

Administration of oxygen therapy

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Summary

This article aims to increase nurses' knowledge of the safe administration of oxygen therapy in acute care. The administration and potential complications associated with delivery of oxygen to patients with chronic obstructive pulmonary disease (COPD) and type II respiratory failure are discussed.

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OXYGEN, the colourless, odourless gas, is a drug commonly used in a variety of settings to treat or prevent tissue hypoxia – diminished amount of oxygen in the tissues (Jamieson *et al* 2007).

However, despite its wide and frequent use in health care, oxygen is often inaccurately prescribed, resulting in inappropriate administration, monitoring and evaluation of the therapy (Kor and Lim 2000, Wong *et al* 2000, Thomson *et al* 2002, kbar and Campbell 2006).

Indications for oxygen therapy

Respiratory failure is one of the main indications for oxygen therapy. Respiratory failure is the inability to maintain adequate gas exchange and is characterised by abnormal arterial blood concentrations of oxygen and, in certain cases, carbon dioxide (CO_2) (British Thoracic Society (BTS) Standards of Care Committee 2002).

A raised respiratory rate is another indication for oxygen therapy. The accurate monitoring and recording of respiratory rate for acutely ill patients are key markers for the deteriorating patient (National Institute for Health and Clinical Excellence (NICE) 2007).

Documented or suspected hypoxaemia – a diminished amount of oxygen in arterial blood –

is a further indication for oxygen therapy.

Hypoxaemia is characterised by either the arterial oxygen level being below 8kPa (BTS Standards of Care Committee 2002, Resuscitation Council (UK) 2005), the oxygen saturations being less than 90%, or either value being below the desirable range for the clinical situation.

Importantly, the arterial CO_2 level is normal or low. This is also referred to as type I respiratory failure (BTS Standards of Care Committee 2002).

Patients with type II respiratory failure also have an arterial oxygen concentration level below 8kPa, however, the arterial CO_2 concentration is greater than 6kPa (BTS Standards of Care Committee 2002). The hypercapnia (raised CO_2 level in arterial blood) and hypoxaemia occur as a result of decreased alveolar ventilation.

Hypoxia can also result from cardiac respiratory arrest; acute myocardial infarction resulting in a reduced cardiac output; severe trauma, including severe head injury; anaemia with reduced haemoglobin available to transport the oxygen; infection through increased metabolic demand; surgical intervention; and anaesthesia (Kallstrom and American Association for Respiratory Care (AARC) 2002, Pruitt and Jacobs 2003, Higgins 2006).

Oxygen therapy and COPD

Respiratory failure results in an inadequate level of circulating oxygen and sometimes an inadequate removal of CO_2 . It is classified as either type I or type II respiratory failure.

COPD can be associated with type II respiratory failure. However, the use of oxygen therapy for patients with type II respiratory failure with associated COPD remains a confusing and contentious issue (Bateman and Leach 1998, Bennett 2003). To deliver oxygen therapy to patients with COPD safely, Woodrow (2005) stated that it is essential to understand the underlying physiology.

Under normal conditions, CO_2 levels in the blood stimulate the respiratory centre. As the CO_2 levels rise in arterial blood, the CO_2 diffuses across the blood-brain barrier into the cerebrospinal fluid (CSF), until equilibrium is achieved between the blood and the CSF. As the CO_2 diffuses into the

CSF, the pH of the CSF drops, stimulating the chemoreceptors in the central nervous system (CNS). These chemoreceptors stimulate the respiratory centre in the CNS to increase the rate and depth of breathing to excrete the CO₂. Some, but not all, patients with COPD also experience type II respiratory failure (Bateman and Leach 1998). In type II respiratory failure, the sensitivity of the chemoreceptors is lost. Instead of the arterial CO₂ stimulating the chemoreceptors, it is the fall in arterial oxygen that stimulates the respiratory centre.

As a consequence, some authors state that giving high concentrations of oxygen to patients with COPD will reduce the respiratory drive, resulting in respiratory depression (Bennett 2003). However, Bateman and Leach (1998) stated that the number of patients with both COPD and type II respiratory failure is only 10-15%, and that patients with COPD could die as a result of hypoxia if oxygen is withheld because of fear of a raised CO₂ level reducing the respiratory drive.

The Resuscitation Council (UK) (2005) advocates high concentrations of oxygen in such patients when they are acutely ill. NICE (2004) guidelines for the management of adult patients with COPD in primary and secondary care recommend that oxygen is delivered at 40% and titrated upwards if oxygen saturations fall below 90%. NICE (2004) also recommends that if the patient becomes drowsy, or if the oxygen saturations rise above 93-94%, the percentage of oxygen is reduced. This lack of understanding results in a higher proportion of patients with COPD who do not have associated type II respiratory failure being at risk of hypoxia and ultimately death as a result of inadequate oxygen administration (Bateman and Leach 1998).

Patient assessment

Assessment should include careful observation of the patient's chest movement, which should be equal and symmetrical. Both sides of the chest should be moving. The depth and rate of breathing should be recorded. NICE (2007) highlights the respiratory rate as a key parameter, which should be recorded and monitored accurately and regularly. This is because tachypnoea (a respiratory rate greater than 20 breaths per minute) is an early indication of respiratory distress (Jevon and Ewens 2001). Other signs to indicate respiratory distress include the use of accessory muscles, cyanosis, distress and anxiety or abnormal respiratory sounds such as stridor, expiratory wheeze or orthopnoea (Jevon and Ewens 2001).

Arterial blood gas analysis and pulse oximetry recordings can be used to assess the patient's clinical condition (Woodrow 2004). For the

treatment of hypoxia, oxygen at high concentrations is required, although caution is necessary for patients with type II respiratory failure (Woodrow 2005).

Oxygen delivery devices

Oxygen delivery devices fall into two main categories – low flow delivery systems or high flow delivery systems (Bennett 2003).

Low flow delivery systems are also referred to as variable performance systems. These systems deliver oxygen at a low flow rate and provide a variable oxygen concentration to the patient. This occurs because the patient's inspiratory flow rate is greater than the flow rate of oxygen; the patient will draw air in from the atmosphere, which will dilute the oxygen concentration delivered. Low flow oxygen systems include nasal cannula and low flow masks.

Nasal cannula Simple and convenient to use, a nasal cannula is a narrow flexible tubing used to deliver oxygen through the nostrils of a nasally breathing patient. The tubing is fitted over the ears and brought together under the chin by a sliding connector. The maximum flow rate for this device is 4L per minute. The oxygen concentration delivered is variable depending on how much air the patient is mouth breathing. These devices are well tolerated and allow patients to eat, drink and talk while receiving oxygen therapy (MacKenzie 2004). High flow nasal cannula devices are also available, providing a greater concentration of oxygen.

Low flow oxygen masks Simple oxygen masks are plastic devices contoured to fit over the patient's mouth and nose. Simple masks are used mainly for type I respiratory failure. Oxygen is delivered into the mask; air is entrained from the atmosphere through the side ports in the mask. Consequently, the concentration of oxygen delivered depends on the amount of air the patient is drawing in through the mask in relation to the flow of oxygen. Therefore, the oxygen delivery is variable and not accurate (Kallstrom and AARC 2002).

Cooper and Cramp (2003) stated that the flow rate for a simple mask should not be below 5L per minute, because the patient could easily breathe in exhaled air that would not be flushed from the mask at the lower flow rates. However, it is essential to refer to the manufacturer's guidelines because many manufacturers of variable performance masks indicate that both 24% and 28% oxygen are delivered at flow rates below 5L per minute. If the patient is breathing hard and fast, the concentration of oxygen delivered will be lower because the oxygen will be diluted by large volumes of atmospheric air entrained into the mask. If, however, the patient is breathing slowly and deeply, less ambient air is drawn in and the

concentration of oxygen delivered through the mask will be greater.

A non-rebreather mask is similar to a simple face mask, however, it has multiple one-way valves in the side ports and a reservoir bag attached. The one-way valve prevents air from being drawn into the mask, but enables the exhaled CO₂ to leave the mask, therefore preventing the risk of rebreathing. The reservoir bag fills with oxygen thus providing an oxygen reservoir available for the patient to inspire. The one-way valve between the mask and the reservoir bag prevents exhaled air entering the reservoir bag. Being a variable delivery device, oxygen can be delivered at between 10L and 15L per minute and can provide 80-90% oxygen (Pruitt and Jacobs 2003).

High flow delivery systems High flow devices are also known as fixed performance masks. These deliver oxygen rates above the normal inspiratory flow rate. These systems are often referred to as Venturi masks because they work using the Venturi principle. Oxygen is passed through a narrow inlet entraining air from the atmosphere. The concentration of oxygen delivered depends on the flow of oxygen via the inlet and the size of the holes through which the air is entrained. The bigger the hole on the port, the greater the volume of air entrained into the mask and the lower the concentration of oxygen delivered. These masks can deliver between 24% and 60% oxygen, depending on which adapter is used (Bennett 2003). These masks are used for patients who require a high or accurate concentration of oxygen.

Complications

Oxygen is thought to affect lung tissue. It is generally accepted that the administration of oxygen at a concentration greater than 60% for longer than 24 hours can result in decreased lung compliance. Changes to the lung tissue caused by high concentrations of oxygen are referred to as oxygen toxicity (Jevon and Ewens 2001).

High concentrations of oxygen can reduce the production of surfactant, resulting in atelectasis – the collapse of alveoli leading to a reduction in gas exchange. The nurse should monitor oxygen therapy and reduce supplementary oxygen as soon as possible to prevent the risk of this occurring (Woodrow 2005). Patients can become non-concordant with oxygen therapy because of discomfort. Oxygen can easily dehydrate exposed membranes in the upper respiratory tract. Oral fluids will rehydrate the mucosa; if not mouth care is essential. Humidification can be added to the oxygen therapy to warm and moisten the gas (Jevon and Ewens 2001), however, cold water humidification systems are more likely to be used on general wards providing up to 60% oxygen via elephant tubing. Humidification can mobilise secretions and enhance patient comfort (Woodrow 2005).

Conclusion

Oxygen is a life-saving therapy. However, it is essential to recognise that oxygen is a drug and should be prescribed accurately with the required flow rate and delivery device clearly identified. Careful monitoring by the nurse will prevent hypoxaemia, hypercapnia and oxygen toxicity NS

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