, if unavoidable, when interpreting data. If appropriate, the patient’s clothing should be removed because this may act as a barrier to visible and auscultation assessment, again distorting findings. Some patients may be aware that their respiratory function is being assessed and this may lead to a subconscious response that influences their breathing rate. Closed questions should be used to minimise any distress in the acutely breathless patient. Generally, respiratory assessment can be broken down into four areas: inspection, palpation, percussion and auscultation. Nurses do not perform percussion as a mode of respiratory assessment unless additional training has been undertaken. Nurses should identify and determine the meaning of different sounds over different parts of the thorax. This is an advanced and complex skill.

**Time out 3**

With reference to Time out 2, reflect on the following:

1. How many of these patients have their respiratory status recorded regularly on an observation chart?
2. List any other respiratory observations made by nursing staff.

**Inspection** Inspection involves a direct, critical, purposeful observation, which includes vision, hearing and smell. The purpose of inspection is to observe for normal patient data and deviations, paying attention to obvious and subtle changes which will require further investigation.

**Rate** The ratio of respiration to pulse rate in the healthy adult is 1:4 (Moore 2004). The respiratory rate should be counted for one full minute and categorised into one of the following:

- Eupnoea or ‘normal’ rate. Opinion as to what the normal rate should be varies, but parameters are between 10-17 breaths per minute.
- Tachypnoea, greater than 18 breaths per minute, is usually the first indication of respiratory distress. Possible causes include anxiety, pain, left ventricular failure and circulatory problems such as anaemia.
- Bradypnoea, less than 10 breaths per minute, may be an indication of increased intracranial pressure, depression of the respiratory centre, narcotic overdose and severe deterioration in the patient’s condition.
- Hypopnoea or abnormally shallow respirations may vary with age. Shallow breathing is considered part of the normal ageing process.

**Rhythm** The normal respiratory rhythm has regular cycles, with the expiratory phase slightly longer than the inspiratory phase. A short pause is normal between expiration and the next inspiration. Chest movement should be equal, bilateral and symmetrical (Ahern and Philpot 2002). Generally, respiratory rhythm varies between men and women. In men, the respiratory rhythm appears to originate from the abdomen or diaphragm whereas women have a tendency to breathe via their thorax or costal muscle. Patients who are sleeping are also inclined to use their abdominal muscles when breathing. There is an assumption that the use of abdominal muscles relates to an increase in respiratory effort (Moore 2004). It is important that nurses are aware of the different circumstances in which patients appear to use their abdominal muscles because this will prevent incorrect diagnosis. Altered rhythms may indicate underlying disorders, for example, Kussmaul respirations or rapid deep breathing resulting from the stimulation of the respiratory centre in the brain is caused by metabolic acidosis and occurs in diabetic ketoacidosis. Cheyne-Stokes respirations, periods of apnoea alternating with periods of hypoxia, may indicate left ventricular failure or cerebral injury and are sometimes present at the end stage of life.

**Quality of breathing** Normally, there is symmetry in chest movement. Failure of the chest wall to rise adequately may indicate fibrosis, collapse of upper lobes or bronchial obstruction. It may also indicate severe pleural thickening, which may cause flattening of the anterior chest wall and diminished respiratory effort. Sudden, sharp chest pain, for example, caused by pneumothorax, can inhibit the patient from taking deep breaths, resulting in hypoventilation of the alveoli.

**Degree of effort** The use of accessory muscles such as the sternocleidomastoid muscle, which passes obliquely across the side of the neck, the scalenus muscles at the side of the neck and the trapezius muscle spanning from the neck, shoulders and...
Vertebral symptoms, may suggest that the patient has difficulty breathing. The patient may also have orthopnoea or even platypnoea. Patients who have difficulty in expiration may have abnormalities of lung recoil and/or airway resistance, such as emphysema, pulmonary oedema or asthma. Increased inspiratory effort can indicate upper airway obstruction, for example, anaphylaxis and epiglottitis. Tracheal deviation may indicate pneumothorax. The influence of the severity of breathlessness on restricted activity such as walking or talking should be noted. Other physical symptoms indicative of difficulty in breathing may include breathing through ‘pursed lips’ on expiration as patients try to force air out of the overstretched alveoli. Nasal flaring can indicate respiratory distress in adults, although this is more common in children.

**Skin colour** Cyanosis, a bluish colour of the skin and mucous membranes, may occur when large amounts of unsaturated haemoglobin are present, and may be detectable when oxygen saturation of arterial blood drops below 85% (Moyle 2002). Cyanosis is usually considered a late sign of respiratory dysfunction, however, this is subject to considerable variability. Cyanosis is often difficult to appreciate in artificial lighting, unless quite defined and is best seen on the lips and under the tongue. There are two types of cyanosis. Peripheral cyanosis, usually indicating poor circulation, is observed in the skin and nail beds and is most noticeable around the lips, ear lobes and fingertips. Central cyanosis, usually indicating circulatory or ventilatory problems, is indicated by a bluish colour of the tongue and lips. Cyanosis can easily be overlooked and requires diligent observation. In the absence of central cyanosis, peripheral cyanosis normally indicates circulatory problems rather than respiratory disease (Casey 2001).

Prolonged hypoxaemia can lead to erythrocytosis and produces a ruddy appearance of the skin. Particular caution needs to be taken when assessing skin colour on patients with dark pigmentation because colour changes, particularly cyanosis, are not easily detectable. It is important to note that anaemic patients may have insufficient haemoglobin to produce the blue colour of the mucous membrane that characterises cyanosis. **Deformities** Clubbing of the finger digits occurs as a result of a chronic condition forming over a long period of time. This may be indicative of hypoxaemia from chronic pulmonary or cardiovascular disease. Deformities of the posterior thorax can affect the quality of breathing. The diameter of the anterior and posterior chest should be compared with the side-to-side diameter. If the anterior and posterior diameter is approximately double the measurement of the side-to-side diameter, this indicates a ‘barrel chest’ caused by emphysema. Spinal deformities such as kyphosis also influence lung expansion.

**Mental status** A reduction in the patient’s level of consciousness and/or altered mental status may indicate hypoxaemia. Symptoms may include inappropriate behaviour, drowsiness and confusion. Any change in mental status should be reported immediately because this may signal that the brain is being deprived of oxygen. If appropriate and immediate action is not taken the patient could deteriorate into unconsciousness, which may result in irreversible brain damage. Assessment of the patient’s mental status should be conducted with care because he or she may demonstrate fear and anxiety, but may not be hypoxic. Language barriers and cultural approaches to disorders should also be considered during the assessment process because some patients may not understand certain instructions or questions.

**Cough** Assessment of the patient’s cough is important because it can indicate if a patient has difficulties in clearing the lungs of sputum or fluid. The assessment of a patient’s cough should include a number of important observations (Box 2). Sputum is a useful indicator of lung pathology (Box 3).

**Palpation** Palpation is used to assess bilateral movements of the chest and diaphragm. It is also used to assess surgical emphysema. The palm of the hand, which should be warm, is placed on an area of the patient’s chest where vibrations are felt. **Auscultation** Assessment of breath sounds, with or without a stethoscope, should form part of nursing assessment. Knowledge of the different types of breath sounds aids description and diagnosis. Without a stethoscope, normal breathing should be quiet. Normal breath sounds are categorised as vesicular, bronchovesicular and bronchial:

- Normally, vesicular sounds, which are low pitched, low intensity and often described as ‘soft and breezy’, can be heard over most of the lung fields.
- Bronchovesicular sounds should be heard in the anterior region, near the main stem bronchi and posterior chest wall only between the scapulae. Bronchovesicular sounds are usually more moderate in pitch and intensity.
- Bronchial sounds are high pitched, loud and hollow. These sounds are usually heard over the larger airways and the trachea. If bronchial sounds are heard in other areas this could indicate consolidation of lung tissue, for example, in pneumonia.

Abnormal breath sounds, known as adventitious sounds, including crackles, as heard in pulmonary
Events that may interfere with the reading include:

- Oxygen saturation status and detect hypoxaemia.
- Patient’s ventilatory status, but can calculate not provide comprehensive information on the important to remember that pulse oximetry does not calculate blood. Changes can be detected immediately. It is base of the lungs near the end of inspiration and usually represent the opening of the alveoli.

Medium crackles are lower in pitch and are heard during the middle or latter part of inspiration.

Course crackles heard on both inspiration and expiration are usually associated with mucus, which may clear after the patient has coughed.

**Pulse oximetry**

The main function of pulse oximetry is to detect hypoxaemia before obvious symptoms are displayed (Moyle 2002). The pulse oximeter provides continuous, non-invasive monitoring of the oxygen saturation from haemoglobin in arterial blood. A pulse oximeter is a clip-like device that measures the amount of haemoglobin saturation in the tissue capillaries. The device transmits a beam of light through the tissue to a receiver. The wavelengths of the transmitted light are altered by the amount of saturated haemoglobin. Light is translated by the receiver into a percentage of oxygen saturation of the blood. Changes can be detected immediately. It is important to remember that pulse oximetry does not provide comprehensive information on the patient’s ventilatory status, but can calculate oxygen saturation status and detect hypoxaemia.

Events that may interfere with the reading include:

- Nail polish – particularly dark colours, for example, black, dark blue (Wahr and Tremper 1996) and green.
- Poor peripheral perfusion – possibly resulting from hypotension, may lead to poor readings. It may help to rotate or transfer the probe to different sites frequently because peripheral perfusion may be better in different parts of the body. Probes that are applied too tight will cause vasoconstriction and interfere with readings. A dampened waveform could indicate a reduction in arterial flow or a misaligned sensor.

In the case of misalignment, the probe will need to be repositioned. The probe should be checked regularly for tightness and misalignment. If this occurs the tape should be loosened or the position of the probe should be changed.

- Recording blood pressure – the pulse oximetry sensor needs to be placed on a finger of the opposite side of the arm where the blood pressure is being taken because inflation of the cuff will cause the readings to be inaccurate.

- Carbon monoxide poisoning – patients with, or suspected of, carbon monoxide poisoning should not be monitored using pulse oximetry. Carbon monoxide poisoning causes abnormal haemoglobins in the case of carboxy-haemoglobin, which can occur in patients with carbon monoxide poisoning resulting from smoke inhalation. The pulse oximetry sensor cannot differentiate between oxyhaemoglobin and carboxyhaemoglobin (Moyle 2002), and will therefore provide a falsely evaluated oxygen saturation reading. It is considered dangerous practice to rely on pulse oximeter readings in this situation. Instead, ABG analysis should be undertaken (Moore 2004).

- Movement – sudden movements and restlessness may cause the pulse oximetry sensor to partially dislodge, or cause motion artefact (distortion of the wave form caused by

**BOX 2**

**Criteria for assessing a cough**

- Regularity.
- Length of time taken to cough.
- Presence or absence of pain.
- Distinctive sounds, for example, whoop or bark.
- Strength of cough.
- Secretions.

**BOX 3**

**Types of sputum**

- Frothy white, sometimes blood-stained sputum, indicates pulmonary oedema.
- Bloody sputum (frank blood – haemoptysis) could be indicative of a pulmonary embolism.
- Blood-stained sputum (streaks of blood) may indicate pneumonia, lung abscess or aspiration of stomach contents.
- Green and purulent sputum often seen in lung infection or pneumonia.
- Yellow/green sputum and copious in amount may denote advanced chronic bronchitis.
- Black (tar) sputum is seen in smokers.
- Old blood may be a sign of tuberculosis or lung cancer.
learning zone respiratory focus

movement). This affects the ability of light to travel from the light-emitting diode to the photo detector in the pulse oximeter probe. It may also be difficult to determine the pulse in patients who have rhythmic movement, for example, seizures and shivering. The importance of keeping still should be explained to the patient. If the patient is unable to limit his or her movement, nursing staff should consider moving the probe to the ear lobe because movement here least affects the equipment. However, it is important to consult the pulse oximetry manufacturer’s guidance for alternative sites. To minimise potential problems, it may be useful for nursing staff to test the equipment on themselves before placing it on the patient. The pulse reading should be correlated with the patient’s heart rate. Variation between pulse and heart rate may indicate that not all pulsations are being detected. In this case a replacement monitor may be required.

Respiratory failure

Respiratory failure is a syndrome in which the respiratory system fails in one or both of its gas exchange functions: oxygenation and/or carbon dioxide elimination (Sharma 2006). The condition can be acute or chronic. Chronic respiratory failure develops over several days or longer, allowing time for metabolic compensation and an increase in bicarbonate concentration. Therefore, the pH of arterial blood usually only decreases slightly. Acute respiratory failure is characterised by life-threatening derangements in ABGs and acid-base status. The manifestations of chronic respiratory failure are less dramatic and may not be as readily apparent. Blood gas disturbances occur as a result of ventilation to perfusion inequality, inadequate alveolar ventilation or a combination of both. Unventilated alveoli result in vasoconstriction, production of surfactant via the alveolar cells and a bounding pulse (the effect of carbon dioxide blood levels are affected (Box 4). As the alveoli are microscopic and prone to collapse, the secretion of surfactant via the alveolar cells facilitates its expansion during inspiration. However, surfactant production is inhibited by hypoxia, acidosis, poor perfusion, smoking and dry gas, for example, unhumidified oxygen.

Type I respiratory failure: oxygenation

Oxygenation respiratory failure occurs in the presence of hypoxia without hypercapnia (Box 4). It is typically caused by a reduction in inspired oxygen, a ventilation to perfusion mismatch and alveolar hypoventilation (Priestley and Huh 2006). Most pulmonary and cardiac conditions can result in respiratory failure with inadequate oxygenation, pulmonary oedema and COPD being the more common causes. As a result of alveolar hypoventilation the PaCO₂ rises resulting in a fall in PaO₂. Respiratory failure that develops slowly allows renal compensation with retention of bicarbonate, often resulting in near normal pH levels. A change in the pH of the blood, together with an increase in carbon dioxide, affects the saturation of haemoglobin (Moyle 2002). This is the most common form of respiratory failure, and can be associated with virtually all acute diseases of the lung, which generally involve fluid filling or collapse of the alveoli. Examples of type I respiratory failure include cardiogenic, non-cardiogenic, pulmonary oedema, pneumonia and pulmonary haemorrhage (Sharma 2006).

The distinction between acute and chronic hypoxic respiratory failure cannot readily be made on the basis of ABGs.

Clinical features Type I respiratory failure may have a variety of clinical manifestations (Box 5). However, these are non-specific and respiratory failure may be present in the absence of dramatic signs or symptoms. This emphasises the importance of ABG measurements in all patients who are acutely or critically ill in those where respiratory failure is suspected.

Pulmonary arteries respond to hypoxia by vasoconstriction, producing vascular resistance and pulmonary hypertension. Right ventricular enlargement or right-sided heart failure develops later. Nursing care should be directed at preventing the patient from developing late clinical features, through early identification of increased respiratory rate, reduced oxygen saturation and neurological changes.

Type II respiratory failure: ventilation Type II respiratory failure can be caused by increased airway resistance and reduced lung compliance, as indicated in severe asthma and pulmonary oedema (Sharma 2006). Both oxygen and carbon dioxide blood levels are affected (Box 4). As the alveoli are microscopic and prone to collapse, the secretion of surfactant via the alveolar cells facilitates its expansion during inspiration.
Management

Identifying the type of respiratory failure is important as it determines the intervention. Underlying causes of respiratory failure, such as chest infections or trauma, should always be treated. The aim of managing respiratory failure is to enable adequate oxygen delivery to the tissues with an adequate PaO₂. This can be achieved through supplementary oxygen via nasal cannula or a face mask. In the case of severe hypoxaemia, intubation and mechanical ventilation may be warranted. Generally, type I respiratory failure may require supplementary oxygen. However, some local policies advocate non-invasive ventilation therapy. Type II respiratory failure requires additional intervention, for example, bi-level non-invasive ventilation, continuous positive airway pressure or full ventilation. Treating hypoxaemia will not improve the PaCO₂ and may make it worse (Lumb 2005). It is therefore essential to ensure that palliative relief of hypoxia does not result in hypercapnia, and arterial PaCO₂ should be monitored closely. Hypercapnia unaccompanied by hypoxaemia is well tolerated and is not likely to threaten organ function unless accompanied by severe acidosis (Sharma 2006). Many experts believe that hypercapnia should be tolerated until the arterial blood pH falls below 7.2 (Sharma 2006). Appropriate management of the underlying disease is an important component in the management of patients with respiratory failure.

Oxygen therapy

The need for oxygen therapy should be assessed in patients with cyanosis, oxygen saturations less than or equal to 92% without additional oxygen support and all patients with severe air flow obstruction (National Institute for Clinical Excellence 2004). With the exception of resuscitation, oxygen should always be prescribed by a doctor, with clear guidance regarding the flow rate, delivery system, duration and monitoring of treatment. When oxygen is being administered, the patient should be positioned upright if possible, to maximise lung expansion. If using nasal cannula, the flow rate of oxygen must not exceed four litres per minute, to prevent discomfort and damage to the nasal mucosa. A full respiratory assessment should be undertaken and the patient should be closely monitored throughout treatment.

Masks

Fixed performance masks provide a steady concentration of inspired oxygen. Such masks should always be used in patients who have COPD unless the patient’s PaCO₂ is known to be normal. The flow of oxygen delivered by variable performance masks varies with changes in the breathing pattern. Masks can be used when there is no danger of carbon dioxide retention. If the patient is severely hypoxic a non-rebreathing mask with a reservoir bag attached can be used. A reservoir bag fills up with oxygen during the patient’s inspiratory phase and this oxygen is breathed in during inspiration. The use of a reservoir bag enables the delivery of high concentrations of oxygen to the patient.

Nasal cannulae

Patients who are expectorating copious amounts of sputum, as in the case of gross pulmonary oedema, may be required to receive oxygen via nasal cannula. Nasal cannulae are simple, unobtrusive and allow eating, talking, breathing and other activities.

BOX 4

Respiratory failure

Type I respiratory failure is defined as:

- PaO₂ <8KPa
- PaCO₂ <6KPa

Type II respiratory failure is defined as:

- PaO₂ <8KPa
- PaCO₂ >6KPa

PaO₂ = partial pressure of oxygen in arterial blood.
PaCO₂ = partial pressure of carbon dioxide in arterial blood.
KPa = kilopascals (a type of unit used to measure pressure).

(Box British Thoracic Society Standards of Care Committee 2002)

BOX 5

Clinical features of type I respiratory failure

Early clinical signs include:

- Irritability, altered level of consciousness, confusion.
- Restlessness, anxiety, fatigue.
- Cool and dry skin.
- Increased cardiac output, tachycardia and headache as a result of stimulations of ventilation via the carotid chemoreceptors.

Intermediate clinical signs include:

- Confusion.
- Aggression.
- Lethargy.
- Tachypnoea.
- Dyspnoea can cause an uncomfortable sensation of breathing.
- Hypotension.
- Tachycardia, bradycardia and a variety of arrhythmias may result from hypoxaemia and acidosis.

Late clinical signs include:

- Cyanosis.
- Oxygen saturations of less than 75%.
- Diaphoresis or sweating.
- Coma and convulsions.
- Cardiac arrhythmias.
- Respiratory arrest.
learning zone respiratory focus

and washing to continue relatively unimpeded. As an approximate guide 21 minutes produces an inspired oxygen concentration of 25-30% (Sharma 2006). Nasal cannulae can cause drying and nasal crusting, which in turn can result in obstruction, therefore, maintaining nasal hygiene is important.

Conclusion

A comprehensive assessment of respiratory status should be performed on all patients who have an identified respiratory disorder and those who are classified as acutely or critically ill. Respiratory assessment should be performed by a competent nurse and used to identify potential respiratory problems. Early intervention is essential to improve the prognosis of patients

References


Respiratory assessment

TEST YOUR KNOWLEDGE AND WIN A £50 BOOK TOKEN

HOW TO USE THIS ASSESSMENT

This self-assessment questionnaire (SAQ) will help you to test your knowledge. Each week you will find ten multiple-choice questions which are broadly linked to the learning zone article.

Note: There is only one correct answer for each question.

Ways to use this assessment

- You could test your subject knowledge by attempting the questions before reading the article, and then go back over them to see if you would answer any differently.
- You might like to read the article to update yourself before attempting the questions.

The answers will be published in Nursing Standard two weeks after the article appears.

Prize draw

Each week there is a draw for correct entries. Send your answers on a postcard to: Nursing Standard, The Heights, 59-65 Lowlands Road, Harrow, Middlesex HA1 3AW, or via email to: zena.latcham@rcnpublishing.co.uk

Ensure you include your name and address and the SAQ number. This is SAQ No. 405.

Entries must be received by 10am on Tuesday August 28, 2007.

1. Hypoxaemia is:
   a) Diminished amount of oxygen in the tissues
   b) Difficulty in breathing
   c) Insufficient oxygen content in arterial blood
   d) Diminished carbon dioxide in arterial blood

2. The main function of the respiratory system is to:
   a) Provide oxygen to the cells in the body
   b) Deliver carbon dioxide to the tissues
   c) Remove oxygen from the cells
   d) Increase cellular metabolism

3. Central cyanosis is indicated by a bluish colour in the patient’s:
   a) Ear lobes and finger tips
   b) Tongue and lips
   c) Extremities
   d) Nasal skin and nail beds

4. Peripheral chemoreceptors are located in the:
   a) Pons varolii
   b) Medulla oblongata
   c) Carotid and aortic bodies
   d) Alveolar capillary membrane

5. Which of the following terms is used to describe abnormally shallow respirations:
   a) Eupnoea
   b) Platypnoea
   c) Hypopnoea
   d) Orthopnoea

6. The rate at which oxygen diffuses across the alveolar capillary membrane is dependent on:
   a) Adequacy of pulmonary circulation
   b) Partial pressure of oxygen molecules
   c) Conditions within the alveoli
   d) All of the above

7. An assessment of the patient’s cough should include:
   a) Nature of the secretions
   b) Presence or absence of pain
   c) Length of time taken to cough
   d) All of the above

8. When administering oxygen using nasal cannula the flow rate should not exceed:
   a) 2 litres per minute
   b) 4 litres per minute
   c) 6 litres per minute
   d) 8 litres per minute

9. Tracheal deviation may indicate that the patient has:
   a) Asthma
   b) A pneumothorax
   c) Anaphylaxis
   d) Emphysema

10. Approximately how many millilitres of oxygen is transported to the cells every minute?
    a) 250
    b) 500
    c) 750
    d) 1,000

This self-assessment questionnaire was compiled by Gwen Clarke

Answers

Answers to SAQ no. 403
1. b  2. b  3. b  4. c  5. a
6. d  7. c  8. d  9. c  10. b

Report back

This activity has taken me ____ hours to complete.

Other comments:

Now that I have read this article and completed this assessment, I think my knowledge is:

Excellent
Good
Satisfactory
Unsatisfactory
Poor

As a result of this I intend to:

58  august 15 :: vol 21 no 49 :: 2007