GUIDELINES FOR ACUTE OXYGEN THERAPY FOR WESTERN AUSTRALIAN HOSPITALS

Key Points

1. No patient should be denied oxygen therapy in an emergency.
   - Patients in cardiac and/or respiratory arrest should be managed according to the guidelines for Basic & Advanced Life Support. For any other patient with suspected or known tissue hypoxia, oxygen therapy should be initiated without delay by the attending health care professional (doctor, nurse, midwife or physiotherapist).
   - The indications for acute oxygen therapy and recommendations for initial oxygen doses and target oxygen saturations are tabulated in the supplementary notes and the oxygen prescription form.

2. Patients commenced on acute oxygen therapy should be assessed promptly, carefully and regularly.
   - The patient should be examined by a doctor as soon as possible and, if possible, initial investigations should include an arterial blood gas, haemoglobin level and chest x-ray.
   - Initial and regular observations should include vital signs (pulse rate, blood pressure, respiratory rate), the level of consciousness and pulse oximetry.
   - Specialist medical advice should be sought promptly (e.g. Respiratory, Intensive Care, Anaesthetist, Royal Flying Doctor Service) if the patient cannot be stabilised.

3. Once the patient is stable, oxygen therapy must be prescribed on a dedicated oxygen prescription form by a doctor or, where this is not possible, an appropriately authorized nurse.
   - The prescription should define the indication, target oxygen saturation, oxygen therapy delivery device, range for oxygen flow or percent of inspired oxygen and when oxygen is to be applied. The prescription should be signed and dated and the name of the prescriber printed legibly.
   - An oxygen prescription sticker should be placed in the medication chart to indicate there is an oxygen prescription on a separate form.
   - The prescription should be reviewed daily.

4. In patients receiving oxygen therapy
   - Oxygen saturation should be monitored by pulse oximetry at least as frequently as observations of vital signs and clearly documented on the patient’s observation chart with the inspired oxygen concentration.
   - Oxygen therapy should be titrated to the lowest concentration that meets oxygenation goals. This should be an oxygen saturation of 88-92% for patients with or at risk of hypercapnoeic respiratory failure and 94-98% for all other patients. Special considerations apply for specific poisons (see supplementary notes).
   - Urgent medical review and arterial blood gas measurement is required if the patient develops signs of deterioration including any of the following: acute breathlessness,
decreasing conscious state, oxygen saturations ≤90% and falling, oxygen requirements increasing.

5. **Oxygen therapy should be prescribed cautiously to patients with severe chronic lung disease and other conditions at risk of hypercapnoeic respiratory failure** (e.g. morbid obesity, neuromuscular disorders, chest wall disorder).

- All such patients who require oxygen therapy should be commenced on oxygen using a Venturi mask at 24-28%. Oxygen requirements should be titrated to maintain an oxygen saturation of 88-92%. Arterial blood gases should be measured at baseline.

- In patients with hypercapnoea, non-invasive ventilation should be considered in patients with respiratory acidaemia (pH<7.35) and arterial blood gases should be repeated within 4 hours.

- Once stabilised, it may be possible to replace the Venturi mask with nasal cannulae at low flow (0.5-4 L/min) to maintain an oxygen saturation of 88-92%.

- If nebulised bronchodilators are required, the nebuliser is best driven by compressed air with oxygen therapy given concurrently by nasal cannulae at 2-4 L/min to maintain an oxygen saturation of 88-92%. If compressed air is not available, the nebuliser can be driven by oxygen at flow rate of 6-10 L/min for 3-4 minutes.

- All patients with chronic lung disease and other at-risk conditions who have had an episode of hypercapnoeic respiratory failure should be issued with an oxygen alert card and a 24 and 28% Venturi mask. The card should state that the patient has had an episode of hypercapnoeic respiratory failure, that oxygen therapy should be commenced with a Venturi mask and oxygen saturation maintained between 88 and 92% (see example in supplementary notes). The patient should be instructed to show the card to their local doctor, ambulance crew and emergency department staff in the event of an exacerbation.

6. **Oxygen therapy should be reduced and discontinued in stable patients with satisfactory oxygen saturation**

- The dose of oxygen should be reduced if the patient is clinically stable and the oxygen saturation is above the target range or has been at the upper end of the target range for at least 4 hours. This may require a change to the oxygen delivery device.

- Oxygen therapy should be ceased when the patient is able to maintain oxygen saturation in the target range when breathing room air. Oxygen saturation on room air should be monitored for at least 5 mins after discontinuing oxygen therapy and should be rechecked at 1 hour. Oxygen therapy should be recommenced if the oxygen saturation falls below the target range.

- When a decision has been made to discontinue oxygen therapy, the prescription should be crossed off.
Background
Oxygen therapy is a medical treatment used for tissue hypoxia. It is prescribed to improve oxygen supply and reduce the work of breathing. It has the potential to improve medical outcomes and save lives when used appropriately and to cause harm if used inappropriately.

Indications for oxygen
The main indication for acute oxygen therapy is the presence of tissue hypoxia. This may occur because of:
1. Arterial hypoxaemia (inadequate arterial oxygen content) or
2. Failure of the oxygen-haemoglobin transport system.

Arterial hypoxaemia is defined as an oxygen saturation of less than 90% or an oxygen tension (PaO₂) of less than 60mmHg. It may result from impaired gas exchange in the lung, inadequate alveolar ventilation or a shunt that allows venous blood into the arterial circulation. An arterial blood gas measurement helps to discriminate between these possibilities.

Tissue hypoxia may occur in the absence of arterial hypoxaemia because of a failure of the oxygen-haemoglobin transport system. This can result from a reduced oxygen carrying capacity in blood (e.g. anaemia, carbon monoxide poisoning) or reduced tissue perfusion (e.g. shock). Successful treatment of tissue hypoxia requires early recognition and correction of contributing factors.

Normoxic patients. Most normoxic breathless patients do not benefit from oxygen therapy (O’Neill et al. 2006; Cranston et al. 2008). Oxygen therapy is commonly used in normoxic patients with acute coronary syndromes and stroke although there is no evidence of benefit (Cabello et al. 2010; Moradkhan & Sinoway 2010; Nicholson 2004; Beasley et al. 2007; Ronning & Guldvog 1999) and some potential for harm. Hyperoxia is known to cause coronary vasoconstriction and reduce coronary blood flow (Farquhar et al. 2009; McNulty et al. 2005) and oxygen therapy has been associated with reduced survival in minor and moderate stroke (Ronning & Guldvog 1999). Oxygen therapy is not indicated in normoxic patients with drug overdoses or metabolic acidosis.

Pregnancy. All women with hypoxaemia who are more than 20 weeks pregnant should be managed with left lateral tilt to improve cardiac output and oxygen therapy to maintain an oxygen saturation of 94-98%. There is no role for oxygen therapy during labour for women who are normoxic. The use of oxygen therapy during labour in normoxic mothers has been associated with acidosis of cord blood and this suggests potential harm to the foetus (Fawole 2003). The indications for acute oxygen therapy and recommendations for initial oxygen doses and target oxygen saturations are listed in Table 1 on the next page.

No Longer Applicable
Withdrawn July 2017
Table 1

<table>
<thead>
<tr>
<th>Indication</th>
<th>Examples</th>
<th>Oxygen prescription</th>
<th>Initial Therapy</th>
<th>Target oxygen saturation</th>
</tr>
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<tr>
<td>Critical illness</td>
<td>Major trauma Shock</td>
<td>Reservoir mask 15 L/min</td>
<td>94-98%</td>
<td></td>
</tr>
<tr>
<td>Suspected Type I (hypoxaemic) respiratory failure</td>
<td>Severe asthma Pulmonary embolism Pulmonary oedema Pneumonia Diffuse lung disease</td>
<td>Nasal cannulae 2-4 L/min or Simple face mask 5-10 L/min</td>
<td>94-98%</td>
<td></td>
</tr>
<tr>
<td>Suspected Type II (hypercapnoeic) respiratory failure</td>
<td>Severe COPD Severe bronchiectasis Morbid obesity Sleep apnoea Neuromuscular disease Kyphoscoliosis or other chest wall deformity</td>
<td>Venturi mask 24-28%</td>
<td>88-92%</td>
<td></td>
</tr>
<tr>
<td>Normoxic patients in whom O₂ therapy is commonly used but of uncertain value</td>
<td>Acute coronary syndromes Opioid infusions Advanced malignancy</td>
<td>Nasal cannulae 2-4 L/min or Simple face mask 5-10 L/min</td>
<td>94-98%</td>
<td></td>
</tr>
<tr>
<td>Special cases of poisoning A</td>
<td>Carbon monoxide</td>
<td>Reservoir mask 15 L/min</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td>Special cases of poisoning B</td>
<td>Paraquat Bleomycin lung toxicity</td>
<td>Venturi mask 24-28%</td>
<td>88-92%</td>
<td></td>
</tr>
</tbody>
</table>

### Oxygen transport

The solubility of oxygen in blood is low. As a result, about 98.5% of oxygen in arterial blood is bound to haemoglobin and only about 1.5% in solution. Oxygen therapy increases oxygen transport by increasing the oxygen saturation of haemoglobin and the amount of oxygen carried in solution. The administration of supplemental oxygen to patients with normal or near normal oxygen saturation (94-98%) has very little effect on blood oxygen content.

### Dose of oxygen therapy

Oxygen therapy should be prescribed at a dose to achieve normal or near normal oxygen saturation (94-98%) for all acutely ill patients apart from those at risk of hypercapnoeic respiratory failure and some poisonings. The dose of oxygen therapy depends on the condition being treated. Both inappropriately low or high concentrations have the potential for harm. Exposure to high concentrations of inspired oxygen can cause absorptive atelectasis and injury to airways and lung parenchyma. In patients with chronic lung disease and other conditions associated with hypercapnoeic (Type II) respiratory failure (e.g. morbid obesity, sleep apnoea, neuromuscular diseases, chest wall deformities), high oxygen concentrations may lead to worsening hypercapnoea, reduced consciousness and respiratory arrest. This should be suspected when the
serum bicarbonate is elevated. However, in the acute situation, inadequate oxygen therapy accounts for more deaths and permanent disability than high concentrations of oxygen.

**Monitoring oxygen therapy**
The effect of oxygen therapy can be monitored by pulse oximetry or arterial blood gas measurements, respiratory rate and distress and level of alertness. Pulse oximetry may not be accurate in patients with poor peripheral circulation. This may occur with some medical conditions (e.g. heart failure, hypothermia, diabetes mellitus). Capillary and venous PO$_2$ measurements are not a substitute for arterial PO$_2$. However capillary pH, bicarbonate and PCO$_2$ and venous pH and bicarbonate can correlate closely with arterial measurements when collected correctly (Toftegaard et al. 2008; Zavorsky et al. 2007). A venous PCO$_2$<45 mmHg excludes significant arterial hypercapnoea (PaCO$_2$>50 mmHg) (Kelly et al. 2002). In situations where arterial blood gases are not available, pulse oximetry may be used.

Local anaesthesia should be used for arterial blood gas collection except in an emergency, if the patient is unconscious or anaesthetised or if the patient prefers not to have local anaesthesia.

**Posture and oxygenation**
Oxygenation is reduced in the supine posture so fully conscious hypoxaemic patients should be nursed in the most upright posture possible unless (a) this is uncomfortable for the patient, (b) immobilisation is required for suspected or actual skeletal or spinal trauma, (c) the patient is hypotensive or (d) the patient is recovering from a seizure.

**Humidification**
The upper airway normally humidifies inspired gases. Humidified oxygen is required when the upper airway is bypassed by tracheostomy or other artificial airway. In such patients, passive (e.g. Swedish nose) or active (heated pass-over) humidification systems should be used. In patients with intact upper airways, humidification is not required for low-flow oxygen therapy or short-term use of high-flow oxygen therapy. Humidification using a heated pass-over humidifier should be used for patients using high flow nasal cannulae, high flow oxygen via face masks used for more than 24 hours, patients who report upper airway discomfort due to dryness when using oxygen therapy and recent haemoptysis.

**Special cases of poisoning**

A. Carbon monoxide
Carbon monoxide reduces arterial oxygenation by displacing oxygen that is normally bound to haemoglobin. The severity of carbon monoxide poisoning cannot be determined from arterial blood gases (because arterial PO$_2$ remains normal) or pulse oximetry (because oximetry cannot differentiate carboxyhaemoglobin from oxyhaemoglobin). Measurement of blood carboxyhaemoglobin level is required. The half-life of carboxyhaemoglobin is decreased by high concentrations of oxygen so such patients should be treated high oxygen concentrations. This can be achieved with high flow oxygen via a reservoir mask in spontaneously breathing patients and with endotracheal intubation and mechanical ventilation with 100% oxygen in comatose patients or those with severe impairment of consciousness.

B. Paraquat and bleomycin lung toxicity
Patients with paraquat poisoning or bleomycin lung injury may be harmed by oxygen therapy so it should be avoided unless the patient is hypoxaemic. If oxygen therapy is required, the target oxygen saturation is 88-92%.
### Oxygen Delivery Systems

<table>
<thead>
<tr>
<th>Delivery System</th>
<th>Flow (L/min)</th>
<th>Approx. FiO₂ (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simple nasal cannulae</td>
<td>0.5 - 4</td>
<td>22 - 40</td>
</tr>
<tr>
<td>High flow (humidified) nasal cannulae</td>
<td>6 - 40</td>
<td></td>
</tr>
<tr>
<td>Simple (Hudson) mask</td>
<td>5 - 10</td>
<td>40 - 60</td>
</tr>
<tr>
<td>Venturi mask</td>
<td>Variable</td>
<td>24 - 50</td>
</tr>
<tr>
<td>Reservoir (non-rebreathing) mask</td>
<td>10 - 15</td>
<td>60 - 90</td>
</tr>
<tr>
<td>Positive airways pressure mask, i.e. CPAP or NIV</td>
<td>0.5 - 60</td>
<td>22 - 60</td>
</tr>
<tr>
<td>Swedish nose</td>
<td>0.5 - 4</td>
<td>22 - 50</td>
</tr>
</tbody>
</table>

**Simple nasal cannulae**

Simple nasal cannulae can be used to deliver low and medium dose oxygen concentrations (22-40%). The actual inspired oxygen concentration varies between patients because of differences in the pattern of breathing. Mouth breathing does not appear to reduce the efficacy of nasal cannulae (Wettstein et al. 2005). They are generally preferred by patients over face masks (Stausholm et al. 1995) and have the advantages of improved comfort, less claustrophobia, ability to eat and speak freely, less easily dislodged, less inspiratory resistance and no risk of CO₂ rebreathing. Their disadvantages are that flow above 4 L/min tends to cause nasal dryness and discomfort if maintained for several hours and they may not be effective in patients with severe nasal obstruction.

**Simple (Hudson) mask**

Simple face masks deliver oxygen concentrations up to 60%. The flow should be at least 5 L/min because lower flows can cause resistance to inspiration and rebreathing of exhaled CO₂. The mask is suitable for patients with hypoxaemic (Type I) respiratory failure but not for patients with hypercapnoeic (Type II) respiratory failure.

**Venturi mask**

Venturi masks provide a more predictable oxygen concentration to the patient. The masks are available in the following concentrations: 24%, 28%, 31%, 35%, 40% and 50% depending on the colour-coded mask attachment used. They are suitable for all patients needing a known concentration of oxygen. The 24% and 28% Venturi masks are particularly suited to those at risk of CO₂ retention. The oxygen flow required to achieve the appropriate concentration is defined on the colour-coded mask attachment. The accuracy of the oxygen concentration delivered is greatly reduced if the mask is not accurately placed on the patient’s face, the flow is too low or the diluter jet is obstructed. The latter may be caused by clothing and can be prevented by the use of a protective hood over the jet diluter.

**Reservoir (non-rebreathing) mask**

Reservoir masks can be used to provide a higher FiO₂ than simple masks. They are most suitable in an emergency (e.g. shock, trauma) where CO₂ retention is less relevant. The bag must be...
inflated before application to the patient and should remain inflated. Deflation suggests a leak or inadequate oxygen flow and may be a sign of deterioration.

**Oxygen Alert card**

Example

| Name: ____________________________________ |
| I am at risk of type II respiratory failure with a raised CO₂ level. |
| In the event of an exacerbation, please use my Venturi mask with the 24 or 28% colour-coded attachment to achieve an oxygen saturation of 88-92%. The oxygen flow required to deliver this is recorded on the colour-coded attachment. |
| Use compressed air to drive nebulisers (with nasal oxygen at 2 L/min). |
| If compressed air not available, limit oxygen-driven nebulisers to 6 minutes. |
References


