

Oxygen therapy in adults

Introduction

Oxygen therapy is the provision of oxygen in greater inspired concentrations (FiO_2) than room air (21%) to prevent tissue hypoxia. Oxygen is used widely in acute and elective situations across a range of specialties, by ambulance personnel, nurses and medical staff. In the emergency or acute setting it is important to appreciate that each professional group may work to different oxygen protocols as their patients are at different stages of the patient pathway. This article outlines the indications and hazards of oxygen therapy and emphasizes the importance of individualized oxygen prescribing, depending on the diagnosis and objective measurement of oxygenation (widely available with pulse oximetry).

Indications

There are a number of different conditions in which oxygen therapy is indicated (Table 1). These include the following.

Hypoxaemia

Oxygen therapy is indicated in all situations where hypoxaemia (arterial partial pressure of oxygen (pO_2) <8 kPa, oxygen saturations $<90\%$) is present and in any acute situation when hypoxia is suspected until this can be assessed objectively. This level corresponds with the shoulder of the oxyhaemoglobin dissociation curve. Below this value the oxygen-carrying capacity of the blood decreases more rapidly as oxygen tension declines. In some conditions, such as acute severe asthma, pulmonary oedema or pulmonary embolism, high FiO_2 is recommended until a response to specific treatment is documented. Even chronic obstructive pulmonary disease (COPD) patients require adequate oxygen and it is recommended to aim for target saturations of 90–92% to ensure sufficient reversal of hypoxia while minimizing the risk of respiratory acidosis occurring secondary to hyperoxia (Murphy et al, 2001) (see below) until more detailed assessment of acid–base balance can be done with blood gases.

Dr Helen Booth is Consultant Thoracic Physician in the Department of Thoracic Medicine, University College London, London WC1E 5DB

Hypoxia without hypoxaemia

Severe trauma

Severe trauma or shock is likely to result in problems with oxygen delivery to tissues from inadequate intravascular volume and/or cardiac output. Maximizing the amount of oxygen attached to haemoglobin and dissolved in the plasma with high FiO_2 may minimize tissue hypoxia in the short term but definitive treatment should be directed to the primary cause.

Other acute medical problems

Supplemental oxygen therapy is widely recommended in guidelines for patients with acute coronary syndromes (Thomson et al, 2002). The evidence for this practice is limited (see below).

High FiO_2 (40–60%) is recommended for patients admitted with pneumothoraces as increasing the nitrogen gradient between the air in the pleural cavity and the pleural blood vessels increases the rate of reabsorption of the pneumothorax.

In carbon monoxide poisoning the formation of carboxyhaemoglobin molecules reduces the oxygen-carrying ability of the blood as well as shifting the oxygen dissociation curve impairing oxygen release at the tissue level. Treatment is with high FiO_2 80–100% via a non-rebreathe mask.

Contraindications and hazards

Although oxygen is life saving it is also potentially toxic. Hyperoxia has been implicated in causing retinopathy of pre-

maturity and chronic lung disease in pre-term infants, probably secondary to production of reactive oxygen species. Similar mechanisms are presumed to account for the toxic effects of oxygen in the setting of rare drug-induced lung diseases, e.g. bleomycin, amiodarone, and paraquat poisoning, when oxygen therapy is relatively contraindicated.

More widely appreciated is respiratory acidosis developing with excessive oxygen therapy in a subset of COPD patients and rarer patients with acute on chronic hypercapnic respiratory failure (e.g. kyphoscoliosis, neuromuscular disorders, obesity-hypoventilation). It is usually taught that the mechanism for this is abolition of hypoxic drive but it is likely to be multifactorial.

It is estimated that about 25% of patients presenting with acute breathlessness to accident and emergency (A&E) departments (6% of all attendances) are secondary to acute exacerbations of COPD. In a 1-year prevalence study 20% of these patients, not requiring immediate intubation, had a respiratory acidosis ($\text{pH} <7.35$), 5% severe acidosis ($\text{pH} <7.25$) with acidosis present in 50% of patients whose pO_2 was >10 kPa, i.e. hyperoxic (Plant et al, 2000). A fifth of acidotic patients reversed their acidosis between A&E and the ward, presumably as a result of standard treatment including controlled oxygen and nebulized bronchodilators. Only about 1% of patients who present with breath-

Table 1. Indications for oxygen treatment in the acute setting

| | Initial target saturations | Suggested device | FiO_2 |
|---------------------------------------|----------------------------|--|-----------------------|
| Hypoxaemia | Non-COPD: $>90\%$ | Simple mask | 40–60% |
| | COPD: 90–92% | Ambulance: simple mask A&E: Venturi | Titrate Sufficient |
| Trauma/shock | 100% | Non-rebreathe/reservoir bag | 80% |
| Pneumothorax | $>90\%$ | Simple mask | 40–60% |
| Acute asthma/pulmonary oedema | $>90\%$ | Simple mask | 40–60% |
| Uncomplicated acute coronary syndrome | $>90\%$ | Nasal cannulae | 2 litres/min |
| CO poisoning | $>90\%$ | Non-rebreathe/reservoir bag | 80% |

A&E = accident and emergency department; CO = carbon monoxide; COPD = chronic obstructive pulmonary disease; FiO_2 = inspired oxygen concentration

lessness have COPD and are at risk of developing respiratory acidosis with uncontrolled oxygen.

The challenge is early identification of patients with COPD as often even the patients do not know their diagnosis. Treatment involves tailoring oxygen therapy in this group to a target range of 90–92%, and prompt assessment of acid–base balance with blood gases (Table 2).

Persistent acidosis despite these measures is an indication for a respiratory stimulant (doxapram), non-invasive ventilation or intubation with mechanical ventilation. Remember that the presence of hypercapnia and respiratory acidosis in other conditions, e.g. asthma or pulmonary oedema, is an indication of severity and is not caused by oxygen therapy. These patients require high FiO₂ and immediate transfer to a high/intensive care facility.

Oxygen is routinely given to patients with acute medical conditions. In experi-

mental models hyperoxia can actually cause reflex vasoconstriction. Indeed higher mortality rates have been reported in randomized studies of normoxic patients receiving supplemental oxygen as part of the initial management of uncomplicated myocardial infarction or stroke (Thomson et al, 2002). Prolonged administration of high FiO₂ >50% can result in absorption atelectasis, pulmonary toxicity, depression of ciliary function and leucocyte function and should be avoided if possible. This emphasizes the importance of using a minimum FiO₂ to obtain the desired target saturations. When the patient is stable they should be assessed regularly regarding the need for continued treatment by daily measurement of saturations on room air at least 10 minutes after coming off oxygen.

Oxygen is a fire hazard and deaths, burns and inhalation injuries have been reported in patients who smoke simultaneous with oxygen therapy. Despite the risks and the

importance of smoking in the pathogenesis of COPD, up to 20% of patients with home oxygen continue to smoke.

Oxygen delivery systems

There are a number of different oxygen delivery systems available, as listed in Table 3. These include both variable and fixed performance devices.

Variable performance devices

These deliver oxygen at flow rates below the patient's inspiratory flow rate so that room air is entrained into the devices which effectively dilutes the oxygen. The FiO₂ therefore can vary significantly dependent on the patient's ventilatory pattern.

Simple oxygen masks/medium concentration masks

These are widely available and cheap. They need to be driven by a minimum of 5 litres/min oxygen to prevent rebreathing of exhaled air which could collect in the mask otherwise. These masks deliver about 30–50% FiO₂ and are a reasonable choice in the pre-hospital setting or non-COPD patients awaiting diagnosis or assessment.

Nasal cannulae

At a flow rate of 2 litres/min the FiO₂ ranged from 23.7–34.9% in stable COPD (Bazauye et al, 1992). Rates over 4 litres/min cause nasal drying and should be avoided. They have the advantage of allowing patients to eat and talk. They are generally used for stable patients with minimal respiratory distress.

Table 2. Reducing the risk of respiratory acidosis in patients with COPD or chronic hypercapnic respiratory failure

| | |
|--|---|
| Ensure that patients with COPD are told their diagnosis and that it is not asthma | |
| In patients with chronic hypercapnia consider warning of the risk of hyperoxia | 'Message in a bottle' Credit-card sized alert card Personal information held in ambulance control |
| Ambulance staff to titrate oxygen to 90–92% saturations in those identified with COPD | |
| In A&E convert to venturi controlled oxygen mask sufficient to give saturations of 90–92% | |
| If unable to maintain adequate oxygenation without increasing respiratory acidosis (pH < 7.35) further management includes the use of a respiratory stimulant (doxapram), non-invasive ventilation or intubation with mechanical ventilation | |
| <small>A&E = accident and emergency department; COPD = chronic obstructive pulmonary disease</small> | |

Table 3. Oxygen delivery devices

| | Performance | Pros | Cons | Main indication |
|--|---|---|--|--|
| Nasal cannulae | 1–6 litres/min 24–40% FiO ₂ | Convenient, relatively unintrusive Cheap | Variable FiO ₂ Dries nose if used > 4 litres/min Not humidified | Stable hypoxaemic patients not requiring high FiO ₂ |
| Simple face mask | 5–10 litres/min 35–50% FiO ₂ | Readily available Cheap | Variable FiO ₂ Rebreathe if < 5 litres/min | Initial mask in most acute situations until assessment Avoid in hypercapnic COPD patients |
| Non-rebreathe/reservoir bag mask | 10–15 litres/min 60–80% FiO ₂ | Highest FiO ₂ approx 80% | Not humidified Avoid prolonged use | Severely hypoxaemic patients or severe trauma |
| Mask with venturi valves (x5) | 2–12 litres/min 24, 28, 35, 40, 60% FiO ₂ | Fixed FiO ₂ | Need to change valve to change FiO ₂ | COPD with hypercapnia |
| Humidified systems with adjustable venturi valves | 5–8 litres/min 35, 40, 60% FiO ₂ | Humidified oxygen | Relatively expensive Cumbersome | Patients on prolonged continuous high FiO ₂ or who have thick retained secretions |
| <small>COPD = chronic obstructive pulmonary disease; FiO₂ = inspired oxygen concentration</small> | | | | |

Non-rebreathe or mask with reservoir bag

These are used in emergency situations to provide high inspired oxygen concentrations. At 10–15 litres/min, sufficient to keep the reservoir bag partially inflated during inspiration, they can provide an approximate FiO_2 of 80%. They cannot be used with humidified systems. They should not be used for more than 24 hours because of the risk of absorption atelectasis and ciliary dysfunction.

Fixed performance devices

Venturi devices

Oxygen is forced through a small orifice which accelerates the gas. Different colour-coded valves entrain a set amount of air to deliver a predetermined FiO_2 ranging from 24–60% at the flow rate specified on the valve.

Humidifier systems

These also run on a venturi system to give relatively fixed FiO_2 at flow rates indicated on the dial. These are relatively expensive but are recommended for patients on prolonged oxygen therapy, particularly those on high FiO_2 and/or those who have retained secretions and would benefit from humidification.

Prescribing oxygen therapy

Oxygen should be prescribed and dispensed accurately, as with any other drug, but numerous studies confirm that this is rarely done (Small et al, 1992). A prescription should include the target saturations, device, and duration of therapy, e.g. continuous or as needed. It is a clinical governance issue as to how this is achieved in hospitals.

Home oxygen

There have been recent major changes in the provision of home oxygen (British Thoracic Society Working Group on Home Oxygen Services, 2006). Hospital doctors can now prescribe home oxygen by sending completed home oxygen form (HOOFF) directly to their regional home oxygen provider rather than going through the GP. It is important, therefore, that hospital doctors are aware of the three different forms and indications of home oxygen provision which now includes ambulatory oxygen.

Long-term oxygen therapy

Definition

Long-term oxygen therapy (LTOT) is the provision of continuous oxygen from an oxygen concentrator usually for ≥ 15 hours a day including night time.

Indications

Patients with a variety of lung or heart diseases, e.g. COPD, interstitial lung disease or chronic heart failure, who are fully assessed, on maximal treatment and have been stable for the preceding 5 weeks or more. They should have chronic hypoxaemia defined as $\text{pO}_2 < 7.3$ kPa, or < 8 kPa with associated secondary polycythaemia or cor pulmonale. Gases should be measured on two separate occasions more than 3 weeks apart. LTOT can be used for palliation of breathlessness in patients who are terminally ill.

Ambulatory oxygen therapy

Definition

Ambulatory oxygen is the provision of oxygen therapy from lightweight cylinders or liquid oxygen during exercise and activities of daily living. Oxygen-conserving devices can be used with cylinders to increase the period of oxygen supply.

Indications

Patients on LTOT and those who desaturate on exercise by $\geq 4\%$ below 90% who demonstrate improvement in exercise tolerance or dyspnoea with sufficient ambulatory oxygen to prevent desaturation. The choice of device depends on how much it will be used.

Short burst oxygen therapy

Definition

This is the use of supplemental oxygen from a cylinder at home for short periods, usually for 10–20 minutes at a time, to relieve breathlessness.

Indications

In patients with severe COPD, interstitial lung disease, heart failure or for palliation where oxygen relieves episodic breathlessness and/or improves exercise tolerance. This is a pragmatic indication as there is no evidence of benefit of short burst oxygen therapy.

Conclusions

This article has emphasized that oxygen therapy, in both the emergency and domiciliary situation, usually requires objective measurement of hypoxaemia and an individualized written prescription of the minimum oxygen concentration required to achieve a target oxygen saturation using an appropriate delivery device. Patients on controlled oxygen with increasing respiratory acidosis require additional support, not a reduction in their FiO_2 . **BJHM**

Conflict of interest: none.

- American Association for Respiratory Care (2002) Clinical Practice Guideline Oxygen therapy for adults in the acute care facility – 2002 revision and update. *Resp Care* **47**: 717–20
- Bazaue EA, Stone TN, Corris PA, Gibson GJ (1992) Variability of inspired oxygen concentration with nasal cannulas. *Thorax* **47**: 609–11
- British Thoracic Society Working Group on Home Oxygen Services (2006) *Clinical Component for the Home Oxygen Service in England and Wales*. British Thoracic Society, London (www.brit-thoracic.org.uk/c2/uploads/clinical%20adult%20oxygenjan06.pdf)
- Murphy R, Mackway-Jones K, Sammy I et al (2001) Emergency oxygen therapy for the breathless patient. Guidelines prepared by the North West Oxygen group. *Emerg Med J* **18**: 421–3
- Plant PK, Owen JL, Elliot MW (2000) One year period prevalence study of respiratory acidosis in acute exacerbations of COPD: implications for the provision of non-invasive ventilation and oxygen administration. *Thorax* **55**: 550–4
- Small D, Duha A, Wiekopf B et al (1992) Uses and misuses of oxygen in hospitalised patients. *Am J Med* **92**: 591–5
- Thomson AJ, Webb DJ, Maxwell, Grant IS (2002) Oxygen therapy in acute medical care. The potential dangers of hyperoxia need to be recognised. *BMJ* **324**: 1406–7

KEY POINTS

- The main indication of oxygen therapy is hypoxaemia.
- Oxygen therapy should be prescribed and delivered accurately.
- Target oxygen saturation for patients with chronic obstructive pulmonary disease (COPD)/chronic hypercapnic respiratory failure (CHRF) should be 90–92% until blood gases can be performed.
- Patients with COPD/CHRF who have saturations 90–92% with a fixed performance oxygen delivery system but a persistent $\text{pH} < 7.35$ despite initial treatment require ventilatory support not a reduction in their inspired oxygen concentration.
- New changes in home oxygen provision were introduced in 2006.