British Thoracic Society

Guideline for emergency oxygen use in adult patients

Full Text of Guideline

Working draft updated 28th July 2007

Endorsed by Guideline Development Committee (see Section 16)

Endorsed by British Thoracic Society Standards of Care Committee March 2007

Reviewed by Peer Reviewers June 2007

For subsequent endorsement by:

Association of Respiratory Nurse Specialists
Association for Respiratory Technology and Physiology
British Association for Emergency Medicine
British Cardiovascular Society
British Paramedic Association
Chartered Society of Physiotherapy
General Practice Airways Group (GPIAG)
Intensive Care Society

Joint Royal Colleges Ambulance Liaison Committee
Resuscitation Council (UK)
Royal College of Anaesthetists
Royal College of General Practitioners
Royal College of Nursing
Royal College of Obstetricians and Gynaecologists
Royal College of Physicians
Royal College of Surgeons
Resuscitation Council (UK)
Society for Acute Medicine

To be posted on the BTS Website from 1st August 2007 for 4 weeks

To make a comment on these draft documents, please use this link to access the “Emergency Oxygen Guideline Blog” on the BTS Website:

http://www.brit-thoracic.org.uk/oxygen.html

This will allow you to submit your comments directly to the Co-Chairs of the Guideline Development Group, Ronan O’Driscoll and Tony Davison. They will respond to simple comments and queries and, if necessary, they will direct your comments to specialists within the Guideline Development Group. Your comments and the response from the Guideline Development Group will be available on the public section of the BTS website so that all interested parties can be aware of the discussion that takes place

If you prefer not to use the blog site but would like to reply to either or use individually please email: ronan.o’driscoll@srft.nhs.uk and/or adavison@southend.nhs.uk.

NB. Appendix 1-14 are included here for comment before publication. These appendices will not be included in the final document in Thorax but will be available to download from the BTS website. However, the contents of Appendix 1 (Summary of Guideline and Recommendations) will be available as a pull-out supplement within the main Guideline.
Executive summary of the Guideline

1. Philosophy of the Guideline
   - The essence of this guideline can be summarised simply as a requirement for oxygen to be prescribed according to a target saturation range and for those who administer oxygen therapy to monitor the patient and keep within the target saturation range.
   - The Guideline suggests aiming to achieve a normal or near-normal oxygen saturation for all acutely ill patients apart from those at risk of hypercapnic respiratory failure.
   - In life-threatening emergencies, high concentration oxygen should be administered immediately (Table 1 and Chart 1) and this should be recorded afterwards in the patient's health record.

2. Assessing patients
   - The oxygen saturation should be checked by pulse oximetry in all breathless and acutely ill patients, "the fifth vital sign", (supplemented by blood gases when necessary) and the inspired oxygen concentration should be recorded on the observation chart with the oximetry result.
   - Pulse oximetry must be available in all locations where emergency oxygen is used.

3. Oxygen prescription
   - Oxygen should be prescribed to achieve a target saturation of 94-98% for patients aged below 70 and 92-98% for those aged 70 or above or 88-92% for those at risk of hypercapnic respiratory failure (Table 1).
   - The target saturation should be written (or ringed) on the drug chart (Guidance on Chart 1).

4. Oxygen administration
   - Oxygen should be administered by staff who are trained in oxygen administration.
   - These staff should use appropriate devices and flow rates in order to achieve the target saturation range (Chart 2).
   - Oxygen should be signed for on the drug chart on each drug round.

5. Monitoring and maintenance of target saturation
   - Oxygen saturation and delivery code should be recorded on the patient's monitoring chart alongside the oximetry result.
   - Oxygen delivery devices and flow rates should be adjusted to keep the oxygen saturation in the target range.

6. Weaning and discontinuation of oxygen therapy
   - Oxygen should be reduced in stable patients with satisfactory oxygen saturation.
   - Oxygen should be crossed off the drug chart once oxygen is discontinued.

Oxygen is one of the most widely used drugs and is used across the whole range of specialities. The Guideline group recognise that many doctors and nurses will initially wish to read an abbreviated version of this guideline which is provided in this detachable Appendix which is also available to download from the BTS Website. www.brit-thoracic.org.uk/guidelines.html
Summary of key recommendations

Desirable oxygen saturation ranges in acute illness  Sections 6.9 and 6.12

1 This Guideline suggests aiming to achieve a normal or near-normal oxygen saturation for all acutely ill patients apart from those at risk of hypercapnic respiratory failure. Grade D

2 The suggested target saturation for patients not at risk of hypercapnic respiratory failure, aged below 70 is 94-98% and the target range for those aged 70 and above is 92-98% to reflect the wider normal range in the latter age group. Grade D

3 A sudden reduction of more than 3% in a patient's oxygen saturation within the target saturation range should prompt fuller assessment of the patient because this may be the first evidence of an acute illness. (The narrower target range in younger patients is to ensure prompt assessment if a patient falls outside the age-specific normal range, not due to greater vulnerability to hypoxia). Grade D

4 For most patients with known COPD or other known risk factors for hypercapnic respiratory failure (e.g. morbid obesity, chest wall deformities or neuro-muscular disorders), a target saturation range of 88-92% is suggested pending the availability of blood gas results. Grade C

5 Some patients with COPD are vulnerable to repeated episodes of hypercapnic respiratory failure. In these cases, it is recommended that treatment should be based on the results of previous blood gas estimations during acute exacerbations. Grade D

6 Because oxygenation is reduced in the supine position, fully conscious hypoxaemic patients should ideally be allowed to maintain the most upright posture possible (or the most comfortable posture for the patient) unless there are good reasons to immobilise the patient (e.g. skeletal or spinal trauma). Grade C

Clinical and laboratory assessment of hypoxaemia and hypercapnia  Section 7.1

7 The care of seriously ill patients should be undertaken or supervised by fully trained clinicians and expert assistance from specialists in intensive care or from other disciplines should be sought at an early stage if patients are thought to have major life-threatening illnesses. Grade D

8 The oxygen saturation should be checked by pulse oximetry in all breathless and acutely ill patients, “the fifth vital sign”, (supplemented by blood gases when necessary) and the inspired oxygen concentration should be recorded on the observation chart with the oximetry result. Grade D

9 All clinical staff who use oximeters must be trained in their use and made aware of the limitations of oximetry. Grade C

10 All patients should be fully clinically assessed, including pulse, blood pressure, assessment of circulatory blood volume and respiratory rate. The “ABC” (Airway, Breathing, Circulation) should be used when assessing any patient with apparent cardio-respiratory compromise. Grade C

11 Clinical assessment of acutely unwell patients should include the use of a recognised physiological assessment such as the Modified Early Warning Scoring System (mEWS). Grade C

12 Changes in the physiology monitoring systems such as mEWS indicate that there should be medical review of the patient even if there is no change in oxygen saturation. Grade C

13 Assess for severe anaemia because this has a major influence on oxygen delivery to the tissues. Grade D

14 Clinicians should be prepared to call for assistance when necessary including a call for a 999 Ambulance in pre-hospital care or a call for the Resuscitation Team or ICU team in hospital care. Grade C
The presence of a normal SpO2 does not always negate the need for blood gas measurements because pulse oximetry may be normal in a patient with abnormal blood pH or PCO2 or with a low blood oxygen content due to anaemia. Therefore, blood gases and full blood count tests are required as early as possible in all situations where these measurements may affect patient outcomes. Grade D

It is advised that oximetry measurements on sleeping patients should be recorded over several minutes to avoid the possibility of being misled by a normal transient nocturnal “dip” in oxygen saturation. Grade C

In cases of carbon monoxide poisoning, a normal or high oximetry reading should be disregarded because an apparently “normal” saturation may be produced by carboxyhaemoglobin in a patient who needs high dose oxygen therapy due to tissue hypoxaemia. Grade C

Pulse oximetry can be misleadingly normal in smokers because of raised blood carboxyhaemoglobin levels which can conceal the presence of arterial hypoxaemia. Therefore, blood gases should be checked in patients with borderline oximetry levels who have smoked cigarettes in the previous few hours (i.e. 93% or less if aged 70 or above and 95% or less if aged below 70). Grade B

For most patients who require blood gas sampling, either ABG or arteriolised Earlobe Blood Gases (ELBG) may be used but the PaO2 is less accurate in ELBG samples so oximetry should be monitored carefully if ELBG specimens are used. Grade B

For critically ill patients or those with shock or hypotension (systolic blood pressure below 90 mm Hg), the initial blood gas measurement should be obtained from an arterial specimen. Grade D

The technique of patient preparation, sample acquisition and sample processing for arteriolised capillary gases is complex and should only be undertaken by fully trained staff. Grade D

Local anaesthesia should be used for all arterial blood gas specimens except in emergencies or if the patient is unconscious or anaesthetised. Grade B

Emergency oxygen use in hospital setting Sections 8.3 and 8.4

- Oxygen saturation should be measured in all breathless and acutely ill patients. (Recommendation 8)

Oxygen therapy should be given to hypoxaemic patients (see table 1) but most normoxaemic patients do not require oxygen therapy. (Patients on oxygen with SpO2 above 98% do not require oxygen therapy or may require a lower dose) Grade D.

All patients with shock, major trauma, sepsis or other critical illness should be managed initially with high concentration oxygen therapy from a reservoir mask at 10-15 l/min. The dose can be adjusted subsequently once the results of blood gas estimations are known and/or the patient is stable. Grade D

Which patients require blood gas measurements? Section 8.4

Blood gases should be checked in the following situations.

- Unexpected or inappropriate hypoxaemia (SpO2 below 94% in patients aged up to 70 breathing room air or oxygen or SpO2 below 92% in patients aged 70 and above) or any patient requiring oxygen to achieve the above targets. (Allowance should be made for transient dips in saturation to 90% or less in normal subjects during sleep). Grade D

- Deteriorating oxygen saturation or increasing breathlessness in a patient with previously stable hypoxaemia (e.g. severe COPD).
- Any previously stable patient who deteriorates and requires a significantly increased fraction of inspired oxygen (FIO2) to maintain a constant oxygen saturation. Grade D

- Any patient with risk factors for hypercapnic respiratory failure who develops acute breathlessness, deteriorating oxygen saturation, or drowsiness or other symptoms of CO2 retention. Grade D

- Breathless patients who are thought to be at risk of metabolic conditions such as diabetic ketoacidosis or metabolic acidosis due to renal failure. Grade D

- Any other evidence from the patient’s medical condition that would indicate that blood gas results would be useful in the patient’s management (e.g. unexpected sudden rise of several units in modified Early Warning Score or an unexpected fall in oxygen saturation of 3% or more, even if within the target range). Grade D

Table 1

<table>
<thead>
<tr>
<th>Critical illnesses requiring high levels of supplemental oxygen</th>
<th>Section 8.10</th>
</tr>
</thead>
</table>

The initial oxygen therapy is a reservoir mask at 15 l/min.

Once stable, reduce the oxygen dose and aim for target saturation range of 94-98% if aged <70 and 92-98% saturation if aged ≥70

Patients with risk factors for hypercapnia who develop critical illness should have the same initial target saturations as other critically ill patients pending the results of blood gas results after which these patients may need controlled oxygen therapy or supported ventilation if there is severe hypoxia and/or hypercapnia.

<table>
<thead>
<tr>
<th>Additional Comments</th>
<th>Recommendation number and grade</th>
</tr>
</thead>
</table>
| Cardiac Arrest or Resuscitation | Use bag-mask during active resuscitation.  
Aim for maximum possible oxygen saturation.  
26 Grade D |
| Shock Sepsis Major Trauma Near-Drowning Anaphylaxis Major Pulmonary Haemorrhage | Once stable, reduce the oxygen dose and aim for target saturation range of 94-98% if aged <70 and 92-98% saturation if aged ≥70  
27 Grade D  
27 Grade D  
27 Grade D |
| Major Head Injury | Early intubation and ventilation if comatose.  
31 Grade D |
| Carbon | Aim for 100% saturation.  
32 Grade C |
### Monoxide Poisoning

Oximeter reading and blood gas PaO2 are misleading. Check carboxyhaemoglobin levels.

### Table 2

**Serious illnesses requiring moderate levels of supplemental oxygen if the patient is hypoxaemic**  
*Section 8.11*

The initial oxygen therapy is nasal cannulae at 2-6 l/min or simple face mask at 5-10 l/min unless stated otherwise.

**Recommended initial oxygen saturation target range is 94-98% if aged <70 and 92-98% saturation if aged ≥70**

Change to reservoir mask if the desired saturation range cannot be maintained with nasal cannulae or simple face mask (and ensure that the patient is assessed by senior medical staff)

If these patients have co-existing COPD or other risk factors for hypercapnic respiratory failure, aim at a saturation of 88-92% pending blood gas results but adjust to 92-98% if the PaCO2 is normal (unless there is a history of previous hypercapnic respiratory failure) and recheck blood gases after one hour.

<table>
<thead>
<tr>
<th><strong>Additional Comments</strong></th>
<th><strong>Recommendation number and grade</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acute hypoxaemia-cause not yet diagnosed</strong></td>
<td>33-35     Grade D</td>
</tr>
<tr>
<td>Reservoir mask at 10-15 l/min if initial SpO2 below 90%, otherwise nasal cannulae or simple face mask. <em>Patients requiring reservoir mask therapy need urgent clinical assessment by senior staff.</em></td>
<td></td>
</tr>
<tr>
<td><strong>Acute Asthma</strong></td>
<td>36        Grade C</td>
</tr>
<tr>
<td><strong>Pneumonia</strong></td>
<td>37        Grade C</td>
</tr>
<tr>
<td><strong>Lung Cancer</strong></td>
<td>38        Grade C</td>
</tr>
<tr>
<td><strong>Post-operative Breathlessness</strong></td>
<td>39        Grade D</td>
</tr>
<tr>
<td>Management depends on underlying cause.</td>
<td></td>
</tr>
<tr>
<td><strong>Acute Heart Failure</strong></td>
<td>40        Grade D</td>
</tr>
<tr>
<td>Consider CPAP or NIV in cases of pulmonary oedema.</td>
<td></td>
</tr>
<tr>
<td><strong>Pulmonary Embolism</strong></td>
<td>41        Grade D</td>
</tr>
<tr>
<td>Most patients with minor pulmonary embolism are not hypoxaemic and do not require oxygen therapy.</td>
<td></td>
</tr>
<tr>
<td><strong>Pleural Effusions</strong></td>
<td>42        Grade D</td>
</tr>
<tr>
<td>Additionally, treat by draining the effusion.</td>
<td></td>
</tr>
<tr>
<td><strong>Pneumothorax</strong></td>
<td>43-44     Grade C and Grade D</td>
</tr>
<tr>
<td>Reservoir mask at 10-15 l/min if admitted for observation. Aim at 100% saturation. 100% oxygen accelerates clearance of pneumothorax if drainage is not required. Needs aspiration or drainage if the patient is hypoxaemic.</td>
<td></td>
</tr>
<tr>
<td><strong>Deterioration of lung fibrosis or other interstitial lung disease</strong></td>
<td>45        Grade D</td>
</tr>
<tr>
<td>Reservoir mask at 10-15 l/min if initial SpO2 below 90%, otherwise nasal cannulae or Simple face mask.</td>
<td></td>
</tr>
<tr>
<td><strong>Severe Anaemia</strong></td>
<td>46-47     Grade B and Grade D</td>
</tr>
<tr>
<td>The main issue is to correct the anaemia. Most anaemic patients do not require oxygen therapy.</td>
<td></td>
</tr>
<tr>
<td><strong>Sickle cell crisis</strong></td>
<td>48        Grade D</td>
</tr>
<tr>
<td>Requires oxygen only if hypoxaemic.</td>
<td></td>
</tr>
</tbody>
</table>
Table 3

### Patients requiring controlled or low-dose oxygen therapy  Section 8.12

The initial oxygen therapy is 24% or 28% Venturi mask

The recommended initial oxygen saturation target range is 88-92% for most at-risk patients pending blood gas results.

Adjust target range to 94-98% (age below 70) or 92-98% (age 70 or above) if the PaCO2 is normal (unless there is a history of previous hypercapnic respiratory failure) and recheck blood gases after one hour (recommendations 53-54 and Chart 1).

Change to nasal cannulae when the patient is clinically stable—see recommendation 92.

<table>
<thead>
<tr>
<th></th>
<th>Initial oxygen therapy</th>
<th>Additional Comments</th>
<th>Recommendation number and grade</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>COPD</strong></td>
<td>24% Venturi at 2 litres/minute or 28% Venturi mask at 4 litres/minute</td>
<td>May need lower range if acidicotic or if known to be very sensitive to oxygen therapy. Ideally use “Alert Cards” to guide therapy based on previous blood gas results. Increase flow by 50% if respiratory rate is above 30, see recommendation 93</td>
<td>49-56 Grade C</td>
</tr>
<tr>
<td><strong>Exacerbation of Cystic Fibrosis</strong></td>
<td>24% Venturi at 2-4 litres/minute or 28% Venturi mask at 4 litres/minute</td>
<td>Admit to Regional CF centre if possible, if not discuss with regional centre or manage according to protocol agreed with regional CF centre. Ideally use “Alert Cards” to guide therapy. Increase flow by 50% if respiratory rate is above 30, see recommendation 93</td>
<td>57 Grade D</td>
</tr>
<tr>
<td><strong>Neuro-Muscular Disorders</strong></td>
<td>Depends on severity of hypoxaemia. Usually 24% Venturi at 2 litres/minute or 28% Venturi mask at 4 litres/minute</td>
<td>May require ventilatory support. Risk of hypercapnic respiratory failure.</td>
<td>58 Grade D</td>
</tr>
<tr>
<td><strong>Chest Wall disorders</strong></td>
<td></td>
<td></td>
<td>58 Grade D</td>
</tr>
<tr>
<td><strong>Morbid obesity</strong></td>
<td></td>
<td></td>
<td>59-60 Grade D</td>
</tr>
</tbody>
</table>
Patients requiring low dose controlled oxygen therapy  Sections 8.3 and 8.4

49 Patients over 50 years of age who are long-term smokers with a history of exertional breathlessness and no other known cause of breathlessness should be treated as if having COPD for the purposes of this guideline. Grade D

50 Patients with a significant likelihood of severe COPD or other illness that may cause hypercapnic respiratory failure should be triaged as very urgent (Orange Status) on arrival in hospital Emergency Departments (and blood gases should be taken on arrival). Grade D

51 Prior to availability of blood gases, use a 24% Venturi mask at 2 l/min or 28% Venturi mask at 4 l/min and aim for an oxygen saturation of 88-92% for patients with risk factors for hypercapnia but no prior history of type 2 respiratory failure. Grade D

- Increase flow by 50% if respiratory rate is above 30  See recommendation 95

52 Aim at a pre-specified target saturation range (if available) in patients with a history of previous respiratory acidosis. In many cases, the ideal target saturation will be specified on patient’s alert card. If no information is available, aim at a saturation of 88-92% pending blood gas results. Grade D

53 If, following blood gases the pH and PCO2 are normal, aim for oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above unless there is a history of previous hypercapnic respiratory failure. Grade D

54 Recheck blood gases after one hour for all patients with COPD or other risk factors for hypercapnic respiratory failure even if the initial PaCO2 measurement was normal. Grade D

55 If the PaCO2 is raised but pH is satisfactory, the patient has probably got long-standing hypercapnia; maintain target range of 88-92% for these patients. Blood gases should be repeated at one hour to check for rising PaCO2 or falling pH. Grade D

56 If the patient is hypercapnic (PaCO2 > 6.0 kPa or 45 mm Hg) and acidic (pH < 7.35) consider non-invasive ventilation, especially if the acidosis has persisted for more than 30 minutes despite appropriate therapy. Grade A

57 Initial therapy of Cystic Fibrosis exacerbations should be similar to the initial therapy of COPD exacerbations (see section 8.12.1). Grade D

58 In the initial management of musculo-skeletal and neurological disorders with acute respiratory failure, aim at an oxygen saturation of 88-92%. Many such patients will be suitable for Non-invasive ventilation. Grade D

59 In the initial management of obesity-hypoventilation syndrome with acute exacerbation, aim at an oxygen saturation of 88-92%  Grade D

60 Non-invasive ventilation should be considered for these patients  Grade C
Table 4

Conditions for which oxygen therapy is not required unless the patient is hypoxaemic  Section 8.13

If hypoxaemic, the initial oxygen therapy is nasal cannulae at 2-6 l/min or simple face mask at 5-10 l/min unless stated otherwise (see comments section)

Recommended initial oxygen saturation target range, unless stated otherwise, is 94-98% if aged <70 and 92-98% saturation if aged ≥70

If patients have COPD or other risk factors for hypercapnic respiratory failure, aim at a saturation of 88-92% pending blood gas results but adjust to 94-98% (age below 70) or 92-98% (age 70 or above) if the PaCO2 is normal (unless there is a history of previous hypercapnic respiratory failure) and re-check blood gases after one hour.

<table>
<thead>
<tr>
<th>Additional Comments</th>
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</tr>
</thead>
<tbody>
<tr>
<td><strong>Myocardial infarction and Acute Coronary Syndromes</strong></td>
<td>Most patients with acute coronary artery syndromes are not hypoxaemic and the benefits/harms of oxygen therapy are unknown in such cases.</td>
</tr>
<tr>
<td><strong>Stroke</strong></td>
<td>Most stroke patients are not hypoxaemic. Oxygen therapy may be harmful for non-hypoxaemic patients with mild-moderate strokes.</td>
</tr>
<tr>
<td><strong>Obstetric Emergencies</strong></td>
<td>Oxygen therapy may be harmful to the fetus if the mother is not hypoxaemic.</td>
</tr>
<tr>
<td><strong>Hyperventilation or dysfunctional breathing</strong></td>
<td>Unlikely to require oxygen therapy. Exclude organic illness</td>
</tr>
<tr>
<td><strong>Most poisonings and drug overdoses</strong></td>
<td>Hypoxia is more likely with respiratory depressant drugs, give antidote if available. e.g. Naloxone for opiate poisoning</td>
</tr>
<tr>
<td><strong>Poisoning with Paraquat Bleomycin or Acid Inhalation</strong></td>
<td>Patients with Paraquat poisoning, Bleomycin lung injury or acid inhalation may be harmed by supplemental oxygen. <strong>Avoid oxygen unless the patient is hypoxaemic.</strong>  <strong>Target saturation is 88-92%</strong></td>
</tr>
<tr>
<td><strong>Metabolic &amp; Renal disorders</strong></td>
<td>Most do not need oxygen</td>
</tr>
</tbody>
</table>
Obstetric Emergencies and Labour Section 8.13.3

63 Women who suffer from major trauma, sepsis or acute illness during pregnancy should receive the same oxygen therapy as any other seriously ill patients, with a target oxygen saturation of 94-98%. The same target range should be applied to women with hypoxaemia due to acute complications of pregnancy (e.g. collapse related to amniotic fluid embolus, eclampsia or antepartum or postpartum haemorrhage) Grade D

64 Women with underlying hypoxaemic conditions (e.g. heart failure) should be given supplemental oxygen during labour to achieve an oxygen saturation of 94-98% Grade D

65 All women with evidence of hypoxaemia in late pregnancy should be managed with left lateral tilt to improve cardiac output. Grade B

66 The use of oxygen during labour is widespread, but with evidence this may be harmful to the fetus this is not currently recommended in situations where the mother is not hypoxaemic (except as part of a controlled trial). Grade A

Anxiety and hyperventilation or dysfunctional breathing Section 8.13.4

67 Organic illness must be excluded before making a diagnosis of hyperventilation. Grade C

68 Patients with a definite diagnosis of hyperventilation should have their oxygen saturation monitored. Those with normal or high SpO2 do not require oxygen therapy. Grade B

69 Re-breathing from a paper bag can be dangerous and is NOT recommended as a treatment for hyperventilation. Grade C

Emergency use of oxygen in ambulances, community and pre-hospital setting Sections 9.1-9.8 and 10.4

73 Pulse oximetry must be available in all locations where emergency oxygen is being used. Grade D

74 Emergency oxygen should be available in primary care medical centres; preferably using oxygen cylinders with integral high-flow regulators. Alternatively, oxygen cylinders fitted with high-flow regulators (delivering over 6 L/min) must be used. Grade D

75 All documents which record oximetry measurements should state whether the patient is breathing air or a specified dose of supplemental oxygen. Grade C

- Clinical assessment of a breathless patient starts with ABC (Airway, Breathing, Circulation, See recommendation 9)
- A brief history should be taken from the patient or other informant.
- Initial assessment should include pulse and respiratory rate in all cases. See recommendations 11-12
- Pulse oximetry should always be measured in patients with breathlessness or suspected hypoxia. See recommendation 8
- Disease-specific measurements should also be recorded (e.g. Peak Expiratory Flow in asthma, Blood Pressure in cardiac disease.
- The initial oxygen therapy to be used in the various clinical situations is given in tables 1-4.
- If there is a clear history of asthma or heart failure, or other treatable illness, then appropriate treatment should be instituted in accordance with guidelines or standard management plans for each disease.
76 The oxygen saturation should be monitored continuously until the patient is stable or arrives at hospital for a full assessment. The oxygen flow should be adjusted upwards or downwards to maintain the target saturation range. Grade D

77 In most emergency situations, oxygen is given to patients immediately without a formal prescription. However, a subsequent written record must be made of what oxygen therapy has been given to every patient (in a similar manner to the recording of all other emergency treatment). Grade D

78 Patients with COPD should initially be given oxygen via a Venturi 24% mask at a flow rate of 2 l/min or 28% mask at a flow rate of 4 l/min and oxygen saturation should be 88-92% in most cases or else an individualised saturation range based on the patient's blood gas measurements during previous exacerbations. Grade C

- Patients over 50 years of age who are long-term smokers with a history of exertional breathlessness and no other known cause of breathlessness should be treated as if having COPD. See recommendation 52

79 COPD patients and other patients who have had an episode of hypercapnic respiratory failure should be issued with an oxygen warning card and with a 24% or 28% Venturi mask. They should be instructed to show the card to the ambulance crew and Emergency Department staff in the event of an exacerbation. Grade C

80 The content of the Alert Card should be specified by the physician in charge of the patient’s care, based on previous blood gas results. Grade D

81 The primary care team and ambulance control should also be informed by a responsible clinician that the patient has had an episode of hypercapnic respiratory failure and carries an Oxygen Alert Card. These patients home addresses and ideal oxygen dose or target saturation ranges can be flagged in the ambulance control systems and disseminated to ambulance crews when required. Grade D

82 Out of hours services providing emergency Primary Care services should be informed by a responsible clinician that the patient has had an episode of hypercapnic respiratory failure and carries an Oxygen Alert card. Use of oxygen in these patients will be guided by the instructions on the Alert Card. Grade D

83 During ambulance journeys, oxygen driven nebulisers should be used for patients with asthma and may be used for COPD patients in the absence of an air-driven compressor system. If oxygen is used for patients with known COPD, it should be limited to 6 minutes. This will deliver most of the nebulised drug dose but limit the risk of hypercapnic respiratory failure. (section 10.8.2) Grade D.

84 It is recommended that the following delivery devices should be available in pre-hospital settings where oxygen is administered. Grade D See Recommendations 88-95 in section 10.5.4

1. High concentration reservoir mask (non-rebreathe mask) for high-dose oxygen therapy.
2. Nasal cannulae (preferably) or simple face mask for medium dose oxygen therapy.
3. 28% Venturi mask for patients with known previous hypercapnic respiratory failure with inappropriately high arterial blood oxygen values (patients who have an oxygen alert card may have their own 24% or 28% Venturi mask)
4. Tracheostomy masks for patients with tracheostomy or previous laryngectomy.
85 Trusts should take measures to minimise the risk of oxygen tubing being connected to the incorrect wall oxygen outlet or to outlets that deliver compressed air instead of oxygen. Air flow meters should be removed from the wall sockets or covered with a designated air outlet cover when not in use. Special care should be taken if twin oxygen outlets are in use. Grade D

- Emergency oxygen should be available in primary care medical centres; preferably using oxygen cylinders with integral high-flow regulators. Alternatively, oxygen cylinders fitted with high-flow regulators (delivering over 6 L/min) must be used. See recommendation 74

86 Most patients can be managed with one of 4 types of oxygen delivery device. Grade D

1. High concentration reservoir mask (non-rebreath mask) for high-dose oxygen therapy.
2. Nasal cannulae or simple face mask for medium and low dose oxygen therapy. (Nasal cannulae are the preferred option unless there is a specific reason to use a simple face mask)
3. 24% or 28% Venturi mask for patients with known previous hypercapnic respiratory failure with inappropriately high arterial blood oxygen values.
4. Tracheostomy masks for patients with tracheostomy or previous laryngectomy.

87 The high-dose reservoir mask at 10-15 l/min is the preferred means for delivering high dose oxygen to critically ill patients. Grade D

88 Nasal cannulae should be used rather than simple face masks in most situations requiring medium-dose oxygen therapy. Nasal cannulae are preferred by patients for reasons of comfort and they are less likely to be removed during meals etc. (see section 10.2.4) There is also a cost saving. Grade C

89 The flow rate from nasal cannulae should be adjusted between 2 and 6 litres per minute to achieve the desired target saturation. Grade C

90 The flow rate from simple face masks should be adjusted between 5 and 10 litres per minute to achieve the desired target saturation. Grade C

91 Venturi Masks are recommended for patients at risk of hypercapnic respiratory failure. Venturi masks can deliver a constant FIO2 of 24% or 28% oxygen with a greater gas-flow than a simple face mask. This achieves a reduced risk of carbon-dioxide rebreathing compared with a simple face mask and less likelihood of dilution of the oxygen stream by room air if the patient’s inspiratory flow rate exceeds the flow rate delivered by the face-mask. Grade D

92 Venturi masks can be substituted with nasal cannulae at low flow rates (1-2 l/min to achieve the same target range) once patients have stabilized. Grade D

93 Patients with a respiratory rate above 30 breaths per minute should have the flow rate set to 50% above the minimum flow rate specified on the Venturi mask and/or packaging. Grade C.

94 Humidification is not required for the short term delivery of low flow oxygen (up to 3 days oxygen). It is not therefore required in pre-hospital care. Grade B

95 In the emergency situation, humidified oxygen use can be confined to patients with tracheostomy or an artificial airway but these patients can be managed without humidification for short periods f time (e.g. ambulance journeys). Grade D

96 Humidification may also be of benefit to patients with viscous secretions causing difficulty with expectoration. This benefit can be achieved using nebulised normal saline. Grade C
97 Bubble bottles should not be used because there is no evidence of clinical benefit but there is an infection risk. Grade C

98 When oxygen is required by patients with prior laryngectomy, a tracheostomy mask (varying the flow as necessary) should achieve the desired oxygen saturation (tables 1 to 4). An alternative delivery device, usually a two piece device fitted directly to the tracheostomy tube may be necessary if the patient deteriorates. Grade D

99 For patients with asthma, nebulisers should be driven by piped oxygen or from an oxygen cylinder fitted with a high flow regulator capable of delivering a flow rate over 6L/min. The patient should be changed back to their usual mask when nebuliser therapy is complete. If the cylinder does not produce this flow rate, then an air-driven nebuliser (with electrical compressor) should be used with supplemental oxygen by nasal cannulae at 2-6L/m to maintain an appropriate oxygen saturation level. Grade D

100 When nebulised broncho-dilators are given to hypercapnic acidotic patients, they should be driven by compressed air and, if necessary, supplementary oxygen should be given concurrently by nasal prongs at 2-4 litres per minute to maintain an oxygen saturation of 88-92%. The same precautions should be applied to patients who are at risk of hypercapnic respiratory failure prior to the availability of blood gas results. Grade D

101 Once the nebulised treatment is completed for patients at risk of hypercapnia, controlled oxygen therapy with a fixed concentration (Venturi) device should be re-instituted. Grade D

- During ambulance journeys, oxygen driven nebulisers may be used in the absence of an air-driven compressor system. If oxygen is used, it should be limited to 6 minutes for patients with known COPD. This will deliver most of the nebulised drug dose but limit the risk of hypercapnic respiratory failure. See Recommendation 84

**Prescription, Administration and Monitoring of oxygen therapy Section 11**

**General Section 11.1.6**

102 Every health care facility should have a standard oxygen prescription document or, preferably, a designated oxygen section on all drug prescribing cards. Grade C

- Oxygen saturation should be measured in all breathless patients and supplemental oxygen should be given to all breathless hypoxaemic patients and to all critically ill patients. Oxygen saturation should be measured under as optimal conditions as possible e.g. nail varnish should be removed. See recommendation 8

- Clinicians should assess the clinical status of the patient prior to prescribing oxygen and the patient's condition should be reassessed frequently during oxygen use. See recommendations 11-12

103 All oxygen should be prescribed except in life-threatening emergencies when it should be started immediately. Grade D

104 Doctors should prescribe oxygen using a target saturation range (Sections 6, 8, 9 and 11) and sign the drug chart. (Chart 3) Grade D

105 In all situations where repeated blood gases are required, they should be measured as soon as possible to determine if the proposed target saturations are appropriate. Grade D

106 The oxygen dose should be increased by staff who have been trained to administer oxygen if the oxygen saturation falls below the pre-specified range and the dose should be reduced if the saturation rises above this range. If the monitoring of oxygen saturations is performed by other staff (e.g. Health Care Assistants) they should inform staff who are trained to administer oxygen if the oxygen saturation is above or below the target saturation. Grade D Also see recommendations 115, 116, 122, 124 and 136
107  All clinicians prescribing oxygen should have appropriate training and access to written or electronic oxygen prescribing guidelines based on this national guideline. Grade D

108  Every hospital should have a training programme to ensure that clinical staff are familiar with the hospital's oxygen administration policies. Grade D

- For hypoxaemic patients, oxygen therapy should continue during other treatments such as nebulised therapy. See recommendations 99-100

**Use of Drug Chart** Section 11.1.7

109  In most emergency situations, oxygen is given to patients immediately without a formal prescription. However, a subsequent written record must be made of what oxygen therapy has been given to every patient (in a similar manner to the recording of all other emergency treatment). Grade D

110  The prescription should be signed by the doctor or other prescribing clinician and and check that that the patient is receiving oxygen therapy. Grade D

111  Nurses should sign the drug chart at every drug round and check that patient’s oxygen saturation is within the target range. Grade D

112  Most patients are prescribed continuous oxygen. However some patients may be prescribed oxygen PRN (as required). In this scenario if patients are on air at the time of the drug round, nurses should still sign the drug chart but the observation chart should be filled in using the code AX (see Chart 4) Grade D

**Starting oxygen therapy** Section 11.2

- The administering health care professional should note the oxygen saturation prior to commencing oxygen therapy. See recommendation 8

113  The health care professional should commence oxygen therapy using an appropriate delivery system and flow rate as specified in sections 8, 9 and 10 of this guideline. The target oxygen saturation and whether the patient is having continuous oxygen, PRN or no oxygen therapy should be circled on the respiratory section of the observation chart. Grade D

114  Whenever possible, patients should be given an oxygen information sheet (example in appendix 5 of this Guideline) Grade D

**Monitoring oxygen therapy** Section 11.3.2 - 11.3.3

- All measurements of oxygen saturation should be recorded in the observation chart along with the code for the oxygen delivery system that is being used (including the various codes if the patient is breathing air) Chart 4. See recommendation 8

- Arterial or arteriolsed capillary blood gases should be measured and the inspired oxygen concentration noted on arrival at hospital (or at the time when oxygen therapy becomes necessary) for most patients requiring emergency oxygen therapy. See recommendation 25

- Blood gases should be repeated in all critically ill patients and in many other cases according to response to treatment. See recommendation 25
- All acutely ill patients should have physiological monitoring using Early Warning Scores or a similar physiological assessment system in addition to pulse oximetry - see section 7. See recommendation 11

Monitoring during the first hour of oxygen therapy  

Monitoring during the first hour of oxygen therapy
Section 11.3.4

115 All patients should have their oxygen saturation observed for the first five minutes after starting oxygen therapy. Grade D

116 If the oxygen saturation should fall below the target saturation and the patient is unstable medical advice should be sought. Grade D

117 If the oxygen saturation is above the target saturation range and the patient is stable, the delivery system and oxygen flow rate should be reduced accordingly. Grade D

118 Patients who have a target saturation of 88-92% should have their blood gases measured within 30-60 minutes. This is to ensure that the carbon-dioxide level is not rising. This recommendation also applies to those who are at risk of developing hypercapnic respiratory failure but who have a normal PaCO2 on the initial blood gas measurement. Grade D

119 Stable patients whose oxygen saturation is within their target saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above do not need repeat blood gases within 30-60 minutes if there is no risk of hypercapnia and acidosis and may not need any further blood gas measurements. Grade D

Subsequent monitoring  

Section 11.3.5

120 Stable patients on oxygen treatment should have SpO2 measured four times-a-day. Grade D

121 In those who are unstable, oxygen saturation should be monitored continuously and the patient should ideally be managed in a high-dependency area. Grade D

122 If the patient is clinically stable and the oxygen saturation is within the target range treatment should be continued (or eventually lowered) depending on the clinical situation. Grade D

123 Any sudden fall in oxygen saturation should lead to clinical evaluation of the patient and, in most cases, measurement of blood gases. Grade D

124 Oxygen therapy should be increased if the saturation is below the desired range and decreased if the saturation is above the desired range (and eventually discontinued as the patient recovers). Grade D

125 Monitor the saturation continuously for 5 minutes after any increase or decrease in oxygen dose to ensure that the patient achieve the desired saturation range. Grade D

126 Record the new saturation (and the new delivery system) on the patient's observation chart after 5 minutes of therapy at the new oxygen dose. Each change should be recorded by the clinician trained to administer oxygen by signing the observation chart (only changes should be signed for). Grade D

127 Repeat blood gases are not required for stable patients who require a reduced dose of oxygen (or cessation of oxygen therapy) to maintain the desired target saturation. Grade D

128 Patients with no risk of hypercapnia do not always need repeat blood gases after an increase in oxygen dose but should have clinical review to determine why the oxygen saturation has fallen. Grade D
Patients at risk of hypercapnia (usually those with a target range of 88-92%; see Table 3) require repeat blood gas estimation 30-60 minutes after an increase in oxygen therapy. Grade D

For patients with no risk of hypercapnia, monitoring by pulse oximeter is sufficient (repeated blood gases not required) provided the saturation remains in the desired range, usually 94-98% if aged below 70 and 92-98% if aged 70 and above.

**When to increase oxygen therapy**  Section 11.3.6

131 If a patient's oxygen saturation is lower than the prescribed target range, first check all aspects of the oxygen delivery system for faults or errors. Grade D

132 If a patient's oxygen saturation is consistently lower than the prescribed target range, there should usually be a medical review and the oxygen therapy should be increased according to an agreed written protocol. Grade D

133 The patient should be observed for five minutes after oxygen therapy has been increased. Grade D

134 If the oxygen saturation fails to rise following 5-10 minutes of increased oxygen therapy or if there is clinical concern following medical review, then blood gases should be repeated. Grade D

135 If the target saturation is between 88-92% range, blood gases should be repeated at 30-60 minutes after any increase in oxygen therapy to ensure that the carbon dioxide level is not rising. Grade D

**When to lower oxygen therapy**  Section 11.3.7

136 Lower the oxygen dose if the target saturation is higher than the prescribed range. Grade D

137 Lower the oxygen dose if the patient is clinically stable and the oxygen saturation has been in the upper zone of the target range for some time (usually 4-8 hours). Grade D

138 Saturations should be observed for five minutes following a change of oxygen therapy. Grade D

139 If the target saturation is maintained, the new delivery system and flow should be continued. Repeat blood gases are not required. If the patient is stable the process can be repeated and the patient can eventually be weaned off oxygen-see section 12. Grade D

**Discontinuation of oxygen therapy**  Section 12.1

- Reduce oxygen therapy gradually for stable patients. See section 11.3.7 and recommendations 136-139

140 The lowest dose of oxygen for most stable convalescent patients will be 2 litres per minute via nasal cannulae and 1 litre per minute via nasal cannulae or a 24% Venturi mask for patients at risk of hypercapnic respiratory failure. Grade D

141 Stop oxygen therapy once a patient is clinically stable on low-dose oxygen and the oxygen saturation is within the desired range on 2 consecutive observations. Oxygen should also be stopped if the patient is on a written protocol of timed oxygen e.g post-operatively. Grade D

142 Monitor the oxygen saturation on room air for 5 minutes after stopping oxygen therapy. If it remains in the desired range, recheck at one hour. Grade D
143 If the oxygen saturation and mEWS is satisfactory at one hour, the patient has safely discontinued oxygen therapy but continue to monitor saturation and mEWS on a regular basis according to the patient's underlying clinical condition. Grade D

144 If the saturation falls on stopping oxygen therapy, recommence the lowest dose that maintained the patient in the target range and monitor for 5 minutes. If this restores the saturation into the target range, continue oxygen therapy at this level and attempt discontinuation of oxygen therapy again at a later date provided the patient remains clinically stable. Grade D

145 If a patient requires oxygen therapy to be restarted at a higher dose than before to maintain the same target saturation range, then the patient should have clinical review to establish the cause for this deterioration. Grade D

146 Some patients may have episodic hypoxaemia (e.g. after minor exertion) after they have safely discontinued continuous oxygen therapy. If these patients require intermittent oxygen therapy, they should have a prescription for oxygen as required ("PRN"). Grade D

147 Cross oxygen off the drug chart when oxygen discontinued (and sign to confirm discontinuation). Grade C
Is this patient at risk of hypercapnic respiratory failure (Type 2 Respiratory Failure)?

**The main risk factor is severe or moderate COPD**

*Especially patients with previous Respiratory Failure or on Long Term Oxygen*

**Other patients at risk include people with severe chest wall or spinal disease (e.g. kypho-scoliosis), neuro-muscular disease, severe obesity, cystic fibrosis, bronchiectasis or previously un-recognised COPD**

Narcotic/ sedative overdose not covered by this algorithm. Treat with opioid antagonist and O₂ as appropriate.

---

**YES**

Target saturation is 88-92% whilst awaiting blood gas results

Obtain ABGs

- **pH < 7.35 * and PCO₂ > 6.0**
  - **Respiratory Acidosis**
  - or patient tiring
  - Seek immediate senior review
  - Consider NIV or invasive ventilation

- **pH ≥ 7.35 and PCO₂ > 6.0**
  - **Hypercapnia**
  - Treat with the lowest strength Venturi mask that will keep SpO₂ between 88-92%
  - Repeat ABGs at 1 hour:
    - If **Respiratory Acidosis** (pH < 7.35 & PCO₂>6.0)
      - Seek immediate senior review, consider NIV/ICU.
      - Consider reducing FIO₂ if PO₂ ≥ 8.0 kPa

---

**NO**

Aim for SpO₂ 94-98% if aged <70 and 92-98% if aged ≥70

SpO₂ ≤ 92% on air or oxygen (<94% if aged below 70)

or if requiring oxygen to achieve above targets

- **PCO₂ ≤ 6.0* (Normal or low)**
  - **Seek immediate senior review**
  - Consider reducing FIO₂ if PO₂ ≥ 8.0 kPa
  - Treat appropriately aiming to keep SpO₂ between 94-98% if aged below 70 and 92-98% if aged 70 and above
  - If target saturation not achieved, seek medical review.

- **PCO₂ ≥6.0 or patient tiring**
  - **Seek immediate senior review**
  - Consider NIV or invasive ventilation
  - Treat urgently. Aim for SpO₂ 94-98% if aged below 70 and 92-98% if aged 70 and above pending senior review. Also consider COPD or other undiagnosed chronic hypercapnic respiratory failure
  - If likely aim for SpO₂ of 88-92%

- **Monitor SpO₂**
  - O₂ may not be required or be prescribed prn

---

Any increase in FIO₂ must be followed by repeat ABGs in 1 hour (or sooner if conscious level deteriorates)

*If pH is < 7.35 with normal or low PaCO₂, investigate and treat for Metabolic Acidosis and keep SpO₂ 94-98% if aged below 70 and 92-98% if aged 70 and above*
**Repeat ABG in 1 hour for all patients at risk of Type 2 respiratory failure (even if initial PaCO2 is normal)**
Chart 2
Flow chart for oxygen administration on general wards

See patient’s drug chart for starting dose and target saturation
Choose the most suitable delivery system and flow rate

Titrate oxygen **up or down** to maintain the target oxygen saturation.

*The table below shows available options for stepping dosage up or down*
*Allow at least 5 minutes at each dose before adjusting further upwards or downwards (except with major and sudden fall in saturation)*

Once your patient has adequate and stable saturation on minimal oxygen dose, consider discontinuation of oxygen therapy.

![Flow chart](chart.png)

**Signs of Respiratory Deterioration**
- ↑ Respiratory rate
- ↓ SpO₂
- Increased oxygen dose needed to keep saturation in target range
- ↑ Work of breathing
- ↑ EWS
- CO₂ Retention
  - Drowsiness
  - Headache
  - Flushed face
  - Tremor

Seek Medical Advice

**All Patients must** have ABG or Earlobe Blood Gases (ELBG) within 1 hour of requiring increased oxygen dose

**PATIENTS IN A PERI ARREST SITUATION AND CRITICALLY ILL PATIENTS SHOULD BE GIVEN MAXIMAL OXYGEN VIA RESERVOIR MASK OR RESUSITATION MASK WHILST IMMEDIATE MEDICAL HELP IS ARRIVING.**
*(EXCEPT FOR COPD PATIENTS WITH KNOWN OXYGEN SENSIVITY RECORDED ON EPR OR IN PATIENT’S CASE NOTES AND DRUG CARD; KEEP SATURATION AT 88-92% FOR THESE PATIENTS)*
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References

(References are still in “random order” but will be strictly ordered by a publishing professional prior to publication in Thorax)
The following appendices will be available to download on the BTS website. They are shown at the end of this draft version of the Guideline for comment as part of the peer review process.

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Appendix 7  Example of audit tool
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Appendix 9  Teaching aids on emergency oxygen use for nurses.
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<tr>
<td>ABG</td>
<td>Arterial Blood Gases</td>
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<tr>
<td>BTS</td>
<td>British Thoracic Society</td>
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<tr>
<td>CaO₂</td>
<td>Oxygen Content of blood</td>
</tr>
<tr>
<td>CO</td>
<td>Cardiac Output (expressed in L/min)</td>
</tr>
<tr>
<td>CO₂</td>
<td>Carbon Dioxide</td>
</tr>
<tr>
<td>COPD</td>
<td>Chronic Obstructive Pulmonary Disease</td>
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<td>CPAP</td>
<td>Continuous Positive Airway Pressure</td>
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<td>DO₂</td>
<td>Oxygen delivery from the lungs to the tissues (mls/min)</td>
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<td>Di-Phospho Glycerate (affects oxygen carriage by Haemoglobin)</td>
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<td>Fraction of Inspired Oxygen (e.g. 21% oxygen = FIO₂ 0.21)</td>
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<td>General Practitioner</td>
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<tr>
<td>Hb</td>
<td>Haemoglobin (carries oxygen in the bloodstream)</td>
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<tr>
<td>HPV</td>
<td>Hypoxic Pulmonary Vasoconstriction</td>
</tr>
<tr>
<td>ICU</td>
<td>Intensive Care Unit</td>
</tr>
<tr>
<td>kPa</td>
<td>kilo Pascal (Unit of measurement for pressures) 1 kPa = 7.5 mm Hg</td>
</tr>
<tr>
<td>MC Mask</td>
<td>Medium Concentration Mask (also known as simple face mask)</td>
</tr>
<tr>
<td>mm Hg</td>
<td>Millimetres of mercury (unit of measurement for pressures)</td>
</tr>
<tr>
<td>NICE</td>
<td>National Institute for health and Clinical Excellence</td>
</tr>
<tr>
<td>NIPPV</td>
<td>Non-Invasive Positive Pressure Ventilation</td>
</tr>
<tr>
<td>NIV</td>
<td>Non-Invasive Ventilation</td>
</tr>
<tr>
<td>O₂</td>
<td>Oxygen</td>
</tr>
<tr>
<td>PCO₂</td>
<td>Carbon Dioxide tension (partial pressure), in blood or alveolus</td>
</tr>
<tr>
<td>PO₂</td>
<td>Oxygen tension (partial pressure), in blood or alveolus or tissues.</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>Arterial Carbon Dioxide tension (partial pressure)</td>
</tr>
<tr>
<td></td>
<td>Normal Range is 4.5 to 6.0 kPa (34-45 mm Hg)</td>
</tr>
<tr>
<td>PA/O₂</td>
<td>Alveolar Oxygen tension</td>
</tr>
<tr>
<td>PA/O₂</td>
<td>Alveolar Oxygen tension</td>
</tr>
<tr>
<td>PaO₂</td>
<td>Arterial Oxygen tension. Normal ranges shown in table 4.1</td>
</tr>
<tr>
<td>pH</td>
<td>Unit of measurement for acidity of blood</td>
</tr>
<tr>
<td></td>
<td>Normal range 7.35 to 7.45</td>
</tr>
<tr>
<td></td>
<td>lower levels are acidic, higher levels are alkalotic</td>
</tr>
<tr>
<td>PIO₂</td>
<td>Inspired Oxygen tension</td>
</tr>
<tr>
<td>PRN</td>
<td>(on prescriptions) As required, as the need arises. <em>(From Latin, Pro Re Nata)</em></td>
</tr>
<tr>
<td>ROS</td>
<td>Reactive Oxygen Species</td>
</tr>
<tr>
<td>SD</td>
<td>Standard Deviation from the mean</td>
</tr>
<tr>
<td>SIGN</td>
<td>Scottish Intercollegiate Guideline Network</td>
</tr>
<tr>
<td>SaO₂</td>
<td>Arterial Oxygen saturation</td>
</tr>
<tr>
<td>SpO₂</td>
<td>Arterial Oxygen saturation measured by pulse oximetry</td>
</tr>
<tr>
<td>VO₂</td>
<td>Rate of oxygen consumption by the body (normal ~250mls/min)</td>
</tr>
<tr>
<td>V/Q</td>
<td>Ratio of ventilation to perfusion in the lungs</td>
</tr>
<tr>
<td>V/Q mis-match</td>
<td>Discrepancy between ventilation and blood flow in localised areas of the lung, causing decrease in oxygen level and rise in CO₂ level.</td>
</tr>
</tbody>
</table>

**SYMBOLS**

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;</td>
<td>Greater than or above e.g. PaCO₂ &gt; 6.0 kPa</td>
</tr>
<tr>
<td>&lt;</td>
<td>Less than or below e.g. PaO₂ &lt; 8.0 kPa</td>
</tr>
<tr>
<td>≥</td>
<td>Greater than or equal to e.g. Age ≥ 70</td>
</tr>
<tr>
<td>≤</td>
<td>Less than or equal to e.g. pH ≤ 7.35</td>
</tr>
</tbody>
</table>
Section 1

Introduction

1.1 The clinical context
Oxygen is probably the commonest drug to be used in the care of patients who present with medical emergencies. Currently, Ambulance teams and Emergency Department teams are likely to give oxygen to virtually all breathless patients and to a large number of patients with other conditions such as ischaemic heart disease, sepsis or trauma. The North West Ambulance Service serves a population of about 7.25 million people and transports about 700,000 people to hospital Emergency Departments each year. About 34% of these journeys involve oxygen use at some stage. (381) This translates to about two million instances of emergency oxygen use per annum by all UK ambulance services, with further use in patients’ homes, GP surgeries and in hospitals.

1.2 The prescription of oxygen
Most clinicians who deal with medical emergencies will encounter frequent adverse incidents and occasional deaths due to under-use and over-use of oxygen. Despite being a drug, oxygen is frequently given without prescription, even in non-emergency situations. Audits of oxygen use and oxygen prescription have shown consistently poor performance in many countries. (55, 100, 180, 321-324) One major problem is that health care professionals receive conflicting advice about oxygen use from different “experts” during their training and during their clinical careers and many are confused about the entire area of oxygen prescription and use.

1.3 The need for a guideline for emergency oxygen therapy
Despite the frequent use of oxygen, many health care professionals seem to be unaware that medical oxygen is a drug that should be prescribed and there is considerable controversy concerning the benefits and the risks of oxygen treatment in virtually all of the situations where oxygen is used. Unfortunately, this is an area of medicine where there are many strongly-held beliefs but very few randomised controlled trials. The only published UK guideline for emergency oxygen therapy is the North West Oxygen Guideline published in 2001, based on a systematic literature review by the same authors (247, 248). Against this background, the Standards of Care Committee of the British Thoracic Society established a working party in association with 17 other societies and colleges listed in the title page of this document. The objective was to produce an evidence-based and up to date guideline for emergency oxygen use in the UK.

1.4 Intended users of Guideline
This Guideline is intended for use by all health-care professionals who may be involved in emergency oxygen use. This will include ambulance staff, paramedics, doctors, nurses, physiotherapists, pharmacists and all other health-care professionals who may deal with ill or breathless patients.

Specific versions of this Guideline will be available on the BTS website for use in the following situations:

- Hospital use
- Primary care use
- Ambulance use
- Version for use by nursing staff
These abbreviated versions of the Guideline will contain the key recommendations and tables and charts that are relevant to the particular situation. The “mini-guidelines” can be downloaded by Health Care trusts for use on Trust Intranets and to produce paper versions of the Guideline for key staff.

1.5 Areas covered by this Guideline
The Guideline will address the use of oxygen in three main categories of adult patients in the pre-hospital and hospital setting:

- Critically ill or hypoxic patients
- Patients at risk of hypoxia
- Non-hypoxic patients who might benefit from oxygen (e.g. Carbon-monoxide poisoning)

1.6 Areas not covered by this Guideline
- Oxygen use in Paediatrics. The present guideline applies only to subjects aged 16 and above.
- Oxygen use for high altitude activities
- Oxygen use during air travel
- Underwater diving and diving accidents
- Oxygen use in animal experiments
- Oxygen use during Surgery and Anaesthesia or during Endoscopy
- Oxygen use in High Dependency Units
- Oxygen use in Intensive Care Units
- Inter-hospital Level 3 transfers
- Hyperbaric oxygen
- Use of Heliox mixtures
- Use of Nitrous Oxide/oxygen mixtures (e.g. Entonox)
- Respiratory support techniques including intubation, invasive ventilation, non-invasive ventilation, continuous positive airway pressure (CPAP)
- Self-initiated use of oxygen by patients who have home oxygen for any reason (this is covered by Guidelines for home oxygen use)

1.7 Limitations of the Guideline
This Guideline is based on the best available evidence concerning oxygen therapy. However, a guideline can never be a substitute for clinical judgement in individual cases. There may be cases where it is appropriate for clinicians to act outwith the advice contained in this guideline because of the needs of individual patients. Furthermore, the responsibility for the care of individual patients rests with the clinician in charge of the patient's care and the advice offered in this guideline must, of necessity be of a general nature and should not be relied upon as the only source of advice in the treatment of individual patients. In particular, this guideline gives very little advice about the management of the many medical conditions that may cause hypoxia (apart from the specific issue of managing the patients' hypoxia). Readers are referred to other guidelines for advice on the management of specific conditions such as COPD, pneumonia, heart failure etc.
Section 2

Methodology of guideline production

2.1 Establishment of guideline team.

The need for a national guideline for emergency oxygen use was recognised by the British Thoracic Society Standards of Care Committee in 2003. A working party was established with representatives from a wide range of professions involved in oxygen therapy and a lay representative (see full list of guideline group members in section 16). The original group was expanded in 2006 because it became clear that the development and implementation of a national guideline would require input from a very wide range of professional groups. Most development and editing of the guideline took place subsequent to this expansion of the group. The group agreed the remit of this Guideline and a series of key questions as shown below. The group devised a search strategy for relevant studies. A Medline search for “Oxygen” yields over a quarter million “hits”, most of which are not relevant to this guideline. For this reason, the BTS commissioned the Centre for Reviews and Dissemination and Centre for Health Economics at the University of York to undertake bespoke literature searches using the search strategies shown in detail in Section 18 and on the BTS website. Exact details of the search methodology and search results are available on the BTS website at www.brit-thoracic.org.uk/guidelines.html

2.2 Summary of key questions:

Key question 1. Physiology and patho-physiology of oxygen

- What are the dangers of hypoxia/hypoxaemia (i.e. what happens to the human body)?
- What level of hypoxaemia is dangerous to all patients, (even healthy adults)?
- What level of hypoxaemia is dangerous to vulnerable groups (e.g. IHD, Stroke, Elderly)?
  Repeat the above searches with additional key words:
  Elderly, Stroke, Myocardial Infarction, Heart failure, Chronic Obstructive Pulmonary Disease (COPD, Trauma, Renal failure
- Same questions for hypercarbia / hypercapnia
  Search for “Hypercapnia” combined with terms implying a harmful outcome Death / Tissue Injury / Brain damage / Coma
- What level of hypercapnia is dangerous to all patients?
- What level of hypercapnia is dangerous to vulnerable groups (as above)?
- Same questions for Respiratory acidosis
  Search for “Respiratory Acidosis” combined with terms implying a harmful outcome Death / Tissue Injury / Brain damage / Coma
- What level of Respiratory Acidosis is dangerous to all patients?
What level of Respiratory Acidosis is dangerous to vulnerable groups (as above)?

Key questions 2. Clinical aspects of hypoxaemia and oxygen therapy for common medical emergencies

- How to assess hypoxaemia (Clinical, Early Warning Score systems, Oximetry, Arterial and Capillary Blood Gases)
- How to assess hypercarbia/hypercapnia
- Use of oxygen to relieve symptomatic breathlessness
- Use of oxygen in acute COPD
- Use of oxygen in acute Asthma
- Use of oxygen in Pneumonia
- Use of oxygen for Pulmonary Embolus
- Use of oxygen in Trauma
- Use of oxygen in Heart Failure
- Use of oxygen in Myocardial Infarction
- Use of oxygen in Angina
- Use of oxygen for other patients with less common conditions were searched individually: (e.g. Cystic Fibrosis, Muscular Dystrophy, Motor Neurone Disease, Severe Kyphoscoliosis, Anaphylaxis, Hyperventilation)

Key questions 3. Oxygen prescription, oxygen delivery systems and oxygen transport

- Oxygen carriage in transport (practical issues – safety issues)
- Oxygen delivery systems in ambulances
- Prescription of oxygen
- Local hospital guidelines for oxygen use
- Oxygen delivery systems in hospitals
- Advantages / Disadvantages of each delivery system. (Venturi Masks, Simple face masks, Nasal Cannulae, High-flow masks such as non-rebreathing reservoir masks)
- Use of oxygen-driven nebulisers
- Use of “Alert Cards”, alert bracelets or similar hazard warning systems for patients who are known to be at risk of hypercapnia.

2.3 How the evidence was assimilated into the guideline

The initial search strategy was devised at two meetings of the group in 2004 and 2005. The searches in October 2005 yielded 3306 papers, the abstracts of which were checked for relevance by group members. One hundred and eighty four of these abstracts were considered to be relevant to the present Guideline. Full reprints of all relevant papers were obtained. Further references were obtained from the group’s personal literature collections and from the references contained within the papers which the search yielded and by focused literature searches by members.
of the guideline group. The group continued to monitor the literature up to May 2007 for important new publications or very high quality abstracts from International meetings that were thought to be relevant to this Guideline.

The group was divided into three sub-groups to work on specific areas of oxygen use; (Emergency Care, Hospital Care, Oxygen Physiology and Devices). Evidence from the literature searches was graded according to the levels of evidence used in the NICE COPD Guideline (see section 3).

The guideline development group corresponded by e-mail on a regular basis (usually at least once weekly) for most of 2006 to discuss the evidence and to produce an initial outline of the guideline and its key recommendations. The guideline was consolidated over the course of 2006 and early 2007 with each section being led by nominated group members but taking into account feedback from the complete group. Meetings of the full group were held in February 2006, September 2006 and February 2007. Between November 2006 and February 2007, the group had an intensive review and e-mail discussion of one guideline section per week with the objective of achieving a consensus on all of the key points prior to the final meeting of the group in February 2007. The draft guideline was first submitted to the BTS Standards of Care Committee in March 2007. The guideline was further refined by email discussion following comments by this committee. The resulting draft was sent to various national experts for comments (see Acknowledgments Section 17) and was posted on the BTS website for 3 weeks and comments were invited. The document was then sent back to the Standards of Care Committee and the 18 other Societies and Colleges for endorsement.
Section 3

Hierarchy of evidence and grading of recommendations

Levels of evidence and grades of recommendation

Levels of evidence and Grades of Recommendation are based on the levels of evidence used in the NICE COPD Guideline (see below and reference 325). For most of the topics covered by the guideline, there were either no randomised trials or just a handful of observational studies. Members of the group reviewed the evidence for each topic and assigned the most appropriate grading which was usually Grade C evidence (Case control or Cohort studies) or Grade D evidence (Expert opinion or case reports).

Each recommendation has been allocated a grading which directly reflects that hierarchy of evidence upon which it is based.

Please note that the hierarchy of evidence and the recommendation gradings relate to the strength of the literature, not to clinical importance. This is especially important in the field of oxygen therapy where there are very few controlled trials.

Hierarchy of evidence

<table>
<thead>
<tr>
<th>Level of evidence</th>
<th>Type of evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ia</td>
<td>Evidence from systematic reviews or meta-analysis of Randomised Controlled Trials (RCT)</td>
</tr>
<tr>
<td>Ib</td>
<td>Evidence from at least one RCT</td>
</tr>
<tr>
<td>IIa</td>
<td>Evidence from at least one controlled study without randomisation</td>
</tr>
<tr>
<td>IIb</td>
<td>Evidence from at least one other type of quasi experimental study</td>
</tr>
<tr>
<td>III</td>
<td>Evidence from non experimental descriptive studies, such as comparative studies, correlation and case control studies</td>
</tr>
<tr>
<td>IV</td>
<td>Evidence from expert committee reports or opinions and/or clinical experience of respected authorities</td>
</tr>
</tbody>
</table>

Grading of recommendations

<table>
<thead>
<tr>
<th>Level of evidence</th>
<th>Type of evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Based on hierarchy I evidence</td>
</tr>
<tr>
<td>B</td>
<td>Based on hierarchy II evidence or extrapolated from hierarchy I evidence</td>
</tr>
<tr>
<td>C</td>
<td>Based on hierarchy III evidence or extrapolated from hierarchy I or II evidence</td>
</tr>
<tr>
<td>D</td>
<td>Directly based on hierarchy IV evidence or extrapolated from hierarchy I, II or III evidence</td>
</tr>
</tbody>
</table>
Section 4

Normal values and definitions.

- Normal blood levels of oxygen and carbon dioxide.
- Normal oxygen saturation and normal blood pH.
- Definitions of hypoxaemia, hypoxia, hypercapnia, acidosis, respiratory failure.

Oxygen is essential for mammalian life; severe hypoxaemia such as that seen during cardiac arrest, suffocation or drowning will cause loss of consciousness, rapid organ failure, and death. Oxygen is carried in the bloodstream bound to the haemoglobin molecule and delivered to the tissues. Oxygen demand and oxygen delivery increase during exercise and reduce during rest and sleep. Many disease states lead to a reduced oxygen level and it is standard practice for breathless patients to be treated with oxygen. However, there have been few controlled trials comparing different levels of inspired oxygen for patients with any of the common diseases that lead to hypoxaemia. It must also be remembered that oxygen therapy is only one of several strategies that may be used to increase tissue oxygen delivery for critically ill patients (Section 6.11) There are no published trials supporting the use of oxygen to relieve breathlessness in non-hypoxaemic patients and there is evidence from randomised studies that oxygen does not relieve breathlessness compared with air in non-hypoxaemic COPD patients who are breathless following exertion. (374)

4.1 Blood levels of oxygen and carbon dioxide in health and in disease.

The human lung delivers oxygen to the blood and removes carbon dioxide. Several mechanisms exist to regulate breathing in such a way that both gases are maintained within quite a narrow range (section 4.1.1). If the blood oxygen level falls to extremely low levels for even a few minutes (for example during cardiac arrest), tissue hypoxia and cell death will occur, especially in the brain which appears to be the most vulnerable organ during profound hypoxaemia because brain malfunction is the first symptom of hypoxia and brain injury is the commonest long term complication in survivors of cardiac arrests and other episodes of profound hypoxaemia. Sudden exposure to low arterial oxygen saturations below about 78% can cause altered consciousness even in healthy subjects. It is likely that other organs in patients with critical illness or chronic organ damage are vulnerable to the risk of hypoxic tissue injury at oxygen levels above 78%.

Most experts emphasise the importance of keeping the oxygen saturation above 90% for the majority of acutely ill patients. (15, 315, 363, 367) However, the degree of hypoxia that will cause cellular damage is not well established and probably is not an absolute value. Healthy older adults for instance have lower oxygen saturations at rest than younger adults. Patients with chronic lung diseases may tolerate low levels of blood oxygen saturation chronically. However, although chronically hypoxaemic patients may tolerate an abnormally low oxygen saturation at rest when in a clinically stable condition, these resting oxygen levels may not be adequate for tissue oxygenation during acute illness when the tissue oxygen demand may increase (e.g. sepsis, trauma, pneumonia, head injury etc– see section 8).

4.1.1 Normal ranges for oxygen saturation and oxygen and carbon dioxide tensions in the blood
For adults below the age of 70, the two standard deviation range for oxygen saturation is approximately 94-98% but this declines gradually within this age range. (244) The normal range for oxygen tension in the blood in seated adults is shown in Table 4.1. However, the oxygen tension is 0.8 kPa (6 mm Hg) lower in the supine position than in the upright position (232) and most emergency oximetry measurements are made in the supine position.

<table>
<thead>
<tr>
<th>Age</th>
<th>Mean PaO2 (kPa with SD) (mmHg with SD)</th>
<th>Range PaO2 ± 2SD (kPa with SD) (mmHg with SD)</th>
<th>Mean SaO2 % (SD)</th>
<th>SaO2 ± 2SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>18-24</td>
<td>13.4 (0.71) 99.9 (5.3)</td>
<td>11.98 -14.82 89.3-110.5</td>
<td>96.9 (0.4)</td>
<td>96.1-97.7</td>
</tr>
<tr>
<td>25-34</td>
<td>13.4 (0.66) 99.8 (4.9)</td>
<td>12.08 -14.72 90 – 109.6</td>
<td>96.7 (0.7)</td>
<td>95.3-98.1</td>
</tr>
<tr>
<td>35-44</td>
<td>13.18 (1.02) 98.3 (7.6)</td>
<td>11.14 – 15.22 83.1 – 113.5</td>
<td>96.7 (0.6)</td>
<td>95.5-97.97.9</td>
</tr>
<tr>
<td>45-54</td>
<td>13.0 (1.07) 97 (8)</td>
<td>10.86-15.14 81-113</td>
<td>96.5 (1)</td>
<td>94.4 – 98.5</td>
</tr>
<tr>
<td>55-64</td>
<td>12.09 (0.60) 90.2 (4.5)</td>
<td>10.89 – 13.29 81.2 – 99.2</td>
<td>95.09 (0.7)</td>
<td>94.5 – 97.3</td>
</tr>
<tr>
<td>&gt; 64</td>
<td>11.89 (1.43) 88.7 (10.7)</td>
<td>9.02 – 14.76 67.3 – 110.1</td>
<td>95.5 (1.4)</td>
<td>92.7 – 98.3</td>
</tr>
</tbody>
</table>

Table 4.1.
Mean PaO2, SaO2 with range ± SD, (SD in parentheses) in healthy male and females seated non smoking volunteers at sea level (adapted from 244)

The mean oxygen saturation is lower in older people. The mean saturation in seated adults aged over 64 is 95.5%, compared with 96.9% in adults aged 18-24 and the standard deviation is wider with a 2 SD range of 92.7 to 98.3%.

(244 and Table 4.1). The mean oxygen saturation for recumbent healthy male subjects aged 70 and above is 95.3% (SD 1.4) giving a two SD range of 92.5 to 98.1% for males of this age. (231). The mean saturation is 94.8% for recumbent healthy females aged ≥70 (SD 1.7) with two SD Range 91.5 to 98.2. The authors of this study did not observe any age-related decline in SaO2 beyond age 70. Therefore the mean oxygen saturation of approximately 95.0% for recumbent healthy men and women aged ≥ 70 is below the normal range for seated healthy young adults. This variation with age, gender and posture makes it difficult to give a “normal range” that will apply to all adults who might require oxygen therapy.

The reference range for PaCO2 is 4.5-6.0 kPa (34-45 mm Hg) for healthy young adults. Any value of carbon dioxide of more than 6.0 kPa (45mm Hg) should be considered abnormal, but values up to 6.7 kPa (50 mm Hg) may be obtained by breath-holding.

- Normal daytime haemoglobin oxygen saturation is 96-98% in young adults in the seated position but the lower limit falls with age and is 92.5% in supine male subjects aged 70 and above and 91.5% in supine female subjects aged 70 and above. Evidence III

4.1.2 Oxygen saturation of patients with diseases which cause chronic hypoxia
The situation is even more complex for patients with chronic disease such as COPD, lung fibrosis, neuro-muscular disorders or congenital heart disease who may routinely attend outpatient clinics with oxygen saturation well
below 90% even at a time when their disease is stable. A clinician who was not familiar with such a patient might aim to achieve an oxygen saturation that was well above the patient’s usual oxygen saturation level. This may not be sustainable in the long term although many such patients would qualify for long term oxygen therapy. The UK guideline for long term oxygen therapy for patients with COPD (325) recommends a threshold of 7.3 kPa (55 mm Hg) below which most COPD patients will benefit from long-term oxygen therapy (equivalent to a saturation of about 88-89%) and a PaO2 below 8.0 kPa (60 mm Hg) for patients with established cor pulmonale and some other sub-groups. Even for patients with saturation below these levels, continuous oxygen therapy is not required. There appears to be little additional benefit from more than 15 hours per day of oxygen therapy, allowing the patient to spend up to 9 hours per day with a saturation below this range (often substantially below 88%).

- Many patients with chronic lung disease, congenital cyanotic heart disease, or chronic neuromuscular conditions have oxygen saturations substantially below the normal range, even when clinically stable. Evidence III

### 4.1.3 Variation in oxygen saturation during sleep

Healthy subjects in all age groups have greater variation in oxygen saturation when sleeping than whilst awake. A study of 330 people referred to a sleep laboratory with normal results of overnight polysomnography (patients with cranial facial or neurological abnormalities or previously diagnosed pulmonary disease were excluded) showed that de-saturation routinely occurred with an average minimum saturation or “nadir” of 90.4% during the night (SD 3.1%, 2 SD range 84.2%-96.6%). (243) The mean overnight oxygen saturation “nadir” was 89.3% for subjects aged above 60 years (SD 2.8). (243). In this study, subjects aged 20-30 years spent 10% of the night with oxygen saturation levels below 94.8% and half the night below 96.3% and those aged over 60 spent 10% of the night below 92.8% and half the night below 95.1%. Furthermore, the authors of this study excluded obese patients with any features of sleep apnoea or hypopnoea because these patients are known to desaturate to very low levels during sleep (often below 70%). The variation in oxygen saturation during normal sleep is exaggerated by alcohol and by sedative drugs. This makes it difficult to evaluate a “spot reading” of oxygen saturation on a sleeping patient. It is suggested that the oxygen saturation measurements of sleeping patients should be interpreted with caution and ideally observed for a few minutes to see if the patient has got sustained hypoxaemia or just a transient normal “nocturnal dip.

- All subjects have transient dips in oxygen saturation at night with a mean nadir of 90.4% (2 SD range 84.2%-96.6%) in healthy subjects in all age groups. Evidence III See recommendation 16

### 4.2 Definitions of Hypoxaemia, Hypoxia, Type 1 Respiratory failure and Hyperoxia

**Hypoxaemia** refers to low oxygen tension or partial pressure of oxygen in the blood. For practical reasons, hypoxaemia can also be measured in relation to oxyhaemoglobin saturation. In adults the normal range is influenced by age and co-morbidity and the normal ranges for healthy adults are given in section 4.1.1. The precise level at which a patients becomes hypoxaemic is debatable. One could argue that any saturation below the lower limit of normal constitutes hypoxaemia (i.e. any saturation below 96% in awake young adults or below 92.5% in elderly subjects) and such a patient could be described as “hypoxaemic”. Various authors have defined hypoxaemia as an oxygen saturation; i) below 94%; ii) below 92% ; iii) below 90% ; iv) a partial pressure of oxygen below 60 mm Hg or 8 kPa. (43, 44, 46, 55) The majority of authors who have studied this area have
defined hypoxaemia as a PaO2 below 60 mm Hg (8 kPa) or an oxygen saturation below 90%. There is no known risk of hypoxic tissue injury above this level and many Critical Care Guidelines set 90% as the minimum below which oxygen saturation should not be allowed to fall. (363, 367)

“Type 1 Respiratory Failure” is most widely defined as an arterial oxygen tension below 8 kPa or 60 mm Hg (equivalent to an oxygen saturation of approximately 90%) with a normal or low blood carbon dioxide level. (329)

The term, hypoxia is less specific and refers to low oxygen. Unless otherwise specified, the term "hypoxia" is used throughout this guideline to mean low oxygen content in the blood. Hypoxia may be grouped under 4 headings:

**Hypoxaemic hypoxia** is present when the oxygen content in the blood is low due to reduced partial pressure of oxygen. This occurs naturally at altitude and it occurs in many diseases such as emphysema which impair the efficiency of gas exchange in the lungs.

**Stagnant hypoxia** is a low level of oxygen in the blood due to poor blood circulation. This condition may occur in the extremities if a person is exposed to cold temperatures for prolonged periods of time and it is the cause of gangrene in tissue that is deprived of blood in severe peripheral vascular disease. Stagnant hypoxia may occur in low cardiac output states.

**Anaemic Hypoxia** is present in all cases of anaemia. A patient with anaemia has a low level of haemoglobin available for oxygen transport. This patient has hypoxia (low blood oxygen content) even if the blood oxygen tension (PaO2) and the oxygen saturation measured by oximetry (SpO2) are entirely normal. Such patients are hypoxic, but not technically hypoxaemic. However, it is rare for a practicing clinician to refer to an anaemic patient as being “hypoxic” despite the reduced blood oxygen content. Carbon monoxide poisoning may also produce a form of anaemic hypoxia by impairing the ability of haemoglobin to bind oxygen, thereby reducing oxygen carrying capacity.

**Histotoxic hypoxia** is an inability of the tissues to use oxygen due to interruption of normal cellular metabolism. The best known example of this occurs during cyanide poisoning, which impairs cytochrome function. It is increasingly thought that mitochondrial dysfunction may lead to decreased oxygen utilisation in sepsis despite adequate oxygen delivery. This also has been termed 'cytopathic dysoxia' (371).

**Hyperoxia and hyperoxaemia** are the counterparts to the above terms and in this guideline refer to high oxygen content in the blood and high oxygen tension in the blood, respectively. As stated above, for practical purposes, oxygen tension in the blood is often measured as oxyhaemoglobin saturation. Furthermore, this guideline is centred on providing target saturations for various conditions, however, it should be noted that above a PaO2 of approximately 16kPa (120 mm Hg), the oxyhaemoglobin saturation will obviously not change from 100%, yet the effects of further increases in PaO2 may be important in certain conditions, such as COPD. This is discussed in further detail in Section 6.

**4.3 Definition of hypercapnia and Type 2 Respiratory Failure**
Hypercapnia is present when the blood carbon dioxide level is above the normal range of 4.5 to 6.0 kPa (34-45 mm Hg) and patients with hypercapnia are said to have Type 2 Respiratory Failure, even if the oxygen saturation is in the normal range. (329)

Hypercapnia is most commonly caused by: i) a reduced level of ventilation; ii) VQ mismatch within the lungs; iii) a combination of these factors. The most common cause of hypercapnia is COPD where both factors usually co-exist, but the predominant factor is ventilation-perfusion mis-match (12,25,28,59,83). Many patients with severe COPD have a chronically elevated PaCO2 in addition to a low blood oxygen level. During exacerbations, the oxygen level tends to fall and the carbon dioxide level tends to rise, especially if high doses of oxygen are administered. Depressed respiration for any reason will also give rise to hypercapnia. Examples are opiate overdoses, obesity with hypoventilation and neuro-muscular disorders affecting the muscles of respiration.

4.4 Definition of acidosis (respiratory acidosis and metabolic acidosis)

Acidosis: The normal pH range of the blood in humans is between 7.35 and 7.45 units. Acidosis is defined as a pH below 7.35 and alkalosis is defined as a pH above 7.45. Acidosis can be caused by respiratory or metabolic disorders.

Respiratory acidosis: Carbon dioxide (CO2) can combine with water (H2O) to form carbonic acid (H2CO3) in the blood, which in turn dissociates to bicarbonate (HCO3-) and a hydrogen ion (H+). Acute respiratory acidosis occurs if the pH of the blood falls below 7.35 in the presence of an elevated carbon dioxide level.

If respiratory acidosis has been present for more than a few hours, the kidney retains bicarbonate to buffer the acidity of the blood and, over hours to days, this may be sufficient to produce a normal pH. This situation (high PaCO2 with high bicarbonate and normal pH) is known as "compensated respiratory acidosis". This situation is common in patients with chronic severe but stable COPD, but they may have an additional acute rise in PaCO2 during an acute exacerbation giving rise to "acute on chronic" respiratory acidosis despite their high bicarbonate level. This happens because the bicarbonate level was equilibrated with the previous CO2 level and is insufficient to buffer the sudden further increase in CO2 level that may occur during an exacerbation of COPD. Plant and colleagues have shown that about 20% of patients with acute exacerbations of COPD requiring hospital admission have got respiratory acidosis (48).

Metabolic acidosis: This can be caused by failure to excrete acid produced by the body's normal metabolic processes (e.g. during renal failure) or by increased production of acid from abnormal metabolic conditions such as diabetic ketoacidosis. Alternatively, it may result from direct loss of bicarbonate from the kidney or gut (e.g. chronic diarrhoea). In all forms of metabolic acidosis there is a low blood bicarbonate either due to loss of bicarbonate or due to buffering of excess acid by bicarbonate, which is excreted as CO2. A common cause of metabolic acidosis is lactic acidosis caused by tissue hypoxia. This may result from decreased oxygen delivery such as occurs in hypoxaemia or low cardiac output states or conditions such as sepsis where oxygen consumption is impaired in the face of adequate oxygen delivery. In health, metabolic acidosis will occur at peak exercise, where oxygen delivery is insufficient to meet demand.
Section 5  
General blood gas physiology

Full understanding of blood gas physiology in the body requires a detailed understanding of the anatomy, physiology and biochemistry of respiration and gas exchange. It is recognised that most readers of this guideline may not had have full training in all of these specialties so this physiology section contains a brief overview of basic principles for the non-specialist reader (Section 5) followed by a more detailed overview of the pathophysiology of oxygen for the expert reader. (Section 6) The important recommendations deriving from these considerations are in sub-section 6.9

5.1 Oxygen physiology

Oxygen is the main “fuel” of the cells in mammalian bodies and it is essential for humans to maintain a safe level of oxygen in the bloodstream. Most of the oxygen carried in the blood is bound to an oxygen-carrying protein in red blood cells called haemoglobin. Oxygen itself does not dissolve easily in blood so only a small amount of oxygen is carried dissolved in the bloodstream. As there is a fixed amount of haemoglobin circulating in the blood, the amount of oxygen carried in the blood is often expressed in terms of how saturated with oxygen the circulating haemoglobin is. This is what is meant by “oxygen saturation level”. If this is measured directly from an arterial blood sample, it is called the SaO2. If the measurement is calculated from a pulse oximeter, it is called the SpO2. Alternatively, one can measure the oxygen tension of the blood, known as the “partial pressure of oxygen” in the blood. This measurement can be expressed in kilo Pascals (normal range 12.0-14.6 kPa) or in millimetres of mercury (normal range 90-110 mm Hg for young adults). (244)

The normal arterial oxygen level in healthy adults is maintained within a narrow range of about 92-98% as discussed in section 4.1 above. This means that almost all of the oxygen carrying capacity of haemoglobin in the blood is utilised when the saturation is in the normal range. Therefore, giving supplementary oxygen to a healthy young person will elevate the saturation level only slightly from about 97% to 99% or a maximum of 100%, thus producing only a very small increase in amount of oxygen made available to the tissues.

Sudden exposure to low arterial oxygen saturations below about 78% can cause altered consciousness even in healthy subjects. The brain is the most sensitive organ to the adverse effects of hypoxia, but it is likely that other organs in patients with critical illness or chronic organ damage are vulnerable to the risk of hypoxic tissue injury at oxygen levels above 78%. Most experts emphasise the importance of keeping the oxygen saturation above 90% for the majority of acutely ill patients. (15, 315, 363, 367) The present guideline suggests a desirable target saturation range of 94-98% in those aged below 70 and 92-98% in those aged 70 and above. This allows a further margin of safety above the 90% threshold which is mentioned above. The narrower target range in younger patients is to ensure prompt assessment if a patient falls outside the age-specific normal range, not due to greater vulnerability to hypoxia. Young adults actually tolerate hypoxia better than older people who may have impaired circulation or pre-existing organ dysfunction.
Oxygen passes from inspired air in the lungs into the bloodstream and is delivered to tissues. If oxygen levels fall in the blood, this is sensed by receptors in the carotid body (connected to the carotid artery in the neck) and ventilation is stimulated to increase the concentration of oxygen in the lung and therefore the blood. The lung has the ability to divert blood flow away from areas which are poorly ventilated, so that blood returning from the body can be replenished with oxygen and can also clear carbon dioxide. This occurs through a process called ‘hypoxic vasoconstriction’, whereby localised low oxygen levels in the lung airspaces cause constriction of feeding blood vessels, therefore diverting blood to areas of the lung with more normal oxygen levels.

If the oxygen-carrying capacity of the blood is low, for example in anaemia, this is detected by the kidneys, which produce a hormone, erythropoietin, to stimulate red blood cell production. As one of the goals of the circulation is to deliver oxygen to the tissues of the body, the heart also responds to low oxygen levels by increasing its output, so increasing ‘oxygen delivery’.

Hypoxia, low oxygen blood content, can be caused by a number of mechanisms. The most common form of hypoxia occurs when there is sufficient oxygen-carrying capacity (in patients with a normal level of haemoglobin), but insufficient oxygen taken up in the lungs. This can be the result of poor aeration of areas of lung or due to abnormalities of gas exchange within the lung during serious illnesses such as pneumonia. This form of hypoxia is the easiest to treat with oxygen therapy. Oxygen therapy is less effective in other causes of hypoxaemia including anaemia, where there is low carrying capacity, or where the carrying capacity of haemoglobin has been reduced by a toxic substance, because oxygen availability is not the limiting feature in these conditions. For example carbon monoxide blocks oxygen binding to haemoglobin despite having a normal level of oxygen in the lungs and in the blood.

5.2 Carbon dioxide physiology

Carbon dioxide is a product of the body’s metabolism. It is cleared from the body by being transferred from the blood stream into the alveoli in the lungs and then exhaled from the lungs. In a similar way to oxygen, carbon dioxide levels in the blood are controlled by chemical sensors (both in the carotid body and brainstem).

Carbon dioxide is highly soluble in the blood and is carried in three forms: bicarbonate (70%), dissolved carbon dioxide (10%) and bound to haemoglobin (20%). As carbon dioxide carriage is not limited by a carrier molecule such as haemoglobin, it is not expressed as a saturation. Because its carriage is proportional to the partial pressure (gas tension) of carbon dioxide in the blood, carbon dioxide carriage is usually ‘expressed’ in terms of its partial pressure. The normal range is 4.5 to 6.0 kPa or 34-45 mm Hg.

Increased levels of carbon dioxide will stimulate ventilation, thus increasing clearance from the lungs and therefore from the blood stream. Hypercapnia will occur when there is decreased ventilation for any reason. Safe elimination of carbon dioxide is as important to the body as the intake of oxygen.

Too little oxygen can give rise to organ failure but too much oxygen can also be harmful in some situations especially to some vulnerable patients with Chronic Obstructive Pulmonary Disease (COPD), chest wall deformities or muscle weakness. About a quarter of patients with acute flare-ups of COPD are at risk of carbon dioxide retention if they are given an excessively high dose of oxygen. If high doses of oxygen are given to these
patients, the oxygen level in the blood will rise but the level of carbon dioxide will also rise and this can cause organ dysfunction and when severe, coma. In the past, it was thought that the main problem was that these patients were dependent on the stimulus of a low blood oxygen level, called “hypoxic drive”, to stimulate breathing. It was thought that giving oxygen would cause a rise in the carbon dioxide level by simply reducing the desire to breathe due to ”lack of hypoxic drive”. It is now known that the mechanisms for carbon dioxide retention in some patients are much more complex than this simple model suggested. Much of the rise in carbon dioxide which occurs during high-dose oxygen therapy is due to unequal distribution of blood flow and gas flow in the lungs. This can be avoided by giving controlled lower-dose oxygen therapy to vulnerable patients (see Table 3).

**5.3 Concept of target oxygen saturation ranges**

One might ask why one should not aim for an oxygen saturation of 100% (hyperoxaemia) in all acutely ill patients (and some clinicians took this view in the past). This policy would clearly be risky for vulnerable patients with COPD and chest wall problems but it could also harm other patients in a variety of ways. The more controversial risks of hyperoxaemia include coronary and cerebral vasoconstriction and decreased cardiac output. Although, these physiological effects are well documented, their significance in clinical practice is almost unknown due to a lack of clinical trials of oxygen therapy.

High oxygen concentrations lead to an increase in reactive oxygen species, which may cause tissue damage and may be responsible for some of the detrimental effects observed with high flow oxygen in myocardial infarction and stroke. It is recognised that very high inhaled oxygen levels can give rise to partial collapse of some lung units, a condition known as absorption atelectasis. There is also the potential concern that a high oxygen saturation produced by excessive oxygen therapy could mask a major deterioration in the patient’s clinical condition causing dangerous delays in treatment. An example of this is a patient who has taken an opiate overdose which has produced respiratory depression and the patient is under-breathing. If the patient is given excessive oxygen therapy high or normal oxygen saturations may be recorded at a time when the carbon dioxide levels are dangerously high. The high oxygen saturation could mask the real situation and give the health professionals a false sense of confidence.

As eluded to above, high carbon dioxide levels can be dangerous. In acute circumstances, where carbon dioxide levels have risen rapidly, the kidneys are unable to compensate for the consequent increased acid load. There are good data to show that the lower the pH of the blood, the higher the risk of intubation or death in patients with exacerbations of COPD. (48)

The purpose of oxygen therapy is to increase oxygen delivery to tissues, not just to increase oxygen carried by the blood. Therefore, it must be remembered that there may be other physiological disturbances that need correcting to increase oxygen delivery, such as low cardiac output and severe anaemia. For example, improving these factors will improve oxygen delivery much more than administering oxygen to a patient with a saturation of 90%, which, at most, will produce a 10% rise in delivery. In addition to optimising oxygen delivery from the lungs to the tissues, it is important also to treat problems that might impair delivery of oxygen to the lungs themselves, such as upper airway obstruction, bronchoconstriction and pulmonary oedema. (Remember the ”ABC” of resuscitation – Airway, Breathing, Circulation).
There is uncertainty about defining the ideal target saturation and this is one of the core debates in oxygen therapy. This uncertainty is largely due to a lack of evidence from clinical trials. In some specific disease areas, such as COPD, there are good data to inform the ideal target saturation and these will be covered in Sections 8 and 9. In the general population, without a specific indication for running high or low saturations, historically there has been a tendency to apply oxygen therapy even when saturations are in the ‘normal’ range. There are no data to support this practice in common conditions such as ischaemic heart disease or stroke, and indeed some studies show harm. It should be reassuring that supraphysiological levels of oxygen delivery are not required in critical illness, unless specifically indicated, e.g. carbon monoxide poisoning. The consensus amongst the members of the guideline group is that one should aim for a normal oxygen saturation range of 94-98% for those aged below 70 and 92-98% for those aged 70 years and above for acutely ill patients except those at risk of hypercapnic respiratory failure (see Recommendation 1 which is also based on the more detailed review of oxygen physiology in section 6.5.1 of this Guideline). Other recommendations (2-5) which have a physiological basis are shown in section 6.9.1.
Section 6

Advanced blood gas physiology and pathophysiology

Many of the issues discussed in Section 6 are of a technical nature and may not be easily comprehensible to the general reader. However, the recommendations 1, 2, 3, 4, 5 and 6 in this section all follow logically from the brief overview of this topic in section 5.

The neuro-cardiopulmonary axis is designed to optimise global oxygen delivery and CO2 clearance and the local tissue vascular beds are responsible for the distribution of blood flow.

Oxygen delivery (DO2) is expressed by the equation:

\[ \text{DO2} = \text{CaO2} \times Q \]

where \( \text{CaO2} \) is the oxygen content of the arterial blood and \( Q \) is the cardiac output.

\( \text{CaO2} \) is the sum of oxygen dissolved in the blood and the amount of oxygen carried by haemoglobin. The solubility of oxygen in the blood is very low and therefore \( \text{CaO2} \) is largely determined by the amount of oxygen bound to haemoglobin. The relationship between haemoglobin oxygen saturation and arterial oxygen tension is shown in figure 6a and table 6a. In health, \( \text{CaO2} \) is dependent on arterial PO2, haematocrit and the Bohr effect. Therefore there is not an exact relationship between arterial oxygen saturation and oxygen tension but Table 6a gives approximate equivalents.

**Figure 6.a Oxygen dissociation curve with the Bohr Effect**
<table>
<thead>
<tr>
<th>Arterial oxygen saturations %</th>
<th>56</th>
<th>70</th>
<th>80</th>
<th>88-89</th>
<th>90</th>
<th>93</th>
<th>95</th>
<th>100</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial oxygen tension kPa</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7.3</td>
<td>8</td>
<td>10</td>
<td>13</td>
<td>16 and above</td>
</tr>
<tr>
<td>Arterial oxygen tension mm Hg</td>
<td>30</td>
<td>37</td>
<td>45</td>
<td>55</td>
<td>60</td>
<td>67</td>
<td>90</td>
<td>120</td>
</tr>
</tbody>
</table>

Table 6a. Approximate relationship between arterial blood saturations and arterial oxygen tensions

6.1 Regulation of blood oxygen content (CaO2)

Figure 6b shows the level of oxygen and carbon dioxide in the venous system and pulmonary artery (shown in blue), in the alveolus and room air and in the pulmonary venous circulation which leads directly to the arterial circulation. It can be seen that the oxygen content of the blood rises markedly from a low level in the venous system (about 6 kPa or 45 mm Hg) to an arterial level of about 13 kPa (98 mm Hg) which is quite close to the Alveolar level of 16 kPa (120 mm Hg). However, the gradient of CO2 is much more gradual, falling from about 7 kPa (52 mm Hg) in the venous system and pulmonary artery to about 5 kPa (37 mm Hg) in the pulmonary vein and in the arterial system.

Figure 6.b Alveolar-capillary unit

Inspired air

6.1.1 Arterial PO2 (PaO2)
The pulmonary vasculature maximises PaO2 by ensuring that the well-ventilated areas of the lung receive most of the pulmonary blood-flow, a process called ventilation/perfusion (V/Q) matching. This is largely achieved through
a process called hypoxic pulmonary vasoconstriction (HPV)[339]. The pulmonary circulation is unique in this regard compared with all the other vascular beds in the body, which dilate in response to hypoxia. In poorly-ventilated areas of lung, the pre-capillary pulmonary arterioles constrict in response to sensing low alveolar PO2 (PA\textsubscript{Alv}O\textsubscript{2}). This is a compensating process and despite it, some de-oxygenated blood may still leave poorly ventilated alveolar-capillary units (Figure 6.b). Deoxygenated blood leaving poorly-ventilated alveolar-capillary units cannot be compensated for by mixing with blood from well-ventilated units, as the relationship between PaO\textsubscript{2} and CaO\textsubscript{2} is not linear. This physiological phenomenon is often not fully appreciated and therefore is worth a theoretical worked example:

**EXAMPLE:**

- Before oxygen therapy assume 50% of pulmonary flow is passing through an area of low V/Q and that the pulmonary venous oxyhaemoglobin saturation (SpvO\textsubscript{2}) from this compartment is 80% (i.e., just above mixed venous SO\textsubscript{2}). The other 50% is passing through an area of matched ventilation and perfusion, resulting in a SpvO\textsubscript{2} of 97%. The final mixed SpvO\textsubscript{2} will be 88.5%.
- Following maximal oxygen therapy, assuming no change in flow as a result of release of HPV, SpvO\textsubscript{2} from the low V/Q compartment rises to 85% and SpvO\textsubscript{2} from the matched compartment rises to a maximum of 100%. The resulting mixed SpvO\textsubscript{2} will now only be 92.5%. This has occurred because fully saturated blood cannot increase its oxygen content beyond full saturation despite an increase in PO\textsubscript{2}, apart from the minimal contribution from dissolved oxygen, i.e., the relationship between PO\textsubscript{2} and oxyhaemoglobin saturation/blood oxygen content is not linear.

A much less studied phenomenon that regulates V/Q matching is hypoxic bronchodilation. This effect increases ventilation to poorly ventilated areas of lung.

If PaO\textsubscript{2} falls, the peripheral chemo receptors in the carotid body drive an increase in ventilation to increase PaO\textsubscript{2}[340]. This will not increase PaO\textsubscript{2} leaving already well-ventilated units, but will increase PaO\textsubscript{2} leaving less well-ventilated alveolar units by increasing PA\textsubscript{Alv}O\textsubscript{2} in these units. Although the ventilatory response to the arterial oxygen saturation (SaO\textsubscript{2}) and therefore CaO\textsubscript{2}, and not PaO\textsubscript{2}, is linear (Figure 6.c), the carotid body senses PaO\textsubscript{2} and not CaO\textsubscript{2}. This prevents excessive ventilation in response to anaemia, which would be ineffective in increasing CaO\textsubscript{2}. They are able to do this because the very high ratio of DO\textsubscript{2} to O\textsubscript{2} consumption of the carotid body: this means that the tissue PO\textsubscript{2} in the carotid body continues to reflect PaO\textsubscript{2} and will not fall, resulting in tissue hypoxia even in the presence of anaemic hypoxia. (341, 342)
6.1.2 Haematocrit
Erythropoiesis is controlled by a negative feedback system involving erythropoietin. By contrast with the carotid bodies, the peritubular cells in the kidney are well-suited to sensing oxygen delivery, as oxygen extraction is relatively high compared with oxygen delivery. (343,344) Although oxygen delivery to the kidneys as a whole organ is high due to high renal blood flow, DO2 is reduced to the renal medulla, as oxygen can pass from arterioles to the post capillary venous system by shunt diffusion due to the parallel organisation of arterial and venous systems. (345) Consequently, the peritubular cellular PO2 is low and falls with further reductions in DO2 either as a result of hypoxaemia or haematocrit.

6.1.3 The Bohr Effect
The oxygen carrying capacity of haemoglobin is regulated in response to other metabolic factors in order to increase the efficiency of oxygen pick-up and delivery. (346) Acidosis and hypercapnia shift the oxygen dissociation curve to the right (Figure 6.a), thus favouring the dissociation of oxygen from haemoglobin in metabolically active tissues. The converse would hold true for the lungs, where lower carbon dioxide levels favour oxygen loading of haemoglobin. Chronic hypoxaemia increases 2,3-DPG in erythrocytes, shifting the dissociation curve to the left and therefore increasing oxygen pick-up in the lungs.

6.1.4 Regulation of DO2 (oxygen delivery from the lungs to the tissues)
Acutely, the cardiovascular effects of hypoxaemia will tend to counter the impact of lower CaO2 on DO2 by increasing cardiac output through increased heart rate and myocardial contractility and by decreasing afterload by reducing systemic vascular resistance. (347,348) Anaemic hypoxia is sensed in the aortic body, presumably due to lower perfusion relative to oxygen consumption, unlike the carotid body: consequently, the aortic body can act as a sensor and controller of oxygen delivery. (341)
At local tissue level, oxygen delivery can be adjusted to changes in local oxygen consumption. For example, exercising skeletal muscle receives a greater proportion of total cardiac output than resting skeletal muscle. In part, this relates to hypoxaemia recruiting a larger proportion of the capillary bed by the relaxation of pericytes, and also through arteriolar vasodilatation. (349)

6.2 The Pathophysiology of hypoxaemia and hyperoxia

Hypoxia may result from a number of different diseases discussed in section 8 of this Guideline. In each case, one or more of the following pathophysiological mechanisms may apply:

6.2.1 Hypoxaemic hypoxia (see definition in section 4.1.2)

Hypoxaemic hypoxia in blood leaving an alveolar-capillary unit in the lung may be induced by alveolar hypoxia or incomplete gas exchange producing an increased alveolar-arterial (A-a) gradient of PO₂. The alveolar gas equation calculates the oxygen level in the alveolus using the following formula:

$$P_{Alv}O_2 \approx PIO_2 - PA_{CO2}/RQ$$

($p_{Alv}O_2$ and $p_{Alv}CO_2$ represent alveolar levels of O₂ and CO₂ and RQ the Respiratory Quotient or ratio of CO₂ production to O₂ consumption; and PIO₂ is FIO₂x(Barometric pressure {100 kPa, 750 mm Hg} – water vapour pressure {$\sim$6 kPa, 45 mm Hg} ).

Considering this equation, alveolar hypoxia can be induced by decreased inspired PO₂ (PIO₂) or increased alveolar PCO₂ (PAv/CO2). If an alveolar-capillary unit is relatively under ventilated for its degree of perfusion (low V/Q ratio), PAv/CO₂ will rise due to inadequate clearance and thus PAv/O₂ will fall. In diseases that cause global hypoventilation, such as respiratory muscle weakness, effectively all areas of lung have low V/Q ratios and this explains the hypercapnia and hypoxaemia associated with these conditions.

An extreme form of low V/Q pathophysiology occurs in intrapulmonary and extra pulmonary shunt, where no gas exchange occurs at all. An example of intrapulmonary shunt would be where the airway to a lung segment was obstructed by mucus, creating an area of lung tissue that was perfused but not ventilated, thus acting as a right-to-left shunt. An example of extra pulmonary shunt would be an ventricular septal defect with right-to-left shunting in Eisenmenger’s syndrome.

In health, and at rest, oxygen has equilibrated across the alveolar-capillary membrane one third of the way along the length of the capillary. With increased thickness of this membrane, as in fibrotic lung disease, equilibration may take longer and a gradient between PAv/O₂ and PaO₂ may persist by the end of the capillary (increased A-a gradient). This is exacerbated during exercise, when capillary transit time decreases. Conversely, despite a prolonged transit time in very low cardiac output states, equilibration may still not take place as the oxygen content in mixed venous blood returning to the lung is so greatly reduced.
6.2.2 Other mechanisms of hypoxia (see definitions in section 4.1.2)
Anaemia and carbon monoxide poisoning may result in “anaemic hypoxia” by reducing oxygen carrying capacity. A low cardiac output state will reduce oxygen delivery even in the absence of hypoxaemia. Tissue hypoxia may develop in these circumstances and this is often termed “stagnant hypoxia”.

6.2.3 Hyperoxia
Hyperoxia can be caused by hypoxaemia and polycythaemia. Considering again the alveolar gas equation in the previous section, hypoxaemia can only exist in the presence of high inspired PO2 or low PaCO2 (resulting from hyperventilation). The term “hyperoxia” could technically be used to describe a patient with polycythaemia without hypoxaemia but most clinicians use the term only to describe situations in which the blood oxygen tension is elevated.

6.3 Physiology of CO2

6.3.1 Normal carbon dioxide homeostasis
Carbon dioxide is principally carried in the blood in three forms, CO2, bicarbonate and as a carbamino compound. In the normal physiological range of 4.5 to 6.0 kPa (34–45 mm Hg), the relationship between PaCO2 and CCO2 (CO2 content) can be considered linear.

Figure 6d Total CO2 dissociation curve

6.3.2 Regulation of carbon dioxide

Regulation of PaCO2
PaCO2 is sensed at the peripheral[340](340) and central chemo receptors (in the medulla oblongata) by its effect on intracellular pH. (351) Consequently, the regulation of PaCO2 is intimately related to pH homeostasis (Figure 6.e).
It is often not appreciated how V/Q matching relates to PaCO\(_2\). As discussed in Section 6.2.1, alveolar-capillary units with a low V/Q ratio have increased PAh\(\text{CO}_2\). Because of the high solubility and diffusibility of CO\(_2\), there is little A-a gradient for CO\(_2\) at the end of the capillary, therefore, blood leaving low V/Q alveolar-capillary units has high PCO\(_2\).

As described above, areas of low V/Q are usually minimised through hypoxic pulmonary vasoconstriction. It is also thought that high PCO\(_2\) can cause pulmonary vasoconstriction, adding to the homeostatic mechanisms of the lung, matching perfusion to ventilation. (352, 353) As the relationship between PCO\(_2\) and CO\(_2\) dissolved in the blood is approximately linear (unlike oxygen), blood does not become saturated with CO\(_2\). Pulmonary venous PCO\(_2\) from low V/Q areas can be partially balanced by low pulmonary venous PCO\(_2\) from high V/Q areas. Therefore, by increasing overall alveolar ventilation, the cardiopulmonary system is able to correct abnormally high PaCO\(_2\) levels by increasing ventilation to both low and high V/Q areas.

As with the carriage of O\(_2\) (Bohr Effect), there is a reciprocal relationship between PO\(_2\) and CO\(_2\) carriage. This is known the Haldane effect. (346) Deoxygenated haemoglobin has a higher CO\(_2\) buffering capacity than oxygenated haemoglobin. This favours CO\(_2\) pick-up in the systemic venous circulation and CO\(_2\) offloading in the lungs.

Acutely, CO\(_2\) acts as a sympathomimetic on the heart: it increases heart rate and stroke volume, increasing cardiac output. Peripherally, it causes vasodilation, reducing systemic vascular resistance. Locally, CO\(_2\) acts a vasodilator, thus diverting blood flow to tissues with high metabolic demand. The resulting physical signs of hypercapnia are described in section 3.2.1

### 6.4 The Pathophysiology of hypercapnia and hypocapnia

**Mechanisms of hypercapnia and hypocapnia**

The mechanisms of hypercapnia are simpler than hypoxaemia and there are four possible causes. (330)
1. Increased concentration of carbon dioxide in the inspired gas. This iatrogenic cause of hypercapnia is uncommon but should be excluded at the outset in any patient unexpectedly found to be hypercapnic when breathing from, or being ventilated by, external equipment. The severity of hypercapnia due to rebreathing is limited by the rate at which the PCO2 can increase; (no more than 0.4 to 0.8 kPa per minute, 3 to 6 mm Hg per minute).

2. Increased carbon dioxide production. Again this is likely only to cause hypercapnia if the minute ventilation is fixed by artificial means and if carbon dioxide production is increased for instance due to sepsis or increased work of breathing.

3. Hypoventilation. An inadequate pulmonary minute ventilation is by far the commonest cause of hypercapnia. In clinical practice, COPD is the commonest disease to cause hypercapnia; the problem is secondary to alveolar hypoventilation rather than a reduced minute ventilation per se. Patients adopt a rapid shallow pattern of breathing during an acute exacerbation of COPD with the result that the ratio of dead space to tidal volume is increased with more ventilation therefore being "wasted". A rapid shallow pattern of breathing results in a bigger proportion of each breath being wasted because of the need to ventilate the anatomical dead space. Furthermore, during acute COPD exacerbations, ventilation perfusion mismatch may lead to an increase in physiological dead space, exacerbating the problem further. It is important to note that this commonly occurs in the context of an apparent overall increase in minute ventilation. Alveolar hypoventilation due to a reduction in minute ventilation is seen following medullary respiratory centre depression by drugs, obstruction of a major airway or restriction of the lungs or chest-wall or by respiratory muscle weakness, head injury, intra-cerebral haemorrhage or opioid narcosis.

4. Increased dead space. Again this would be most common in patients breathing through artificial apparatus which has been incorrectly configured. It can also be due to any cause of ventilation perfusion mismatch in which the normal response to hypoxaemia (i.e. to increase ventilation) is compromised because of lung disease. It is important to note therefore that hypercapnia sometimes may be seen in conditions more usually associated with hypocapnia (e.g. pulmonary embolus, pneumonia, etc) when it occurs in patients with lung disease and an increased physiological dead space. Although alveolar hypoventilation is the commonest cause of hypercapnia, it is important to consider the other potential causes, particularly when patients are receiving assisted ventilation and an artificial breathing circuit is used.

Hypoventilation may be physiological, for example in the face of a metabolic alkalosis. Pathological hypoventilation will occur either when the respiratory muscles are unable to ventilate the lungs sufficiently because they are pathologically weak or they are unable to overcome abnormal lung mechanics, such as during an exacerbation of COPD. Reduced respiratory drive caused by drugs with sedative properties or by neurological injury, will also produce hypoventilation

Using the same physiological principles, but in reverse, hyperventilation for any reason will produce hypocapnia. This may occur during pure hyperventilation during an anxiety attack or in response to the physiological hyperventilation that occurs in acute lung disease in an attempt to maintain normal blood oxygen levels.
6.5 Effects and risks of hypoxaemia and recommended minimum oxygen saturation

As this guideline is addressing emergency oxygen therapy, this section will only focus on the effects and risks of acute hypoxaemia. Where it is relevant to discuss issues concerning emergency treatment of acute hypoxaemia in already chronically hypoxaemic patients, these will be discussed in context in Section 8.

The effects and risks of hypoxia are summarised in Table 6b. Severe hypoxia may lead to brain damage and death. In general, many of the physiological effects of hypoxia are mediated by low PaO₂, irrespective of oxygen content. For example even when the total blood oxygen content is normal in the presence of polycythaemia, hypoxaemia will still exert a physiological effect, such as stimulation of ventilation. The risks of hypoxia, however, are usually mediated by low tissue PO₂, which may occur as a consequence of a low PaO₂, and other mechanisms such as severe anaemia and low cardiac output states.

These problems can be illustrated in the pathophysiology of myocardial ischaemia, which will develop when there is an imbalance between myocardial DO₂ and oxygen consumption (VO₂). DO₂ is not only dependent on PaO₂, but also coronary flow and haematocrit. VO₂ will also depend on the stroke work of the heart. Therefore, defining a lower limit of PaO₂, which is considered safe, is impossible given the other variables.

Hypoxaemia refers to an abnormally low oxygen tension in the blood (see section 4.1). However, it is not possible to define a single level of hypoxaemia that is dangerous to all patients. Some patients with chronic lung disease may be accustomed to living with oxygen saturation as low as 80% (arterial oxygen tension about 6kPa or 45 mm Hg) whilst other patients with acute organ failure may be harmed by short-term exposure to saturation below 90% (arterial oxygen tension below 8 kPa or 60 mm Hg).

It has been demonstrated that medical patients with sustained desaturation below 90% have impaired medium term survival compared with medical patients with saturations which stay above 90% (15). However, much of this survival disadvantage is due to the underlying disease which has caused the low oxygen level (e.g. severe COPD or pneumonia) so the contribution of modest hypoxaemia to mortality rates is not known.

Mental functioning becomes impaired if the PaO₂ falls rapidly to less than 6 kPa (45 mm Hg, saturation below 80%) and consciousness is lost below 4 kPa (30 mm Hg, saturation below 56%) in normal subjects. (185, 205, 212, 218) Young subjects tolerate acute hypoxaemia for longer than older subjects in terms of "time of useful consciousness". (218) Safe levels of hypoxaemia in COPD have been discussed in detail in a review by Murphy and colleagues. (248) Many COPD patients have a PaO₂ less than 5 kPa (37.5 Hg) corresponding to a saturation below 70% during an acute exacerbation (192). Furthermore, sudden hypoxaemia is more dangerous than hypoxaemia of gradual onset both in health and in disease. For example, millions of people live at altitudes above 3000 metres despite an average PaO₂ of about 7.3 kPa (55 mm Hg, saturation about 88%) and acclimatised climbers on Mount Everest can tolerate short-term exposure to an oxygen saturation of 70% or less which corresponds to a PaO₂ of about 5 kPa or 37.5 mm Hg. (240). Campbell summarised this issue eloquently in 1967 when he said "Better a year at a PaO₂ of 50 mm (6.7 kPa) than an hour at a PaO₂ of 20 mm Hg (2.7 kPa)". (196) Hypoxic hepatitis has been reported in patients with respiratory failure associated with oxygen levels below 4.5
kPa or 34 mm Hg (35), whereas hypoxic hepatitis in cardiac patients is mainly due to decreased hepatic blood flow (stagnant hypoxia) and occurs at higher blood oxygen levels. (36)

An in-flight study of patients with COPD with mean oxygen saturation of 96% at sea level showed a fall to 90% in flight in a commercial airliner and a further fall to a mean saturation of 87% whilst walking in the aircraft aisles. These patients had no symptoms during these hypoxaemic episodes. (241) A study of healthy airline cabin crew has shown that the oxygen saturation of flight attendants falls to a mean nadir of 88.6% without causing breathlessness or any other symptoms. Individual nadirs of oxygen saturation ranged from 93% down to 80%. (222) Without any randomised evidence, the guideline production team have suggested that the level of saturation which is tolerated by healthy people without any symptoms (about 85% saturation) should be regarded as the safe lower limit of hypoxaemia. However, other co-morbidities may need to be taken into account and expert opinion recommends that the saturation should be maintained above 90% for seriously ill patients. (15, 315, 363, 367) For this reason, a target saturation above 92% is suggested for most hypoxaemic patients to ensure that the actual oxygen level remains above 90% for most of the time with a 2% margin of safety to allow for oximeter error and artefact such as a weak signal or dark coloured skin. The accuracy of and pitfalls of oximetry are addressed in section 7.1.2. Specific targets for oxygen therapy in other diseases will be considered theoretically in this section and practically in Sections 8 and 9.

In assessing an ill patient, the oxygen saturation level is only one of several physiological variables that should be monitored. Many patients with sudden acute illness such as post-operative pulmonary emboli will have a sudden alteration in physiological variables as assessed by modified Early Warning Scoring systems (mEWS). (287, 362) Such patients may have only a small fall in oxygen saturation due to physiological compensation mechanisms such as increased ventilation. Therefore, health care professionals need to be alert for falls in oxygen saturation even within the recommended target ranges.

This Guideline will recommend a target range of 94-98% for patients aged below 70 and 92-98% for other patients. This reflects the normal range of oxygen saturation in healthy adults as discussed in section 4.1. It must be recognised that the normal range varies with age and the lower end of the normal saturation range is 92.5% for males aged 70 and above and 91.5% for females in this age group whereas the lower limit of normal for a young adult is 96%. Despite the different normal ranges in different age groups there is no evidence that young people would be harmed at an oxygen saturation such as 93% that is below the lower limit of normal for a young adult but within the normal range for an older person. If anything, older people are more likely to have a compromised cardio-pulmonary system and impaired organ function and thus, may tolerate significant hypoxaemia less well than young adults. This has been demonstrated in the case of the cerebral response to hypoxia as discussed above. (218)

If the oxygen saturation in an adult below the age of 70 should fall below 94%, the key issue is to identify and treat the cause of the fall in oxygen saturation (e.g. pulmonary embolism) rather than just correcting the hypoxaemia which is not, of itself, dangerous at this level. However, there is a danger that health care workers might fail to respond appropriately to abnormal hypoxia in an adult below the age of 70 if the oxygen saturation was seen to be within the “target range”. For this reason, the Guideline will recommend a target range for adults below the age of 70 of 94-98% to ensure that health care workers will respond appropriately and request medical assessment if a patient should have a significant fall in oxygen saturation. Oxygen saturations has a larger standard deviation in the
elderly (section 4.1) and therefore the guideline recommends a target saturation of 92-98% in those aged 70 and above. Some elderly adults will have a saturation at the upper end of the range and any sudden fall should prompt fuller assessment even if the value is still within the target range, e.g., a fall from 98% to 92%. The guideline committee have recommended a target saturation of 92-98% in the elderly rather than one target range of 94-98% because many elderly patients who are well would be outside of the range of 94-98%.

6.5.1 Desirable oxygen saturation ranges in acute illness

- Acute hypoxaemia is considered dangerous to healthy subjects below a PaO2 of about 6 kPa (45 mm Hg) or a saturation of about 78% due to impaired mentation and risk of tissue hypoxia but patients with acute illness or chronic organ disease or ischaemia are likely to be at risk at oxygen tensions above 6 kPa. Evidence Level III

- Changes in physiological monitoring systems such as mEWS may occur in acute illness with either no change or only a small change in oxygen saturation levels. Evidence level III

- Critical illness may present initially with only a small fall in oxygen saturation level because of compensating mechanisms. Evidence level IV

- The upper end of the recommended range in this Guideline (98%) is the upper limit of oxygen saturation in healthy adults. Evidence level III

- The lower end of the suggested target saturation range (92% for patients aged ≥70) is about the lower end of the normal range in older subjects and ensures that the oxygen saturation remains above 90% most of the time. Evidence Level III

- The lower end (94%) in adults below 70 years of age is about the lower end of normal for these ages. Evidence level III.
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6.6 Effects and risks of hyperoxia

These are summarised in Table 6b and in the review by Downs (228) and in other sources. (209,210, 233) The following paragraphs will summarise the physiology and pathophysiology of hyperoxia.

6.6.1 Respiratory system. The most significant effect of excess oxygen on the respiratory system is hypercapnic respiratory failure in a population of vulnerable patients. This does not occur in the absence of significant pulmonary disease or musculoskeletal disease affecting the thorax. There are at least five mechanisms responsible for this:

- **V/Q mismatch.** During air breathing, poorly ventilated alveolar-capillary units will be hypoxic and, therefore, poorly perfused due to hypoxic pulmonary vasoconstriction (HPV). If high-concentration oxygen is administered, the alveolar oxygen tension will rise, thus reversing the hypoxic pulmonary vasoconstriction and increasing blood flow to that unit. However, although the oxygen in the unit has increased it remains poorly ventilated, with a high PA\textsubscript{A}CO\textsubscript{2} and therefore high pulmonary venous PCO\textsubscript{2}. As more blood is now passing through these units the PaCO\textsubscript{2} will rise. Normally when there is no significant lung disease or thoracic musculoskeletal disease, the respiratory system is able to compensate for these changes by increasing overall ventilation thereby lowering PaCO\textsubscript{2}. However, where respiratory mechanics are such that increased ventilation is not possible, PaCO\textsubscript{2} will rise. Several authors have reported that this mechanism is more important than reduction in ventilatory drive in producing hypercapnia when supplementary oxygen is administered but this continues to be a controversial area of respiratory physiology. (12, 25, 28, 59, 83, 269, 356, 357)

- **Ventilatory drive.** Hypoxaemia drives an increase in ventilation so it follows that relief of hypoxaemia will cause a decrease in ventilation. The consequent rise in PaCO\textsubscript{2} is inversely proportional to the decrease in ventilation, such that a halving of ventilation will lead to a doubling in PaCO\textsubscript{2}, assuming constant CO\textsubscript{2} production. As seen in Figure 6.c any increase in PaO\textsubscript{2} above 8 kPa (60 mmHg) will not reduce ventilation significantly and beyond a PaO\textsubscript{2} of 13 kPa (100 mmHg), further increases in PaO\textsubscript{2} will have no impact on ventilation. Several clinical studies have suggested that "hypoxic drive" makes only a small contribution to the rise in PaCO\textsubscript{2} that is seen clinically when high dose oxygen is given to patients with COPD but one recent study has supported this mechanism. (12, 25, 28, 59, 83, 269, 356, 357)

- **Haldane effect.** The third effect of increasing FIO\textsubscript{2} is to decrease the CO\textsubscript{2} buffering capacity of haemoglobin through the Haldane Effect.

- **Absorption atelectasis.** The fourth effect, absorption atelectasis, is thought to occur as a result of absorption of oxygen from alveoli with high PA\textsubscript{A}O\textsubscript{2} beyond obstructed airways. This can happen at FIO\textsubscript{2} as low as 30-50% and will result in a shunt (increased VQ mismatch). (228)

- **Higher density of oxygen compared with air.** Johnson and colleagues (64) have shown a reduction of FEV1 in patients who were breathing pure oxygen compared with breathing air. They concluded that this
effect was probably related to the slightly increased density and viscosity of oxygen relative to air. This would increase the work of breathing which could contribute to hypercapnia in an exhausted patient.

It has been stated for several decades, that “hyperoxaemia causes hypercapnic respiratory failure by producing decreased respiratory drive in patients with intrinsic lung disease, such as COPD” and many, if not most medical textbooks from the 1960s to the present time refer to "loss of hypoxic drive" as the main cause of hypercapnia and acidosis when high-dose oxygen is given to patients with acute exacerbation of COPD. This assertion is usually attributed to the late EJM Campbell who championed the concept of controlled oxygen therapy in the 1960s. However, Campbell has been widely misquoted. What he actually said in 1967 was as follows; "It is usual to attribute the rise in PaCO2 in these patients to removal of the hypoxic drive to ventilation but I share the doubts of Pain and co-workers (269) that this is the whole story; changes in the pulmonary circulation may also be important." (196) Most, but not all subsequent studies have shown that Campbell was correct in this assumption. (12, 25, 28, 59, 83, 269,356, 357)

The theory of “loss of hypoxic drive” as the cause of hypercapnia is further confounded by the observation that PaCO2 continues to rise as PaO2 is increased above 13 kPa (100 mmHg), which has little impact on decreasing ventilation. Therefore, whilst a small reduction in ventilation may be a contributing factor to the rise in carbon dioxide levels during oxygen therapy in COPD, the major factor is the worsening of V/Q matching. Additional effects of increasing FiO2 will relate to atelectasis and perhaps worsening airflow obstruction due to increased viscosity. In diseases where there is little intrinsic lung disease, but significant respiratory muscle weakness, loss of hypoxic respiratory drive will be a greater factor in the development of hypercapnia. However, hypoxic pulmonary vasoconstriction (HPV) remains a significant regulator of V/Q matching even in non-diseased lung.

6.6.2 **Cardiovascular and cerebrovascular system.** The theoretical risks of hyperoxia have been summarised by Thomson and colleagues in an editorial, which made a strong case for more trials. (233) Hyperoxaemia causes coronary vasoconstriction and if the haematocrit is sufficiently low, this may theoretically cause paradoxical myocardial hypoxia because of overall reduction in DO2.

One randomised double blind trial of oxygen in uncomplicated myocardial infarction found higher rates of sinus tachycardia and a significantly greater myocardial enzyme rise in the oxygen group, suggesting a greater infarct size. (69) There was a three fold increase in mortality on oxygen therapy that did not reach statistical significance (3 deaths out of 77 patients treated with air versus 9 deaths amongst 80 patients given oxygen at 6 l/min via simple face mask for 24 hours). That trial was published in 1976 and oxygen has been given routinely to millions of patients with myocardial infarction and chest pain for a further 30 years without any evidence to support the practice. Furthermore, a more recent trial showed increased mortality in patients with non-hypoxaemic strokes of mild to moderate severity in those randomised to treatment with oxygen. (235) This creates an urgent need for large randomised trials of oxygen therapy for non-hypoxaemic patients with acute cardiac and cerebral ischaemia.
6.6.3 **Reactive oxygen species.** Aside from the potentially detrimental physiological effects of hyperoxaemia, the toxic effects, mediated by reactive oxygen species (ROS), have potential risk. (152) Excess ROS are generated in the presence of high tissue PO2 in the form of hydrogen peroxide and super oxide, causing oxidative stress and free radical damage. At physiological levels, ROS act as signalling molecules, but at higher levels they are cytotoxic, notably being released by primed neutrophils as a host defence mechanism. It is thought that ROS are responsible for the development of bronchopulmonary dysplasia in ventilated, hyper oxygenated premature infants[354] and reperfusion injury post-myocardial infarction. (355) Thompson et al. (233) have suggested that oxygen should be “prescribed, administered, and monitored with care” in order “to achieve optimal tissue oxygenation”, not maximal oxygenation. This view was proposed by other authors such as Bryan and Jenkinson in the 1980s (152) but standard medical practice has not taken note of this advice. Because there are no published data suggesting benefit from hyperoxaemia for most medical conditions and because of the theoretical risks, optimal management should aim for physiological oxygenation. Targets for oxygen therapy in specific circumstances, with evidence, are discussed in Section 8

6.6.4 **Delay in recognition of physiological deterioration.** It was previously believed that a high FIO2 is protective and gives patients a margin of safety. However, Downs has argued that unstable patients may actually be placed at risk by the precautionary use of high dose oxygen therapy. (162, 228) During physiological deterioration, a patient given high dose oxygen therapy would have a normal or high pulse oximeter reading masking a progressive decline in the PaO2/FIO2 ratio and therefore not alerting staff to impending deterioration requiring mechanical support.

6.6.5 **Lung injury in patients with acute paraquat poisoning, bleomycin lung injury and acid aspiration.**

Oxygen is known to be hazardous to patients with paraquat poisoning (273, 274) and oxygen potentiates bleomycin lung injury and may potentiate lung injury from aspiration of acids. (96, 275, 276) Further details concerning these conditions are given in section 8.10.25

**Summary of risks of hyperoxia**

**Physiological Risks**

- Worsened VQ mismatch
- Absorption atelectasis
- Coronary vasoconstriction
- Reduced cardiac output
- Damage from oxygen free-radicals
- Increased systemic vascular resistance

**Clinical Risks**

- Worsening of hypercapnic respiratory failure
- Delay in recognition of clinical deterioration
- Worse outcomes in mild-moderate stroke
- Specific risk in patients with previous Bleomycin lung damage, or with paraquat poisoning or acid aspiration
- Unknown risk-benefit balance in acute coronary artery disease with normal oxygen saturation

- **High dose oxygen therapy to produce hyperoxaemia (above-normal oxygen saturation) can be harmful to patients who are at risk of hypercapnic respiratory failure and may also cause absorption atelectasis, myocardial ischaemia and unfavourable outcomes in some patient groups (e.g. patients with mild and moderate strokes).** Evidence Levels Ib to III

### 6.6.6 Benefits of hyperoxaemia in specific conditions. Hyperoxaemia is useful in some clinical situations. The best example of this is carbon monoxide (CO) poisoning. CO combines with haemoglobin and has a higher affinity for haemoglobin molecule giving rise to carboxyhaemoglobin (COHb). The half-life of the COHb is about 4-5 hours when breathing air but is reduced to about 40 minutes when breathing 100% oxygen. Hyperoxaemia may also be used to accelerate the resolution of pneumothorax in patients who do not require a chest drain. (283, 284)

### 6.6.7 Other potential benefits of oxygen therapy in non-hypoxic patients.

A) Possible reduction of nausea and vomiting in post-operative patients and in ambulances. Although some reports have suggested that oxygen may have a specific anti-emetic effect during ambulance transfers and in the post-operative state, subsequent studies reported no effect on motion sickness and no anti-emetic effect in post-operative patients. (65, 82, 260, 270, 271)

B) Short-term post-operative oxygen therapy (for 2 hours) has been shown to reduce the risk of surgical wound infections in double-blind trials of patients having bowel surgery but not in general surgery. (278-280).

C) Reported to improve anastomotic integrity in animal models (272) and potential benefit in human anastomotic surgery. (281)

D) Reported benefits of oxygen therapy in healing of established wounds and in treatment of wound sepsis are controversial. Hyperbaric oxygen reduced the risk of amputation in patients with chronic diabetic foot ulcers and may improve the chance of healing over one year but the Cochrane reviewers had concerns about the size and quality of existing studies and recommended further trials. (291) It is not known if conventional oxygen therapy has any effect on wound healing. (292)

E) Relief from cluster headaches has been reported in about 60% of cases but this observation is based on very small studies from the 1980s. (282, 285) Although this could be considered as a form of emergency oxygen therapy, these patients are not breathless.

F) Most guidelines for Cardio-Pulmonary Resuscitation and the care of patients with critical illness recommend the use of 100% oxygen in the initial stages of resuscitation. Although these recommendations are not evidence-based, it is unlikely that controlled trials would ever be undertaken using different levels of oxygen therapy in these emergencies and it seems intuitive to maximise oxygen delivery for critically ill patients with circulatory collapse. However, randomised trials have
been undertaken of resuscitation of neonates breathing room air or oxygen and the unexpected outcome of a Cochrane review was that the outcome was possibly better when room air was used. (286) This surprising finding cannot be extrapolated to adult patients but it does emphasise the need for clinical trials even in areas where one might intuitively believe that oxygen would be beneficial.

G) It has been shown that deliberately increasing oxygen delivery to the tissues in critically ill patients as well as high risk surgical patients reduces organ failure, reduces length of ICU stay and most importantly, improves survival. (57,75,78,221) Increased oxygen delivery, in part involves oxygen therapy, however these studies did not show any benefit from aiming at supraphysiological oxygen delivery.

H) Through relief of breathlessness and work of breathing, oxygen therapy may decrease CO2 production and consequently offset some of the potential increase in PaCO2 that might otherwise occur due to the mechanisms described in section 6.6.1 However, there are no controlled trials supporting the use of oxygen for this indication.

6.7 Risks of hypercapnia (and respiratory acidosis)

Hypercapnia and respiratory acidosis are inextricably linked and are best considered together. Some of the consequences of an elevated carbon dioxide tension are a consequence of the resulting acidosis. Sometimes the effect of a raised carbon dioxide tension on a particular organ system is opposed by an opposite effect of acidosis.

Carbon dioxide is a vasodilator so patients with hypercapnia may appear flushed with dilated peripheral veins and a pounding pulse. Cranial vasodilation may cause headache. Carbon dioxide in high concentrations has hypnotic effects and patients with hypercapnia may progress from drowsiness to confusion to coma. (195, 201, 202,203, 204, 248, 331) A link has been shown between severe respiratory acidosis in acute COPD and an increased risk of death or requirement for mechanical ventilation. (48) However, the problem of respiratory acidosis is not confined to patients with COPD. Depressed respiration for any reason will give rise to hypercapnia. Examples are opiate overdoses, obesity with hypoventilation and neuro-muscular disorders affecting the muscles of respiration.

6.7.1 Effects of a raised blood carbon dioxide level

1. Nervous system. Carbon dioxide exerts its effect either directly or as a consequence of acidosis. Hypercapnia increases cerebral blood flow and thereby may influence the CSF pressure. It is the main factor influencing the intracellular pH which has an important effect on cellular metabolism. It exerts an inert gas narcotic effect similar to that of nitrous oxide. It influences the excitability of neurones particularly relevant in the reticular activating system. Carbon dioxide can induce narcosis when the PaCO2 rises above 12 to 16 kPa (90 to 120 mm Hg). (331)

2. Endocrine system. Hypercapnia increases plasma levels of both adrenaline and noradrenaline.
3. Pulmonary circulation. An elevated alveolar PCO2 causes vasoconstriction in the pulmonary circulation although the effect is less marked than that of hypoxia. (332) In healthy subjects an end expiratory PCO2 of 7 kPa (52 mm Hg) increases pulmonary vascular resistance by 32% which along with elevated cardiac output increases mean pulmonary artery pressure by 60%. (333) Changes in pH are thought to be the primary factor responsible for CO2 mediated changes in the pulmonary vasculature. (334, 335) Consequently, as with HPV, changes in PAhCO2 help to match perfusion to ventilation.

4. The respiratory system. As explained in 6.2.1, a raised CO2 may worsen hypoxia and its effects because the concentration of carbon dioxide in the alveolar gas reduces that of oxygen if the concentration of nitrogen remains constant. Also an increase in PaCO2 shifts the oxygen dissociation curve to the right.

5. Cardiovascular system. In general both hypercapnia and acidosis have direct depressant effects on cardiac myocytes and vascular smooth muscle cells. (336) These effects are normally opposed by the increased in catecholamines caused by the elevated PaCO2. The overall effect of carbon dioxide on the cardiovascular system is therefore unpredictable. In artificially ventilated children a rise in CO2 increases cardiac output and reduces total peripheral resistance and blood pressure tends to rise. (337) Although an elevation in CO2 depresses heart rate tachycardia is more common because of the effects of catecholamine stimulation overriding the depressant effects on the heart. Arrhythmias have been reported, but are seldom clinically significant in normal subjects.

6. Kidneys. Renal blood flow and GFR are reduced in the presence of high levels of PaCO2. If severe this can lead to anuria.

7. Blood electrolyte levels. The acidosis that accompanies hypercapnia may cause a rise in potassium if the acidosis is severe and sustained.

6.7.2 Clinical signs

The clinical signs of hypercapnia are described in detail in section 7.2.1, the main signs are as follows:

- Vasodilation producing flushing and warm peripheries with dilated blood vessels (including retinal veins)
- Bounding pulse
- Drowsiness
- Flapping tremor
- Confusion
- Coma

6.8 Risks of acidosis

The major effect of acidosis is depression of the central nervous system with severe acidosis (pH < 7.0) causing disorientation and later coma. However as described above the effects of pH are inextricably linked with both
hypoxia and hypercapnia. As a consequence of opposing effects of acidosis, hypoxia and hypercapnia on different target organs in individual patients together with the fact that derangements of all three components may occur at the same time it is very difficult to predict the effects of acidosis per se in an individual patient. Furthermore, tissue hypoxia will exacerbate acidosis. The consequences will depend upon the interplay of the three variables, complicated by the effects of co morbid disease states. It is well know that in patients with COPD a pH < 7.30 during an acute exacerbation is associated with a much worse prognosis (48).

6.9 Rationale of oxygen therapy

Oxygen therapy is usually defined as the administration of oxygen at concentrations greater than found in ambient air. Usually, it is undertaken to treat or prevent hypoxaemia thereby preventing tissue hypoxia which may result in tissue injury or even cell death. In some circumstances such as carbon monoxide poisoning or cluster headache, oxygen therapy is used to achieve hyperoxia. There are no published trials supporting the use of oxygen to relieve breathlessness in non-hyoxaemic patients and there is evidence from randomised studies that oxygen does not relieve breathlessness compared with air in non-hyoxaemic COPD patients who are breathless following exertion. (374)

At the tissue level, mitochondrial activity requires oxygen for aerobic ATP synthesis for cellular activity. PaO2 of dry air at sea level is 21.2 kPa (159 mmHg) but at the mitochondrion, PO2 is in the range of 0.5-3.0 kPa (4-22 mm Hg), depending on tissue type and local metabolic activity. This gradient from atmosphere to mitochondrion is known as the oxygen cascade. There are many factors in this cascade that affect the final mitochondrial PO2, including alveolar gas exchange, oxygen transport in the blood and tissue perfusion. Under pathological conditions, any change in one step in this cascade may result in hypoxia at the mitochondrial level. Therefore, although not necessarily addressing the underlying cause of tissue hypoxia, increasing FIO2 with oxygen therapy is the simplest and quickest way of avoiding hypoxic tissue damage. Besides oxygen therapy, other steps are usually necessary to improve the delivery of oxygen (DO2) to the tissue (see section 6.12).

6.10 Target oxygen saturation in acute illness (see also Section 6.5)

In many clinical situations, oxygen therapy is applied without a specific end point in mind. It has been suggested by many studies that hyperoxia can have deleterious physiological and clinical effects (see section 6.6), albeit such effects are not widely reported in conditions other than COPD. However, potential for harm may well exist with hyperoxia and good medical practice should be followed as in all drug prescriptions. As the actual PO2 at mitochondrial level is so variable and dependent on many other variables other than PaO2, it is often difficult to set a minimum level of PaO2 below which definite cell damage will occur or above which the host is safe from the effects of hypoxic cell damage. In addition, it is not possible to monitor mitochondrial PO2 clinically and the only clinically available surrogate of mitochondrial hypoxia is lactate production. Although blood lactate levels are useful and
indicate tissue hypoxia, it is a late marker and therefore is an insensitive tool. Thus targets set for ‘ideal’ blood gases are based on arbitrary goals. Due to the natural decline in normal arterial oxygen levels with age, it has been suggested that ideal target arterial PO\(_2\) can be determined by the following equation: (361)

\[
\text{Ideal PaO}_2 = 13.3 \text{ kPa} - 0.04 \times \text{age (in years)}
\]

Or \[
\text{Ideal PaO}_2 = 100 \text{ mmHg} - 0.3 \times \text{age (in years)}
\]

In terms of oxygen saturation measured by the bedside, this would translate into SaO\(_2\) 92-98% in most situations. This strategy avoids tissue hypoxia in almost all patients and also avoids potential deleterious effects of hyperoxia. Thus the standard of practice should be to prescribe oxygen to a specific saturation (or PaO\(_2\)) rather than in terms of FIO2.

Clearly, consideration will need to be given to patients who have oxygen-sensitive CO\(_2\) retention and targets may well have to be set lower for these patients to strike a balance between achieving a desirable and safe SaO\(_2\)/PaO\(_2\) and CO\(_2\) retention. Specific disease states will be addressed in Section 8

Patients with moderate to severe hypoxaemia are usually breathless and have an increased respiratory rate. Apart from causing physical tiredness, this also increases work of breathing, therefore increasing both O2 consumption and CO2 production. Relief of breathlessness decreases oxygen consumption and reduces CO2 production. Therefore, oxygen therapy should theoretically improve breathlessness although this has been difficult to confirm clinically. For example a recent meta-analysis of all published blinded studies of short burst oxygen therapy for COPD patients with breathlessness failed to confirm any clinical benefit despite the widespread belief of doctors and patients that oxygen relieves breathlessness in this condition and a systematic review of oxygen and airflow on the relief of dyspnoea at rest in patients with advanced disease of any cause found only low grade scientific evidence that oxygen and airflow improve dyspnoea in some patients with advanced disease at rest. (258, 374)

**Recommendations from sub-sections 6.5 to 6.10**

1. This Guideline suggests aiming to achieve a normal or near-normal oxygen saturation for all acutely ill patients apart from those at risk of hypercapnic respiratory failure. Grade D

2. The suggested target saturation for patients not at risk of hypercapnic respiratory failure, aged below 70 is 94-98% and the target range for those aged 70 and above is 92-98% to reflect the wider normal range in the latter age group. Grade D

3. A sudden reduction of more than 3% in a patient's oxygen saturation within the target saturation range should prompt fuller assessment of the patient because this may be the first evidence of an acute illness. (The narrower target range in younger patients is to ensure prompt assessment if a patient falls outside the age-specific normal range, not due to greater vulnerability to hypoxia). Grade D

4. For most patients with known COPD or other known risk factors for hypercapnic respiratory failure (e.g. morbid obesity, chest wall deformities or neuro-muscular disorders), a target saturation range of 88-92% is suggested pending the availability of blood gas results. Grade C
Some patients with COPD are vulnerable to repeated episodes of hypercapnic respiratory failure. In these cases, it is recommended that treatment should be based on the results of previous blood gas estimations during acute exacerbations. Grade D

6.11 Physiology of oxygen therapy

Oxygen therapy increases PA\textsubscript{v}O\textsubscript{2} and is therefore only effective when alveolar-capillary units have some functional ventilation. Oxygen therapy is ineffective if there is a pure shunt (such as pulmonary arterio-venous malformations), where mixed venous blood does not pass through an alveolar-capillary unit. There will only be a small overall increase in PaO\textsubscript{2} due to an increase in dissolved oxygen in the pulmonary venous blood from ventilated alveolar-capillary units, which is small compared to the content of oxygen carried by haemoglobin.

In poorly ventilated units (\textit{i.e.}, low V/Q ratio) PA\textsubscript{v}O\textsubscript{2} will be low. Increasing FIO\textsubscript{2} will increase PA\textsubscript{v}O\textsubscript{2} and therefore PaO\textsubscript{2}. Hypoventilation disorders can be considered as lungs made up entirely of low V/Q units.

When there is diffusion limitation due to increased alveolar-capillary membrane thickness, such as in fibrotic lung disease, increasing PA\textsubscript{v}O\textsubscript{2} will augment the rate of diffusion across the alveolar-capillary membrane by increasing the concentration gradient.

Increasing dissolved oxygen in plasma by oxygen therapy may also be used to offset the effects of hypo perfusion to some extent (stagnant hypoxia) and may well be important in certain situations (cardiogenic shock), although the effect is only marginal. Increased inspired oxygen will only marginally mitigate the effects of anaemic hypoxia; but because the CaO\textsubscript{2} in anaemic patient is less than that in patients with normal haemoglobin, the effect of additional oxygen carried in solution may become more important in these situations.

6.12 Strategies for improving oxygenation and delivery

Tissue oxygenation is dependent upon optimal or adequate oxygen delivery to the tissue (DO\textsubscript{2}). This physiological process is composed of various components that independently and interdependently influence and determine DO\textsubscript{2} and therefore, tissue oxygenation. These components can be considered sequentially.

Optimising PaO\textsubscript{2}:

The physiology of oxygen therapy has already been discussed in the previous section. However, increasing FIO\textsubscript{2} is only one component in increasing O2 uptake in the lungs. Other key manoeuvres to ensure oxygen delivery to the alveolar-capillary bed include:

- Maintaining a satisfactory airway
- Ensuring adequate alveolar ventilation
- Reversing any respiratory depressants, such as narcotics
- Invasive or non-invasive ventilation where necessary
Optimising oxygen carriage:
Oxygen is carried in blood mainly by haemoglobin with only a very small amount of oxygen dissolved in the blood itself. Adequate haemoglobin is therefore essential for optimal oxygen content (CaO2) of blood. The ideal haemoglobin level for optimal CaO2 and therefore for optimal DO2 has long been a subject of debate. Previous practices have favoured haemoglobin close to 100g/l (10 g/dl), providing adequate CaO2 as well as reducing viscosity of blood for better perfusion in critically ill patients. However, studies by Canadian researchers in the late 1990s have shown that Hb levels of 70g/l (7 g/dl) were as safe as higher levels and may produce fewer complications in the critically ill. (293) However, this study was conducted using non-leukocyte depleted blood and it is possible that some of the infective complications in the group who were given more transfusions might have been avoided by the use of leukocyte-depleted blood. Therefore, the optimal transfusion target for critically ill patients remains the subject of ongoing discussion amongst experts in critical care medicine. The issue of optimal haemoglobin in patients with coronary artery disease is not settled, however haemoglobin levels of 100g/l (10 g/dl) are recommended for adequate DO2.

Example:
For a septic anaemic patient with haemoglobin of 50 g/l (5 g/dl) and an oxygen saturation of 90%:
Increasing oxygen saturation from 90% to 100% will increase total haemoglobin oxygen content by about 10%.
Transfusing 2 units of packed red cells will increase total haemoglobin oxygen content by about 40%.

Optimising delivery:
Besides adequate CaO2 and PaO2, delivery of oxygen depends upon adequate flow of oxygenated blood. Cardiac output in turn depends upon adequate blood (circulating) volume, adequate venous return and adequate and optimal myocardial function. Thus to avoid tissue hypoxia, attention must be paid to the volume status of the patient and the adequacy of cardiac function, as well as initiating oxygen therapy. In severely shocked patients (e.g., cardiogenic shock, septic shock), invasive monitoring and inotropic/vasopressor therapy will usually be indicated in appropriate higher dependency environments. It has been shown that deliberately increasing oxygen delivery in critically ill patients as well as high risk surgical patients reduces organ failure, reduces length of ICU stay and most importantly, improves mortality. (57,75,78,221) Increased oxygen delivery, in part involves oxygen therapy, however these studies did not show any benefit from aiming at supraphysiological oxygen delivery.

The following worked example illustrates how minor abnormalities in each of the parameters discussed above, when occurring together can result in dramatic falls in oxygen delivery.

Oxygen delivery in health can be calculated as follows, where CO is cardiac output:

\[ DO2 = CO \times CaO2 \]
\[ \text{DO2} = \text{CO} \times \left[ \frac{[\text{SaO2}/100 \times \text{Hb} \times 1.3]}{100} + [\text{PaO2} \times 0.003 \text{mmHg} \times 10] \right] \]

Therefore:
\[ \text{DO2} = 5 \times \left[ (0.98 \times 150 \times 1.3) + (100 \times 0.003 \times 10) \right] \]
\[ \text{DO2} = 970 \text{ ml/min or } \approx 1000 \text{ ml/min} \]

This is well above the normal oxygen consumption (VO\(_2\)) of about 250 ml/min.

Now consider an anaemic patient with a Hb of 10 g/dl, a cardiac output of 3.5 l/min and a SaO\(_2\) of 90% and the oxygen delivery becomes approximately 410 ml/min.
\[ \text{DO2} = 3.5 \times \left[ (0.9 \times 100 \times 1.3) + (60 \times 0.003 \times 10) \right] \]

Although this value is still above the VO\(_2\) at resting physiology, in practice, the VO\(_2\) would most likely have risen to due a number of factors, such as increased work of breathing, increased catabolic state of sepsis etc. This example is not rare and occurs daily in clinical practice. Therefore it is important not to consider oxygen therapy in isolation.

As many patients may not have adequate Hb, cardiac output or blood volume, they may suffer from tissue hypoxia when they become acutely ill. All such patients should have supplemental oxygen therapy until they are evaluated by a responsible healthcare professional.

6.13 Effects of body positioning including restraint systems

Appropriate positioning of a patient can maximise V/Q matching. In the healthy, self-ventilating adult lung, V/Q matching improves from non-dependent to dependent areas. In lung disease there is a disruption of this pattern and in these instances appropriate positioning may be advantageous in optimising V/Q matching, therefore improving gas exchange, oxygenation and CO2 clearance. For these reasons, breathless patients usually prefer to sit upright or near upright provided they are able to do so.

The relationship between dependency and V/Q matching is maintained irrespective of the position of the subject. The physiology is then transferable into alternate side lying positions, for example in left side lying the dependent lung (left) will have the better V/Q matching. This is important in the presence of asymmetrical lung pathology, as the ‘good lung down’ principle will maximise V/Q matching.

Many unwell patients are nursed in the semi recumbent and supine positions. These positions do not facilitate V/Q matching as in the upright and full side lying position due to the hindrance to expansion of dependent lung by the diaphragm and chest wall. Even in healthy subjects, the oxygen tension is 0.7 kPa (5 mm Hg) lower in the supine position compared with the upright position. (232) Similarly, 10% of patients with right hemi paresis and concomitant chest disease were more hypoxaemic in the left lateral position (49). Where there is pathological lung disease, and hence already significant V/Q mismatch, gas exchange may be further impaired. This is discussed in a review of the effects of position on oxygen saturation in acute stroke. (53) Acute stroke patients without respiratory co-morbidities may be permitted to adopt any body position that the patient finds most comfortable, whilst those...
with respiratory compromise should be positioned as upright as possible, avoiding slouched or supine positions to optimise oxygenation. (53).

The semi recumbent/supine position is commonly adopted in an ambulance. In addition, for safety, the patient is strapped into the stretcher using abdominal and chest restraints, with their arms by their side. Whilst there is a lack of specific data regarding this, physiological principles suggest that the use of such positioning and restraints would compromise both respiratory muscle function and gas exchange.

Finally, there are some rare patients with cardiac shunts or lung fibrosis who have "orthodeoxia" which means that they are more hypoxic in the upright position. Other patients with scoliosis or with a paralysed hemi diaphragm may feel more comfortable with the "good lung up". Therefore, these patients should be allowed to choose the position in which their breathing is most comfortable for them.

**Recommendation**

6  Because oxygenation is reduced in the supine position, fully conscious hypoxaemic patients should ideally be allowed to maintain the most upright posture possible (or the most comfortable posture for the patient) unless there are good reasons to immobilise the patient (e.g. skeletal or spinal trauma).  Grade  C
Section 7

Clinical and laboratory assessment of hypoxaemia and hypercapnia

7.1 Assessment of hypoxaemia

7.1.1 Clinical assessment of breathless patients and assessment of cyanosis.
Clinicians examining a critically ill patient should remember the "ABC" of emergency medicine (Airway, Breathing, Circulation). In the case of critically ill patients, it may be necessary to secure the airway and resuscitate a patient before a detailed history can be obtained and before a full physical examination can be undertaken.

In assessing an ill patient, the oxygen saturation level is only one of several physiological variables that should be monitored. Many patients with sudden acute illness such as post-operative pulmonary emboli will have a sudden alteration in physiological variables as assessed by modified Early Warning Scoring systems (mEWS). (287, 362, 382) Such patients may have only a small fall in oxygen saturation due to physiological compensation mechanisms such as increased ventilation. Therefore, clinicians need to be alert for falls in oxygen saturation even within the recommended target ranges.

Recommendation

7 The care of seriously ill patients should be undertaken or supervised by fully trained clinicians and expert assistance from specialists in intensive care or from other disciplines should be sought at an early stage if patients are thought to have major life-threatening illnesses. Grade D

Traditional clinical assessment of hypoxaemia involves clinical inspection of the skin and buccal mucus membranes to decide whether central cyanosis is present or absent. This is a difficult clinical skill, especially in poor lighting conditions. Clinical assessment of hypoxaemia is made even more unreliable by the presence of anaemia or polycythaemia. Some patients may have peripheral cyanosis due to poor peripheral circulation in the presence of normal oxygen saturation. Several studies have shown that hypoxaemia is often not recognised by emergency medical service providers, especially if the patient does not complain of respiratory distress. (92, 94, 120) A systematic review of the literature in 2005 reported that most hypoxaemic patients had at least one vital sign abnormality but skin colour was a poor indicator of hypoxaemia compared with pulse oximetry. (99) For these reasons it is recommended that clinicians should not rely on visual assessments of "cyanosis" but should instead use pulse oximetry to obtain an accurate assessment of a patient's oxygen saturation.

The nature of a patient's presenting illness may make hypoxaemia a likely outcome, thus prompting a careful clinical search for evidence of cyanosis complemented by urgent pulse oximetry. This situation applies to many common acute illnesses such as heart failure, COPD exacerbation, pneumonia and pulmonary embolism. A study of 2276 patients with pneumonia showed that hypoxemia was independently associated with 6 risk factors: age >30 years (odds ratio [OR], 3.2), chronic obstructive pulmonary disease (OR, 1.9), congestive heart failure (OR, 1.5),...
respiratory rate >24 per minute (OR, 2.3), altered mental status (OR, 1.6), and chest radiographic infiltrate involving >1 lobe (OR, 2.2). (123) Acutely ill patients with significant hypoxaemia are likely to have an elevated pulse rate or respiratory rate and, for this reason usually score at least 3 points on a modified early warning scoring system (mEWS). (287, 362, 382) The respiratory rate is the single best predictor of severe illness. (362) However, many patients with marked hypoxaemia may present with non-specific findings such as restlessness and confusion rather than breathlessness and oxygen saturation has been shown to be an independent predictor of mortality in multivariate analysis of the outcome of emergency medical admissions. (375). Furthermore, the work of Thrush et al on normal volunteers has demonstrated that heart rate, blood pressure and respiratory rate are not reliable indicators of hypoxaemia down to saturation levels as low as 70%. (73) This would suggest that the changes in vital signs which are seen in most hypoxic patients are due to the underlying illness rather than due to hypoxaemia per se.

Hypoxaemia may be associated with increased or decreased ventilation. Although some hypoxaemic patients may have reduced levels of ventilation as a causative factor, the majority of hypoxaemic patients have increased minute ventilation in an attempt to increase the blood oxygen level. For example, a patient with an opiate overdose may have reduced ventilation causing hypoxaemia despite having structurally normal lungs whereas a patient with pneumonia or major pulmonary embolism may have significant hypoxaemia due to ventilation-perfusion mis-match despite an increased level of ventilation. The first patient in this example may appear peaceful and non-distressed despite significant hypoventilation and hypoxaemia whilst the second patient is likely to have increased ventilation and tachycardia. Therefore, the clinician needs to make separate assessments of a patient's oxygen saturation and level of ventilation.

Having completed the history and rapid assessment of the patient, more detailed physical examination may reveal signs of an illness such as major pleural effusion, major pneumothorax or unexpected heart failure that may prompt the clinician to anticipate the presence of hypoxaemia.

Advice and recommendations for clinical assessment of patients with suspected hypoxaemia

- The medical history should be taken when possible in an acutely breathless patient and may point to the diagnosis of a particular acute illness such as pneumonia or pulmonary embolism or an exacerbation of a chronic condition such as COPD, asthma or heart failure. Evidence Level IV
- Physical examination may provide evidence of a specific diagnosis such as heart failure or large pleural effusion but it is common for the cause of breathlessness to remain undiagnosed until the results of tests such as chest radiographs are available. – Evidence Level IV
- Patients with severe hypoxaemia may present with a non-respiratory manifestation such as confusion or agitation rather than breathlessness and cyanosis is a difficult physical sign to record confidently (especially in poor light or with an anaemic or plethoric patient). Evidence level IV
- Tachycardia and tachypnoea are commoner than a physical finding of cyanosis. Evidence level III
- Physiological scoring systems such as the Early Warning Scoring system (EWS or mEWS) are extremely valuable in identifying patients with life-threatening illness even if this is not immediately obvious from the patient’s history. Evidence Level III
Recommendations

8 The oxygen saturation should be checked by pulse oximetry in all breathless and acutely ill patients, "the fifth vital sign", (supplemented by blood gases when necessary) and the inspired oxygen concentration should be recorded on the observation chart with the oximetry result. Grade D

9 All clinical staff who use oximeters must be trained in their use and made aware of the limitations of oximetry. Grade C

10 All patients should be fully clinically assessed, including pulse, blood pressure, assessment of circulatory blood volume and respiratory rate. The “ABC” (Airway, Breathing, Circulation) should be used when assessing any patient with apparent cardio-respiratory compromise. Grade C

11 Clinical assessment of acutely unwell patients should include the use of a recognised physiological assessment such as the Modified Early Warning Scoring System (mEWS). Grade C

12 Changes in the physiology monitoring systems such as mEWS indicate that there should be medical review of the patient even if there is no change in oxygen saturation. Grade C

13 Assess for severe anaemia because this has a major influence on oxygen delivery to the tissues. Grade D

14 Clinicians should be prepared to call for assistance when necessary including a call for a 999 Ambulance in pre-hospital care or a call for the Resuscitation Team or ICU team in hospital care. Grade C

7.1.2 Value and limitations of pulse oximetry
Clinical assessment of hypoxaemia has been revolutionised by the advent of pulse oximetry in much the same manner as the clinical assessment of blood pressure was transformed by the invention of the sphygmomanometer. However, it is common to see patients with acute respiratory illness who have had multiple measurements of their blood pressure but no record made of their oxygen saturation, Peak Expiratory Flow or FEV1. In addition to the clinical consequences of under-assessment, Howes and Macnab and colleagues have reported that the availability of a pulse oximeter was highly cost-effective because the finding of normal oximetry (above 94%) in many patients allowed paramedics to use oxygen less frequently with a potential financial saving of up to $2,324 (approximately £1,200) per ambulance per annum. (164, 171)

Pulse oximetry measures haemoglobin oxygen saturation by detecting the absorption of light at two specific wavelengths that correspond to the absorption peaks of oxygenated and de-oxygenated haemoglobin. Oximeters are less reliable at low saturation such as 80% but modern oximeters reflect the arterial oxygen saturation accurately at saturation above about 88%. (72, 84, 126, 134, 135). In almost all clinical circumstances covered by this Guideline, patients with a saturation below 88% will be given intensive therapy to bring the saturation up to at least 90% so the inaccuracy of the instruments at very low saturation levels should not affect patient management.

In one study of 123 adult patients who had simultaneous measurements of pulse oximetry and arterial oxygen saturation measured in arterial blood gases, the 95% confidence interval for the median difference ranged from -0.6 to +0.5%. (126) It has been estimated that an oxygen saturation of 92% or above measured by pulse oximetry has a
sensitivity of 100% and specificity of 86% for excluding hypoxaemia defined as an arterial oxygen saturation below 60 mm Hg (8 kPa). (40)

Oximetry may be less accurate in acutely ill patients on intensive care units but there are no direct comparisons of the accuracy of pulse oximetry in critically ill patients compared with stable patients and healthy individuals. The study of Perkins and colleagues showed a mean SpO2 of 94.6% compared with a mean SaO2 of 95.9% from 1132 simultaneous oximeter and arterial blood gas measurements on an intensive care unit. (366) Fortunately, this average difference of 1.3% was lower for pulse oximeter readings, thus allowing a margin of safety in most cases. This study also showed that fluctuations in oxygen saturation measured by oximetry tended to be greater than changes in arterial oxygen saturation measured with samples from an indwelling radial artery catheter.

Although oximetry is widely used, there are a few clinical studies examining its utility. The Cochrane meta-analysis of the use of oximetry in perioperative monitoring of more than 20,000 patients failed to show any reduction in complications or deaths where oximetry was used although oxygen was given more often to patients who were monitored with pulse oximetry. (246) The authors suggested that the correction of modest hypoxaemia probably does not have much effect on clinical outcomes.

Pulse oximetry gives no information concerning pH, PCO2 or haemoglobin level. Therefore, blood gases and full blood count tests are required as early as possible in all situations where these measurements may affect patient outcomes.

The accuracy of pulse oximetry is diminished in patients with poor peripheral perfusion which may occur chronically in conditions such as systemic sclerosis or acutely in patients with hypotension or hypovolaemia. However, it has been suggested that many types of oximeter may remain accurate at arterial pressures as low as 20 mm Hg so long as the machine is able to obtain a reading despite the low pulse pressure. (104) Most oximeters give an indication of the pulse signal strength. It is important to ensure that the oximeter has a good signal if technically possible and the probe may need to be tried on different fingers or toes or on the earlobe to obtain the best available signal for the individual patient. There are some patients with poor perfusion for whom pulse oximetry measurements cannot be made. This includes patients with cold peripheries, severe hypotension and peripheral “shut-down”.

It must be remembered that oximetry gives a normal reading for oxygen saturation in most patients with anaemia because the oxygen saturation of the available haemoglobin is normal although the total amount of haemoglobin available for oxygen transport is reduced. These patients have normal oxygen saturation levels despite having "anaemic hypoxia" which may cause considerable reduction in the total oxygen content of the blood. It is often not recognised that a patient with SpO2 of 98% but a haemoglobin of 7 g/dl [(7 x 0.98 x 1.34) = 9.2ml O2 / dl]. will have a greatly reduced blood oxygen content compared with a patient with a haemoglobin of 15 g/dl and a saturation of 85%. [(15 x 0.85 x 1.34) = 17ml O2 /dl] (each gm/dl of Hb when fully saturated carries 1.34ml O2)
The accuracy of oximetry is unreliable in the presence of carbon monoxide or methaemoglobin. Both of these substances have similar light absorption characteristics to oxyhaemoglobin so an apparently normal SpO2 in a patient with carbon monoxide poisoning or methaemoglobinemia may be falsely reassuring. Carboxyhaemoglobin levels above 2% may cause falsely elevated SpO2 measurements. (245) Many smokers will have carboxyhaemoglobin levels above this level shortly after smoking a cigarette and the carboxyhaemoglobin level may be elevated to 15% in some smokers and up to 50% or more in acute carbon monoxide poisoning.

Skin pigmentation may also influence the accuracy of pulse oximetry readings (usually over-estimation but sometimes under-estimation). In particular, the accuracy of pulse oximetry is impaired in dark skinned subjects at saturation below about 80-85%. (91, 122, 146) However, this should rarely be a problem in clinical practice if the saturation is maintained in the range suggested in the present guideline (92-98% for most patients) although the work of Jubran and Tobin on ventilated subjects suggested that an oxygen saturation of 92% was useful in predicting a PaO2 above 60 mm (8 kPa) in ventilated white subjects but was less reliable in ventilated black subjects who sometimes had an SpO2 reading that was more than 4% above the directly measured arterial oxygen tension (PaO2). (363) In the case of sickle cell crisis, pulse oximetry may under-estimate the level of oxygenation. (98) In these circumstances, under-reading is safer than over-reading because no truly hypoxaemic patient would be denied oxygen therapy. However, another study found that pulse oximeters did not mis-diagnose either hypoxaemia or normoxaemia during a sickle cell crisis provided a good wave signal was present. (127)

Oximeters can be affected by motion of the patient's hand but this is less of a problem with modern oximeters than with older devices. (86) Motion artefact is more of a problem if the patient also has reduced perfusion of the measuring site. (106) A mal-positioned oximeter sensor can cause artefact which can over-estimate or under-estimate the true oxygen saturation; this can be a particular problem during re-positioning of ill patients. (87)

The site of oximetry is also important. Finger and earlobe measurements are more accurate than measurements from a probe applied to the toe and finger probes may be more accurate than ear probes. (109, 140) Finally, clinical staff needs to remember to remove nail varnish and false nails to avoid artefacts in oximetry measurements.

- **Pulse oximeters are accurate to within 1-2% of directly measured arterial oxygen saturation in most subjects but the error** (usually over-estimation but sometimes under-estimation) **is greater in dark-skinned subjects, especially with very low saturation** (below 80-85%). Evidence level III

- **The accuracy of oximeters in shock, sepsis and hypotension is largely unknown but most errors are likely to result in falsely low readings which would result in additional oxygen being given. Therefore, most errors in oximetry are not likely to place patients at risk, but it is important to ensure that the oximeter has a good signal and it is important to avoid artefact due to motion, nail varnish or other potential sources of error.** Evidence level III

**Recommendations**

- **Pulse oximetry should be used as early as possible in the care of all seriously ill patients.** See recommendation 8.

15 **The presence of a normal SpO2 does not always negate the need for blood gas measurements because pulse oximetry may be normal in a patient with abnormal blood pH or PCO2 or with a low**
blood oxygen content due to anaemia. Therefore, blood gases and full blood count tests are required as early as possible in all situations where these measurements may affect patient outcomes. Grade D

16 It is advised that oximetry measurements on sleeping patients should be recorded over several minutes to avoid the possibility of being misled by a normal transient nocturnal “dip” in oxygen saturation. Grade C

17 In cases of carbon monoxide poisoning, a normal or high oximetry reading should be disregarded because an apparently “normal” saturation may be produced by carboxyhaemoglobin in a patient who needs high dose oxygen therapy due to tissue hypoxaemia. Grade C

18 Pulse oximetry can be misleadingly normal in smokers because of raised blood carboxyhaemoglobin levels which can conceal the presence of arterial hypoxaemia. Therefore, blood gases should be checked in patients with borderline low oximetry levels who have smoked cigarettes in the previous few hours (i.e. 93% or less if aged 70 or above and 95% or less if aged below 70). Grade B

7.1.3 Arterial and arteriolised blood gases.

Arterial blood gases are the "gold standard test" for assessing respiratory failure. However, recent studies have shown that arteriolised capillary gases from the earlobe (but not from the finger) can provide an assessment of pH and PaCO2 that is almost identical to that obtained from an arterial sample. (160, 223, 294, 358, 359). In both acute and stable situations, the earlobe specimen gives a pO2 measurement which is 0.5 to 1 kPa (3.7 - 7.5 mm Hg) lower than the simultaneous arterial measurement with most of the divergence occurring at oxygen tensions above 8-10 kPa or 60-75 mm Hg. (223, 359) This means that most patients can be managed safely based on the pH and PCO2 levels measured from earlobe blood gases supplemented by oxygen saturation measured by a pulse oximeter. (223, 358) In critically ill patients, the initial specimen should be an arterial specimen to guarantee an accurate initial assessment but capillary gases are especially valuable for monitoring progress of the blood gases as a patient stabilises.

Patients who have had simultaneous arterial and earlobe samples rated the earlobe puncture procedure as being considerably less painful than arterial puncture. (295) However, the administration of local anaesthesia prior to arterial blood gas sampling produced a significant reduction in pain. (364) There is a very small risk of arterial damage from arterial puncture, especially if the radial site is used. Most reports of hand ischaemia have involved indwelling radial artery cannulae but the vessel could also be injured by needle puncture. (296) Therefore, the guideline recommends that arteriolised earlobe specimens should be used more widely than at present as a safer and less painful alternative to arterial blood gas sampling. However, the accuracy of earlobe samples in shock or hypotension is not known and it is recommended that arterial blood gases should be used in all case of shock or hypotension (systolic blood pressure below 90 mm Hg).

The technique of patient preparation, sample acquisition and sample processing for arteriolised capillary gases in complex and should only be undertaken by fully trained staff. Capillary gases are very vulnerable to errors in technique and should only be implemented for emergency use in units where staff has been fully trained in their use and where the quality of the technique is monitored constantly.

Draft Guideline updated 28-7-07
Use of arteriolised capillary blood gas measurements

- Patients find earlobe specimens less painful than arterial puncture without local anaesthesia. Level IIa

- Arteriolised earlobe blood gases will provide accurate information about PaCO2 and pH but do not provide an accurate measurement of PaO2 in stable patients or in acute COPD. Level IIa

- The earlobe specimen gives a pO2 measurement which is 0.5 to 1 kPa (4–7.5 mm Hg) lower than the simultaneous arterial measurement with greater divergence at oxygen levels above 8–10 kPa (60–75 mm Hg). Level IIa

- However, a combination of earlobe gases (to monitor pH and PCO2) and oximetry (to measure oxygen levels) will allow safe management of most COPD patients, even in emergency settings. Level IV

Recommendations:

19 For most patients who require blood gas sampling, either ABG or arteriolised Earlobe Blood Gases (ELBG) may be used but the PaO2 is less accurate in ELBG samples so oximetry should be monitored carefully if ELBG specimens are used. Grade B

20 For critically ill patients or those with shock or hypotension (systolic blood pressure below 90 mm Hg), the initial blood gas measurement should be obtained from an arterial specimen. Grade D

21 The technique of patient preparation, sample acquisition and sample processing for arteriolised capillary gases is complex and should only be undertaken by fully trained staff. Grade D

22 Local anaesthesia should be used for all arterial blood gas specimens except in emergencies or if the patient is unconscious or anaesthetised. Grade B

7.1.4 Transcutaneous oxygen assessments
Transcutaneous oxygen devices give different information from pulse oximetry. They are more sensitive to reduced perfusion and may be used to monitor tissue oxygenation in trauma patients but their use is beyond the scope of this guideline. (139)

7.2 Assessment of hypercapnia and acidosis

7.2.1 Clinical assessment
In patients with lung disease hypercapnia is usually accompanied by visible respiratory distress, but this will be absent when hypercapnia is a consequence of a reduction in minute ventilation. Patients may have a flushed face, a full and bounding pulse and muscle twitching together with the characteristic flap of the outstretched hands. In severe cases consciousness may be depressed and convulsions may occur. Gross hypercapnia usually occurs with profound hypoxaemia and it is therefore difficult to disentangle the direct effect of hypercapnia per se. Coma will usually occur when the PaCO2 is in the range 12 to 16 kPa (90 to 120 mm Hg). Survival has been seen following a PaCO2 of 67 kPa (500 mm Hg). [338]
The presence of hypercapnic respiratory failure can be anticipated in patients with severe exacerbations of COPD or other diseases such as severe neuro-muscular disorders. Carbon dioxide is a vasodilator so patients with hypercapnia may appear flushed with dilated peripheral veins and a bounding pulse. Cranial vasodilation may cause headache. Carbon dioxide in high concentrations has hypnotic effects and patients with hypercapnia may progress from drowsiness with flapping tremor to confusion to coma. (195, 201, 202, 203, 204, 248) A study of 127 episodes of acute respiratory acidosis showed that the best clinical predictors of respiratory acidosis were drowsiness (odds ratio 7.1), flushing (odds ratio 4.1), the presence of known COPD (odds ratio 3.3) and the presence of intercostal retraction (odds ratio 2.9). (20)

**Clinical signs of carbon dioxide retention**
- Vasodilation producing flushing and warm peripheries with dilated blood vessels (including retinal veins)
- Bounding pulse
- Drowsiness
- Flapping tremor
- Confusion
- Coma

### 7.2.2 Blood gases (arterial and arteriolar)  See section 7.1.3
Arterial or arteriolar earlobe capillary blood gases will give an accurate estimation of pH and PaCO2. (160, 223, 294). The blood gases will need to be repeated in 30 minutes to one hour in patients with significant hypercapnia or acidosis to monitor the response to therapy. Patients with COPD who remain acidic despite 30-60 minutes of standard therapy (including controlled low-dose oxygen therapy) are likely to need Non-Invasive Ventilation. (48)

### 7.2.3 Venous PCO2 sampling
It has been suggested that the venous PCO2 level can be used to screen for hypercapnia in patients with acute respiratory disease. A study of 196 paired samples of arterial and venous blood from patients with acute respiratory disease showed that the PCO2 in the venous sample was an average of 5.8 mm Hg (0.77 kPa) higher than the simultaneous arterial sample. (39). However, a venous PCO2 below 45 mm Hg (6 kPa) had 100% sensitivity for eliminating the risk of hypercapnia (arterial PCO2 above 6.0 kPa or 45 mm Hg) although the specificity was low at 57%. Therefore, for patients who are not at risk of metabolic acidosis, the presence of a satisfactory oxygen saturation measured by pulse oximetry and a venous PCO2 below 6 kPa (45 mm Hg) can exclude the possibility of arterial hypercapnia and may obviate the need for arterial blood gas measurements.

### 7.2.4 CO2 monitors and non-invasive assessments of hypercapnia
End-tidal CO2 monitors are used primarily to confirm tracheal intubation during anaesthesia, Intensive Care and for any patients requiring endotracheal intubation. They are considered the “gold standard” by the Royal College of Anaesthetists. The absence of any detectable CO2 output indicates a failed intubation. The management of intubated patients is outside the remit of this guideline.

End-tidal CO2 monitors are also useful in the management of cardiac arrest and circulatory collapse. Very low levels of CO2 excretion indicate very low (or absent) cardiac output and a low likelihood of survival. (288-290)
devices are also useful in the care of intubated patients in the Emergency Department because, through visualising a typical "box wave form", they can confirm that the tube is in the airway even in the absence of CO2 production during a cardiac arrest. The appearance of CO2 may be the first sign of spontaneous circulation. (368)

End-tidal CO2 measurements correlate poorly with arterial CO2 levels in patients with COPD, but they may be useful in some research studies of hyperventilation syndromes. However, these devices are inaccurate in patients with airways disease and those with a high respiratory rate so they should not be used in the management of patients with respiratory failure and they will not be discussed further in this guideline.

An exciting new possibility is the development of probes that can assess PCO2 as well as SpO2 from a single probe. Early studies indicate that such devices can be accurate in normal volunteers (119) Transcutaneous CO2 monitors are also being developed in association with transcutaneous oxygen monitors for use in patients with shock and critical illness. (139)
Section 8

Emergency oxygen use in hospital settings

8.1 Assessment and immediate management of breathless patients on arrival in hospital

Breathless patients may arrive in hospital directly (without prior assessment) or in ambulances where they will usually have been assessed by paramedics who may also have initiated emergency treatments including oxygen therapy. As discussed in section 7 of this Guideline, assessment, triage and resuscitation of critically ill patients must be undertaken in parallel with the initiation of oxygen therapy and specific treatment must be given for the underlying medical condition. All critically ill patients and all patients at risk of hypercapnic respiratory failure should be triaged as very urgent and should have blood gases taken on arrival in hospital. Furthermore, all seriously ill patients should be assessed by senior clinicians as early as possible. In many cases, this may involve liaison with intensive care specialists or with appropriate other specialists who can deal effectively with the patient’s major medical or surgical problems.

- Readers are referred to section 7.1.1 and to disease-specific guidelines for advice concerning the immediate assessment and management of seriously ill patients.
- Readers are referred to section 10 for advice concerning choice of oxygen delivery devices and systems.
- Readers are referred to tables 1-4 and charts 1 and 2 for a summary of the key elements of oxygen therapy in common medical emergencies.
- Remember to ask for senior advice or specialist advice early in the care of profoundly ill patients

8.2 Differences in management in hospital compared with a pre-hospital setting.

The immediate management of medical emergencies in hospital settings (prior to the availability of blood gas results) is similar in principle to management in the pre-hospital setting. The main priorities are to avoid harmful levels of hypoxaemia for all patients and to avoid harmful levels of hypercapnia for patients who are at risk of this complication. However, the amount of information available to the health-care professionals increases rapidly in the hospital environment. The hospital management is presented before the pre-hospital management because it represents the “ideal” management. This may also be achievable in some pre-hospital settings such as a well equipped primary care centre. However, in many pre-hospital settings, there will usually be less information available concerning a patient’s history and physiology and less equipment available to assess and treat the patient.

Some differences between hospital settings and pre-hospital settings.

- Pulse oximetry is almost always available in hospital at present. These guidelines also recommend that pulse oximetry must be available in all locations where emergency oxygen is used (9.1.)
- Blood gas results can be available within minutes of arrival in hospital
- Additional diagnostic information may be available from history, clinical examination, test results and from the patient’s hospital records
Additional equipment and resources are available (e.g. ability to ventilate)

Because of the universal availability of oximetry in hospitals, it is rare for the hospital medical team to have to administer oxygen on the basis that a patient “might be hypoxaemic”. However, initial “blind management” is sometimes necessary for patients with shock or with very poor peripheral circulation where a reliable pulse oximetry trace cannot be obtained. Arterial blood gases should be obtained as a matter of urgency in all such cases.

8.3 Which patients need oxygen therapy?
Supplementary oxygen therapy is required for all acutely hypoxaemic patients and for many other patients who are at risk of hypoxaemia, including patients with major trauma and shock. A majority of acutely breathless patients will require supplementary oxygen therapy but there are some situations such as acute hyperventilation or diabetic ketoacidosis where an apparently breathless patient will not benefit from oxygen therapy. There are some other clinical situations such as carbon monoxide poisoning where a patient may benefit from oxygen therapy despite lack of hypoxia because carbon monoxide binds more avidly than oxygen to the haemoglobin molecule.

Recommendations
- Oxygen saturation should be measured in all breathless and acutely ill patients. See recommendation 8
- Oxygen therapy should be given to hypoxaemic patients (see table 1) but most normoxaemic patients do not require oxygen therapy. (Patients on oxygen with SpO2 above 98% do not require oxygen therapy or may require a lower dose) Grade D.
- All patients with shock, major trauma, sepsis or other critical illness should be managed initially with high concentration oxygen therapy from a reservoir mask. The dose can be adjusted subsequently once the results of blood gas estimations are known and/or the patient is stable. Grade D

8.4 Which patients require blood gas measurements?
Blood gases should be measured as soon as possible in most emergency situations involving hypoxaemic patients (130) and are essential in patients who may develop type 2 respiratory failure (carbon dioxide retention with risk of respiratory acidosis). Blood gases should also be checked (and the clinical situation should be reviewed) if the oxygen saturation should fall by more than 3 percentage points, even if the saturation remains within the target range. For example, a fall from 98% to 93% might be due to a significant event such as a pulmonary embolus. In this situation, the saturation of 93% will not harm the patient but the patient will remain at serious risk until the pulmonary embolism is diagnosed and treated.

Blood gas measurements are not usually required for patients with no risk factors for hypercapnic respiratory failure and an oxygen saturation of 92% or above breathing air if aged 70 or above, or an oxygen saturation of 94% or above if aged below 70 unless the patient requires blood gas estimation for other reasons such as suspected metabolic acidosis or diabetic ketoacidosis. The British Thoracic Society asthma guideline recommends that arterial blood gas measurements need not be recorded in patients with acute asthma and an oxygen saturation above 92% and no life-threatening features. (297) Arterial blood gas sampling can be technically difficult, especially for poorly perfused patients and junior staff should ask for assistance from more senior staff in difficult cases.
Following initial clinical assessment and the availability of a pulse oximetry measurement, a decision can be made regarding the need for blood gas estimation within a few minutes of arrival in the hospital environment or if a previously stable patient develops breathlessness within a hospital environment. Oximetry will give no information concerning CO2 or pH levels and a normal pulse oximetry level may provide false reassurance in patients on oxygen therapy who may have unexpected hypercapnia and acidosis. However, careful clinical assessment supplemented by the use of oximetry will allow the setting of an appropriate oxygen saturation target for different groups of patients until blood gas results are available.

**Recommendation**

**25 Blood gases should be checked in the following situations.**

- Unexpected or inappropriate hypoxaemia (SpO2 below 94% in patients aged up to 70 breathing room air or oxygen or SpO2 below 92% in patients aged 70 and above) or any patient requiring oxygen to achieve the above targets. *(Allowance should be made for transient dips in saturation to 90% or less in normal subjects during sleep).* Grade D

- Deteriorating oxygen saturation or increasing breathlessness in a patient with previously stable hypoxaemia (e.g. severe COPD).

- Any previously stable patient who deteriorates and requires a significantly increased fraction of inspired oxygen (FIO2) to maintain a constant oxygen saturation. Grade D

- Any patient with risk factors for hypercapnic respiratory failure who develops acute breathlessness, deteriorating oxygen saturation, or drowsiness or other symptoms of CO2 retention. Grade D

- Breathless patients who are thought to be at risk of metabolic conditions such as diabetic ketoacidosis or metabolic acidosis due to renal failure. Grade D

- Any other evidence from the patient’s medical condition that would indicate that blood gas results would be useful in the patient’s management (e.g. unexpected sudden rise of several units in modified Early Warning Score or an unexpected fall in oxygen saturation of 3% or more, even if within the target range). Grade D

**8.5 Can arteriolised earlobe gases be used as a substitute for arterial blood gases?**

Readers are referred to section 7.1.3 for advice concerning when to use arterial blood gases and when to use arteriolised earlobe blood gases.

**8.6 Should oxygen be prescribed at a fixed “dose” or to achieve a target saturation?**

In the past, oxygen was prescribed at a fixed FIO2, or at a fixed flow rate via nasal cannulae, or variable performance face masks. However, several audits have shown that many (or most) patients do not receive the prescribed dose of oxygen. (55, 100, 180, 321-324) Furthermore, a patient’s oxygen requirement may vary over time so the prescribed oxygen dose may be too high or too low even a short time after the prescription was written. For this reason, it is recommended that oxygen should be prescribed to a target saturation range rather than prescribing a fixed dose of
oxygen or fraction of inspired oxygen. This is analogous to an insulin “Sliding scale” where the prescriber specifies a variable dose of insulin to achieve a target blood glucose range rather than prescribing a fixed dose of insulin. This will allow the appropriate Health-Care Professional, usually a doctor, nurse or physiotherapist, to adjust each patient’s dose of oxygen to achieve the safest oxygen saturation range for each patient.

The prescriber may indicate a starting dose, device or flow-rate but there needs to be an agreed system for adjusting the oxygen dose upwards or downwards according to a patient’s needs (see Charts 1 and 2; Sections 11.3.6, 11.3.7 and Chart 3 and Chart 4). As a patient improves, he or she is likely to require a lower FIO2 over a time period that will vary between patients. Most recovering patients will eventually require no supplemental oxygen. On the other hand, a deteriorating patient may need an increased dose of oxygen. This increase can be initiated by nursing staff or physiotherapists but the requirement for an increased dose of oxygen is an indication for urgent clinical reassessment of the patient (and repeat blood gas measurements in most instances).

- It is recommended that oxygen should be prescribed to a target saturation range rather than prescribing a fixed dose of oxygen or fraction of inspired oxygen. See recommendations 1, 2, 4 and 5

8.7 What should be the target oxygen saturation range for patients receiving supplementary oxygen?

8.7.1 Oxygen saturation target range for most patients: As discussed in sections 4 to 6 of this guideline, there is no evidence of benefit from above-normal oxygen saturation in most medical emergencies and there is evidence that excessive doses of oxygen can have adverse effects, even in some patients who are not at risk of hypercapnic respiratory failure. A target oxygen saturation range of 94-98% for patients below 70 and 92-98% for those aged 70 or more will achieve normal or near-normal oxygen saturation for most patients who are not at risk of hypercapnic respiratory failure. Furthermore, the suggested lower limit of 92% allows a margin of error in the oximeter measurement, thus minimizing the risk of any patient being allowed to desaturate below 90% due to inaccurate oximetry.

8.7.2. Oxygen requirements for specific groups of patients.

- Patients with critical illness requiring high dose oxygen therapy are discussed in section 8.10
- Patients with medical emergencies which frequently cause breathlessness and hypoxia are discussed in section 8.11
- Patients with COPD and other conditions that may predispose to type 2 Respiratory Failure are discussed in section 8.12
- Medical emergencies for which oxygen is commonly given at present but is not actually indicated unless the patient is hypoxaemic are discussed in section 8.13

8.8 Importance of blood gas measurements in guiding oxygen therapy. As soon as blood gas measurements are available, a patient’s further treatment can be guided by the results of this test. For patients with a normal or low PaCO2 and no risk factors for hypercapnic respiratory failure, it is safe to aim at an oxygen saturation in the normal range (94-98% if aged below 70 and 92-98% if aged 70 and above). For patients with a raised PaCO2, a lower oxygen saturation is indicated (88-92%), especially if the patient is acidic. Non-Invasive Ventilation is recommended for patients with COPD who have hypercapnia and a pH below 7.35.
8.9 What should be the initial choice of oxygen delivery system in hospital settings?
The technical and practical aspects of different oxygen delivery systems are discussed in section 10. For major trauma cases and for severely hypoxaemic patients without risk factors for hypercapnic respiratory failure, a non-re-breathe mask (reservoir mask) at 10-15 l/minute is the suggested first choice. The delivery system and FIO2 may be adjusted later to a lower dose of oxygen as a patient improves or towards supported ventilation if the patient deteriorates. The majority of patients with modest hypoxaemia can be treated with nasal cannulae or simple face mask at a flow rate which is adjusted to maintain the oxygen saturation in the target range for their specific clinical presentation. Chart 2 shows a suggested scheme that allows the oxygen level to be adjusted upwards or downwards in gradual increments depending on a patient’s clinical progress. (see also sections 11.3.6 and 11.3.7) Venturi masks are recommended for low-dose oxygen therapy because they deliver a more reliable oxygen concentration than nasal cannulae or variable flow masks and they can be combined with a humidifier system when necessary. The mask and/or flow should be rapidly changed if the initial choice does not achieve the target saturation.

8.9.1 Devices used in emergency oxygen therapy in hospitals
- High concentration oxygen from reservoir mask (10-15 l/min) or resuscitation mask for critical illness or severe hypoxaemia or during resuscitation
- Nasal cannulae (2-6 l/min) or simple face masks (5-10 l/min) for medium-dose oxygen therapy
- 24% Venturi Mask at 2l/min or 28% Venturi masks at 4 l/min for patients at risk of hypercapnic respiratory failure (change to nasal cannulae at 1-2 l/min when the patient has stabilized)
- Tracheostomy masks for patients with prior tracheostomy (adjust flow to achieve desired saturation)

8.10 Recommended oxygen therapy for major medical emergencies and critical illness. (see also Table 1)
There are a number of major medical emergencies where patients are very likely to suffer from hypoxia. High-dose oxygen therapy from a reservoir mask at 10-15 l/min is recommended in the initial management of all such patients prior to stabilization in a critical care area or high dependency unit. Following stabilization, the dose of oxygen can be titrated downwards to maintain a target saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above. It is recommended that patients with COPD or other risk factors for hypercapnia who develop a critical illness should be treated by Emergency Services in the same manner as other critically ill patients until urgent blood gas results become available because the primary issue is the critical illness. Critically ill patients with hypercapnia, hypoxia and acidosis will require immediate assessment by Intensive Care teams and will usually require intubation and mechanical ventilation.

8.10.1 Cardiac Arrest and other conditions requiring Cardio-Pulmonary Resuscitation (CPR).
The 2005 guideline for Adult Advanced Life Support issued by Resuscitation Council UK recommends the use of non re-breathing reservoir masks (or 100% oxygen via a self-inflating bag-mask system) to deliver the highest
possible inspired oxygen level to patients requiring resuscitation. (299). The present guideline endorses these proposals during the period of resuscitation. Subsequent management will depend on the underlying condition and the patient’s degree of recovery. Some patients will require invasive ventilation following CPR but others will recover rapidly and an oxygen saturation target of 94-98% (if aged below 70) and 92-98% (if aged 70 and above) is recommended during the convalescent period.

**Recommendation (See Table 1 on page 3)**

26 Use high dose oxygen from a reservoir mask at 15 l/min or resuscitation mask during resuscitation. Grade D

8.10.2 Critically ill patients including major trauma, shock and major sepsis.

There is evidence that early intervention to normalise oxygen delivery to the tissues using volume expansion and vasoactive agents is beneficial in the management of critically ill patients with shock or sepsis but there is no evidence of benefit from attempts to achieve supra-normal oxygen delivery. (2, 57, 75, 78, 108, 115, 136, 219) In fact, there is evidence that hyperoxia can cause a paradoxical decrease in whole body oxygen consumption in critically ill patients (379) and it has been demonstrated recently that hyperoxia can impair oxygen delivery in septic patients. (380) Most such patients are at risk of multi-organ failure and therefore require Intensive Care assessment as a matter of urgency. Critical Care consensus guidelines set 90% saturation as the minimum level below which oxygen saturation should not be allowed to fall and the Surviving Sepsis Campaign guideline recommends a target arterial oxygen saturation of 88-95% for patients with sepsis. (363, 367, 219) However, these recommendations are based on directly measured arterial oxygen saturations in Critical Care settings with intensive levels of nursing and monitoring. The present Guideline recommends a slightly higher target saturation range prior to the transfer of these seriously ill patients to Critical Care facilities.

For most critically ill or severely hypoxaemic patients, initial oxygen therapy should involve the use of a reservoir mask, aiming at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above. If the patient has concomitant COPD or other risk factors for hypercapnic respiratory failure, the initial saturation target should be 92-98% pending the results of blood gas estimations and assessment by Intensive Care specialists. If critically ill COPD patients have hypercapnia and acidosis, the correction of hypoxaemia must be balanced against the risks of respiratory acidosis and ventilatory support using Non-Invasive or Invasive ventilation should be considered.

It is also recognised that many patients with long bone fractures may develop hypoxaemia even in the absence of injury to the airway or chest (possibly due to opiate treatment and fat embolism) and they should be monitored with oximetry and given oxygen if necessary. (45, 46, 129, 145). These patients, if not critically ill, should have a target oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above or 88-92% if they have co-existing COPD or other risk factors for hypercapnic respiratory failure.

**Recommendation (See Table 1 on page 3)**

27 In critical illness, including major trauma and sepsis, initiate treatment with a reservoir mask at 10-15 l/min and aim at a saturation range of 94-98% if aged below 70 and 92-98% if aged 70 and above. Grade D
8.10.3 Near-drowning
Survivors of near-drowning may have suffered inhalation of fresh or sea water into the lungs and may become hypoxaemic. Supplemental oxygen should be given to all patients with saturation below 92%, aiming at a target saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above.

Recommendation (See Table 1 on page 3)

28 In cases of near-drowning, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above. Grade D

8.10.4 Anaphylaxis
Patients with anaphylaxis are likely to suffer from tissue hypoxia due to a combination of upper and/or lower airway obstruction together with hypotension. In addition to specific treatment of these problems, the Resuscitation Council UK recommends high-flow oxygen (10-15 L/minute- presumably by Reservoir mask if available) for patients with anaphylaxis. (298) The present guideline would endorse this practice in the immediate management of anaphylaxis followed by a target saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above once the patient’s condition has stabilised.

Recommendation (See Table 1 on page 3)

29 In anaphylaxis, initiate treatment with a reservoir mask at 10-15 and aim at a saturation range of 94-98% if aged below 70 and 92-98% if aged 70 and above. Grade D

8.10.5 Major pulmonary haemorrhage or massive haemoptysis
Major pulmonary haemorrhage and massive haemoptysis can occur for a large number of reasons ranging from acute pulmonary vasculitis to erosion of a blood vessel by a lung tumour. In addition to specific treatment of the causative condition, most such patients require supplementary oxygen treatment. A target saturation range of 94-98% is recommended if aged below 70 and 92-98% if aged 70 and above. Treatment should be initiated with 100% oxygen via a reservoir mask and subsequently adjusted according to Chart 2 to maintain a saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above pending the results of blood gas measurements.

Recommendation (See Table 1 on page 3)

30 In pulmonary haemorrhage, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above. Grade D

8.10.6 Major head injury
Patients with major head injury are at risk of hypoxaemia and hypercapnia. They require urgent assessment and maintenance of airway patency, either through positioning, simple adjuncts or early intubation and ventilation to avoid further brain injury due to brain oedema which may be aggravated by hypercapnia. These patients should be referred immediately to appropriately trained specialists, even if this requires an inter-hospital transfer. Initial therapy should include high concentration oxygen via reservoir mask pending availability of satisfactory blood gases or until the airway is secured by intubation. Although hypoxaemia is common in head injured patients, the relative
contribution of hypoxaemia to outcome is not yet established. (30, 44, 47, 131, 226, 227) All authors agree that hypoxaemia should be corrected but a recent review of the literature concluded that there is no evidence of clinical benefit from hyperoxia in brain-injured patients. (220) There are no UK guidelines for oxygen therapy in the immediate post-head injury phase but US Guidelines recommend maintaining an oxygen saturation above 90% for patients with acute brain injury (315). The present guideline advises giving supplementary oxygen if required to maintain an oxygen saturation in the range of 94-98% if aged below 70 and 92-98% if aged 70 and above.

Recommendation  (See Table 1 on page 3 )

31 In cases of major head injury, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above. Initial therapy should involve high concentration oxygen from a reservoir mask at 10-15 l/min pending availability of satisfactory blood gases or until the airway is secured by intubation. Grade D

8.10.7 Carbon monoxide (CO) poisoning.
Patients with carbon monoxide poisoning have a normal level of dissolved oxygen in the blood (PaO2) but a greatly reduced level of oxygen bound to haemoglobin because this has been displaced by carbon monoxide. (318) Pulse oximetry cannot screen for carbon monoxide exposure, as it does not differentiate carboxyhaemoglobin from oxyhaemoglobin and blood gas measurements will show a normal blood oxygen tension (PaO2) in these patients. The blood carboxyhaemoglobin level must be measured to assess the degree of carbon monoxide poisoning. The half-life of COHb in a patient breathing room air is approximately 300 minutes; this decreases to 90 minutes with high-flow oxygen via a non-re-breathing mask. Therefore, the most important treatment for a patient with CO poisoning is to give high dose oxygen via a reservoir mask. Comatose patients or those with severe mental impairment should be intubated and ventilated with 100% oxygen. The role of hyperbaric oxygen remains controversial. A 2005 Cochrane review concluded that existing randomized trials did not establish whether the administration of hyperbaric oxygen to patients with carbon monoxide poisoning reduced the incidence of adverse neurologic outcomes. (376) However, a randomized trial published in 2007 has suggested that patients with loss of consciousness or high carboxyhaemoglobin levels may have less cognitive sequelae if given hyperbaric oxygen. (377)

Recommendation  (See Table 1 on page 3 )

32 In cases of carbon monoxide poisoning, an apparently "normal" oximetry reading may be produced by carboxy-haemoglobin, therefore aim at an oxygen saturation of 100% and use a reservoir mask at 15 l/min irrespective of the oximeter reading and PaO2. Grade C
The initial oxygen therapy is a reservoir mask at 15 l/min.

Once stable, reduce the oxygen dose and aim for target saturation range of 94-98% if aged <70 and 92-98% saturation if aged ≥70

Patients with risk factors for hypercapnia who develop critical illness should have the same initial target saturations as other critically ill patients pending the results of blood gas results after which these patients may need controlled oxygen therapy or supported ventilation if there is severe hypoxia and/or hypercapnia.

<table>
<thead>
<tr>
<th>Additional Comments</th>
<th>Recommendation number and grade</th>
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<tbody>
<tr>
<td>Cardiac Arrest or Resuscitation</td>
<td>Use bag-mask during active resuscitation. Aim for maximum possible oxygen saturation. 26 Grade D</td>
</tr>
<tr>
<td>Shock</td>
<td>Once stable, reduce the oxygen dose and aim for target saturation range of 94-98% if aged &lt;70 and 92-98% saturation if aged ≥70 27 Grade D</td>
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<tr>
<td>Sepsis</td>
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<td>Major Trauma</td>
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<td>Major Head Injury</td>
<td>Early intubation and ventilation if comatose. 31 Grade D</td>
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<tr>
<td>Carbon Monoxide Poisoning</td>
<td>Aim for 100% saturation. Oximeter reading and blood gas PaO2 are misleading. Check carboxyhaemoglobin levels. 32 Grade C</td>
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</table>
8.11 Recommended oxygen therapy for medical emergencies which frequently cause hypoxaemia. (see also Table 2)

Patients who present with acute medical emergencies who are not critically ill or grossly hypoxic and can be treated with medium-dose oxygen therapy from nasal cannulae or a simple face mask with a target saturation range of 94-98% if aged below 70 and 92-98% if aged 70 and above. Some of these patients (e.g. patients with pneumonia) may subsequently deteriorate, requiring high-dose oxygen from a reservoir mask or requiring respiratory support such as invasive ventilation. Others may turn out to have an additional diagnosis of COPD or neuromuscular disease with risk of hypercapnic respiratory failure and they should be managed with a Venturi mask or 2 litres of oxygen via nasal cannulae, aiming at a target saturation of 88-92%. There are no published trials supporting the use of oxygen to relieve breathlessness in non-hypoxaemic patients and there is evidence from randomised studies that oxygen does not relieve breathlessness compared with air in non-hypoxaemic COPD patients who are breathless following exertion. (374)

8.11.1 Patients with acute onset of hypoxaemia of unknown cause with no pre-existing respiratory disorders or risk factors.

It is common for breathless and hypoxaemic patients to have no firm diagnosis at the time of presentation. For most acutely hypoxaemic patients whose medical problem is not yet diagnosed, an oxygen saturation range of 94.98% if aged below 70 and 92-98% if aged 70 and above will avoid the potential hazards associated with hypoxaemia or hyperoxia (see sections 4-6 and table 1). Aiming for an oxygen saturation in the normal range (rather than an abnormally high oxygen level) will also have the effect of allowing the lowest effective FIO2 to be used, thus avoiding risks such as absorption atelectasis and ventilation-perfusion mis-match that may be associated with the use of very high fractions of inspired oxygen (see sections 5 and 6). The priority for such patients is to make a specific diagnosis as early as possible and to institute specific treatment for the underlying condition. Early blood gas measurement is mandatory in the management of patients with sudden unexplained hypoxaemia.

Recommendations (See Table 2 on page 4)

33 For acutely breathless patients not at risk of hypercapnic respiratory failure who have saturations below 90%, treatment should be commenced with a reservoir mask at 10-15 l/min in the first instance. The oxygen dose can be adjusted downwards (using nasal cannulae or simple face mask) to maintain a target saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above once the patient has stabilised. Grade D

34 In all other cases without risk factors for hypercapnic respiratory failure, treatment should be commenced with nasal cannulae (or simple face mask if cannulae are not tolerated or not effective) with the flow rate adjusted to achieve a saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above. Grade D

35 If medium dose therapy with nasal cannulae or simple face mask does not achieve the desired saturation, change to a reservoir mask and seek senior or specialist advice. Grade D

8.11.2 Acute Asthma

The BTS-SIGN Guideline for management of acute asthma recommends that the oxygen saturation should be maintained above 92%. (297) The present guideline suggests a target saturation of 94-98% for patients aged below 70 and 92-98% if aged 70 and above. This recommendation is consistent with the BTS SIGN document for those
aged 70 and above but the lower limit of 94% in this guideline for younger adults is recommended in order to maintain consistency throughout the guideline. The rationale for this approach is explained in 6.5 and in recommendation 3. In essence a drop of oxygen saturation below 94% may indicate deterioration in the younger individual and should prompt a further assessment. Supplementary oxygen should be started using nasal cannulae at 2-4 l/min or simple face mask at 5 L/min or 35-40% Venturi mask and adjusted as necessary to maintain a saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above. (105) Oxygen should not be withheld from patients with severe asthma because of concerns about possible hypercapnia although there is some evidence that this phenomenon does occur.(97) Hypercapnia in acute asthma indicates a near-fatal attack and indicates the need for consideration of Intensive Care admission and ventilation. (297)

Recommendation  (See Table 2 on page 4)

36 In acute asthma, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above. Grade C

8.11.3 Pneumonia
The British Thoracic Society Guideline for pneumonia recommends aiming at an oxygen saturation above 92% and PaO2 above 8 kPa (60mm Hg) in uncomplicated pneumonia with appropriate adjustments for patients with COPD, guided by blood gas measurements. (300). The present guideline endorses these principles. For internal consistency, a saturation range of 94-98% if aged below 70 and 92-98% if aged 70 and above is recommended for patients with uncomplicated pneumonia and it is recommended that patients with COPD complicated by pneumonia should be managed in accordance with the COPD section of the present guideline. For the practical reasons discussed in section 8.11.2, a slightly narrower target range is suggested for patients aged below 70.

Recommendation  (See Table 2 on page 4)

37 In cases of pneumonia, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above. Grade D

8.11.4 Lung cancer and other cancers with pulmonary involvement
Most cancer patients who present with acute breathlessness have got a specific causative factor such as a pleural effusion, pneumonia, COPD, anaemia or collapse of a lobe or of the left or right lung. (61, 80) A small double blind trial has shown that hypoxaemic patients with advanced cancer had reduced dyspnoea breathing oxygen compared with air (Ref 58). However, a systematic review of oxygen and airflow on the relief of dyspnoea at rest in patients with advanced disease of any cause found low grade scientific evidence that oxygen and airflow improve dyspnoea in some patients with advanced disease at rest. (258) Morphine and midazolam are also of benefit in the treatment of breathlessness due to advanced cancer. (68, 80) In addition to specific management of the causative factor, oxygen should be given to maintain a saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above except for patients with co-existing COPD who should be treated in accordance with the COPD section of this guideline.

Monitoring of oxygen saturation may not be necessary in terminal palliative care.

Recommendations  (See Table 2 on page 4)
In breathlessness due to lung cancer, oxygen therapy may be beneficial and a trial of oxygen therapy is recommended. Aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above unless there is co-existing COPD. However, monitoring of oxygen saturation may not be necessary in terminal palliative care. Grade D

8.11.5 Post-operative breathlessness or hypoxaemia on general surgical wards
These guidelines do not cover immediate post-operative care in Post-Anaesthetic Recovery Units, High Dependency Units or Intensive Care Units. Some recent trials have shown a reduced incidence of wound infection when high-dose oxygen was given peri-operatively to patients having bowel surgery but not general surgery. (278-280) This planned use of oxygen post-operatively is also outside the scope of this guideline.

There is some controversy about the use of “routine” supplemental oxygen post-operatively and no good evidence supporting such a policy. (67, 221, 242, 246, 249) The SIGN Guideline on post-operative care recommends supplemental oxygen therapy for certain high-risk groups such as those with coronary artery disease, obesity, thoracic and upper abdominal surgery but acknowledges lack of evidence to support these suggestions and does not specify an oxygen dose or target saturation for such patients. (242) This SIGN Guideline recommends maintaining an oxygen saturation above 92% for post-operative patients which fits well with the suggested target saturation in the present guideline of 94-98% if aged below 70 and 92-98% if aged 70 and above for most patients who require supplementary oxygen therapy.

Patients on general surgical wards can develop sudden breathlessness or hypoxaemia due to a variety of post-operative complications such as pneumonia, pulmonary embolism, opiate analgesia, atelectasis etc. The use of oxygen for specific post-operative complications such as pneumonia should follow the guidance for each condition (for most patients, the target will be 94-98% if aged below 70 and 92-98% if aged 70 and above). Special care must be taken in cases of COPD and other risk factors for hypercapnic respiratory failure. Management of these cases can be enhanced by early specialist referral or the input of expert assistance from “ICU Outreach Teams”. These cases should be identified as being at risk during pre-operative assessment and a target saturation of 88-92% is suggested, pending the availability of blood gas results.

Recommendations  (See Table 2 on page 4)

39 For post-operative surgical patients, aim at a saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above or 88-92% if at risk of hypercapnic respiratory failure. Grade D

- For post-operative surgical patients with COPD or other risk factors for hypercapnic respiratory failure, aim at a saturation of 88-92% pending results of blood gas analysis (if the PaCO2 is normal, adjust target range to 94-98% if aged below 70 and 92-98% if aged 70 and above and repeat blood gases after one hour) See recommendations 53-54 and chart 1.

8.11.6 Acute heart failure
Most patients with acute heart failure are breathless, usually due to pulmonary oedema or low cardiac output, especially if cardiogenic shock if present. The patho-physiology of oxygen transport in cardiogenic shock is discussed in detail in a paper by Creamer and colleagues. (101) It has been shown in an animal model that the
ventilatory failure of cardiogenic shock may be due to an impairment of the contractile process of the respiratory muscles. (216)

In addition to specific treatment for heart failure, patients should be given supplementary oxygen to maintain a saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above. This is consistent with the European Society of Cardiology Task Force and European Society of Intensive Care recommendation that patients with acute heart failure should receive oxygen to maintain SpO2 of 92-96% (236). It is reasonable to initiate therapy with 40% or 60% oxygen for hypoxaemic patients with heart failure, followed by upward or downward adjustment to maintain saturation in the desired range. Patients with marked hypoxaemia (saturation below 85%) should be treated with a reservoir mask initially and patients with co-existing COPD will require a lower target saturation of 88-92% pending the availability of blood gas results.

In hospital settings, patients with acute pulmonary oedema may benefit from CPAP (continuous positive airway pressure) and from non-invasive ventilatory support. (74, 169, 307)

Recommendation (See Table 2)

40 In acute heart failure, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above or 88-92% if the patient is at risk of hypercapnic respiratory failure. Grade D

- Consider treatment with CPAP if there is hypoxaemia and treatment with NIV (BiPAP) if there is co-existent hypercapnia

8.11.7 Pulmonary embolism
Most patients with suspected pulmonary embolism have got a normal oxygen saturation and the main focus of treatment is to reach a specific diagnosis and to commence anti-coagulant treatment. These patients do not require oxygen therapy unless there is hypoxaemia. In these cases, the lowest dose of oxygen that will achieve a target saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above is recommended. However, patients with massive or multiple pulmonary embolism may be profoundly hypoxaemic and should initially be given high-flow oxygen via a reservoir mask to achieve an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above pending definitive therapy such as thrombolysis. It has been suggested that the blood oxygen saturation may under-estimate the severity of pulmonary artery obstruction in acute pulmonary embolism if shock is present. (217)

Recommendation (See Table 2)

41 In pulmonary embolism, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above or 88-92% if the patient is at risk of hypercapnic respiratory failure. Grade D

8.11.8 Pleural effusion
If a pleural effusion is causing significant breathlessness, the most effective therapy is to drain the effusion (but not too quickly in view of the risk of re-expansion pulmonary oedema). Hypoxaemic patients with pleural effusions are likely to benefit from supplementary oxygen therapy. The British Thoracic Society Guidelines for management of Pleural Effusions do not give any specific advice concerning oxygen therapy but it seems reasonable to give
supplementary oxygen to hypoxaemic patients to maintain a saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above.

Recommendation (See Table 2)

42 In pleural effusion, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above or 88-92% if the patient is at risk of hypercapnic respiratory failure. Grade D

8.11.9 Pneumothorax
As with pleural effusions, patients with a large pneumothorax may be breathless and hypoxaemic and may require supplementary oxygen for symptom relief pending definitive treatment by aspiration or drainage. However, high concentration inhaled oxygen can also increase the rate of re-absorption of air from a pneumothorax up to four-fold. (283) For this reason, the BTS Guideline on management of pneumothorax recommends the use of 100% oxygen (reservoir mask) in all non-COPD patients who require hospital admission for observation due to a moderate-sized pneumothorax that does not require drainage. (284) Once a pneumothorax is drained or aspirated successfully, the patient should not require oxygen therapy unless there is additional pathology such as pneumonia, asthma or COPD requiring specific treatment.

Recommendations (See Table 2)

43 In most cases of pneumothorax, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above or 88-92% if the patient is at risk of hypercapnic respiratory failure. Grade D

44 In patients having hospital observation without drainage, the use of high dose oxygen (15 litre flow rate via reservoir mask at 15 l/min) is recommended. Grade C

8.11.10 Deterioration of fibrotic lung conditions and other conditions involving parenchymal lung disease or alveolitis.
It is recognised that patients with fibrosing lung conditions such as Idiopathic Pulmonary Fibrosis may have acute deteriorations or exacerbations, often during intercurrent chest infections. Other patients may present acutely with breathlessness due to extrinsic allergic alveolitis, sarcoidosis or other types of parenchymal lung disorders. These patients often have high degree of ventilation-perfusion mis-match and a requirement for high oxygen concentrations to achieve satisfactory blood gases and they are not at risk of hypercapnia. It is recommended that treatment is started with 60% oxygen from a Venturi mask or 6 litres per minute via nasal cannulae if the patient can tolerate a high nasal flow rate. The oxygen level should be adjusted upwards or downwards to maintain an oxygen saturation in the range of 94-98% if aged below 70 and 92-98% if aged 70 and above but this level may not be achievable or only achievable with a Reservoir mask. Patients with end-stage pulmonary fibrosis are rarely suitable for invasive or non-invasive ventilation because of the progressive nature of the condition.

Recommendation (See Table 2)

45 In acute deterioration of pulmonary fibrosis or other parenchymal lung diseases, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above or the highest possible if these targets cannot be achieved. Grade D
8.11.11 Breathlessness due to severe anaemia.
If breathlessness is due to severe anaemia, the specific treatment is blood transfusion. Studies by Canadian researchers in the late 1990s have shown that Hb levels of 70g/l (7 g/dl) were as safe as higher levels and may produce fewer complications in the critically ill. (293) However, this study was conducted using non-leukocyte depleted blood and it is possible that some of the infective complications in the group who were given more transfusions might have been avoided by the use of leukocyte-depleted blood. Therefore, the optimal transfusion target for critically ill patients remains the subject of ongoing discussion amongst experts in critical care medicine (section 6.11). Giving oxygen to elevate an already normal oxygen saturation will have very little effect on the oxygen-carrying power of the blood but it is reasonable to administer supplemental oxygen to maintain a saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above (if the saturation is below these levels breathing air of if breathlessness is a very prominent symptom).

Recommendations (See Table 2)

46 In anaemia, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above or 88-92% if the patient is at risk of hypercapnic respiratory failure. Grade D

47 Give packed red cells if the haemoglobin level falls below 70 g/L (7 g/dl) Grade B

8.11.12 Sickle cell crisis
Patients with Sickle cell disease frequently present with an "acute chest syndrome" comprising of breathlessness and chest pain and fever with pulmonary infiltrates on the chest radiograph. The exact causes and mechanisms are not well understood but oxygen should be given to avoid further intravascular sickling. There are no randomised studies but it is recommended that the oxygen saturation should be maintained at 94-98% if aged below 70 and 92-98% if aged 70 and above.

Recommendation (See Table 2)

48 In Sickle cell crisis, and acute chest syndrome, aim for an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above. Grade D
Table 2

Serious illnesses requiring moderate levels of supplemental oxygen if the patient is hypoxaemic  *Section 8.11*

The initial oxygen therapy is nasal cannulae at 2-6 l/min or simple face mask at 5-10 l/min unless stated otherwise.

**Recommended initial oxygen saturation target range is 94-98% if aged <70 and 92-98% saturation if aged ≥70**

Change to reservoir mask if the desired saturation range cannot be maintained with nasal cannulae or simple face mask (and ensure that the patient is assessed by senior medical staff)

If these patients have co-existing COPD or other risk factors for hypercapnic respiratory failure, aim at a saturation of 88-92% pending blood gas results but adjust to 92-98% if the PaCO2 is normal (unless there is a history of previous hypercapnic respiratory failure) and recheck blood gases after one hour.

<table>
<thead>
<tr>
<th>Illness</th>
<th>Additional Comments</th>
<th>Recommendation number and grade</th>
</tr>
</thead>
</table>
| Acute hypoxaemia - cause not yet diagnosed | Reservoir mask at 10-15 l/min if initial SpO2 below 90%, otherwise nasal cannulae or simple face mask  
*Patients requiring reservoir mask therapy need urgent clinical assessment by senior staff.* | 33-35  
Grade D |
| Acute Asthma                      |                                                                                      | 36  Grade C                     |
| Pneumonia                         |                                                                                      | 37  Grade C                     |
| Lung Cancer                       |                                                                                      | 38  Grade C                     |
| Post-operative Breathlessness     | Management depends on underlying cause.                                               | 39  Grade D                     |
| Acute Heart Failure               |                                                                                      | 40  Grade D                     |
| Pulmonary Embolism                | Most patients with minor pulmonary embolism are not hypoxaemic and do not require oxygen therapy. | 41  Grade D                     |
| Pleural Effusions                 | Additionally, treat by draining the effusion.                                        | 42  Grade D                     |
| Pneumothorax                      | Reservoir mask at 10-15 l/min if admitted for observation. Aim at 100% saturation. 100% oxygen accelerates clearance of pneumothorax if drainage is not required. Needs aspiration or drainage if the patient is hypoxaemic. | 43-44  
Grade C and Grade D |
| Deterioration of lung fibrosis or other interstitial lung disease | Reservoir mask at 10-15 l/min if initial SpO2 below 90%, otherwise nasal cannulae or Simple face mask | 45  Grade D                     |
| Severe Anaemia                    | The main issue is to correct the anaemia. Most anaemic patients do not require oxygen therapy. | 46-47  
Grade B and Grade D |
| Sickle cell crisis | Requires oxygen only if hypoxaemic. | 48 Grade D |
8.12 Recommended oxygen therapy for patients who may be vulnerable to medium or high doses of oxygen (see also Table 3)

COPD is the best known condition that can predispose to hypercapnic (Type II) respiratory failure, especially if the blood oxygen level is elevated above 10 kPa or 75 mm Hg. (48, 373). However, there are a number of other conditions which can render patients vulnerable to hypercapnic respiratory failure. The emphasis for such patients is to avoid clinically harmful levels of hypoxia or hypercapnia by giving carefully-titrated oxygen therapy or, if necessary, by supporting the patient with the use of non-invasive or invasive mechanical ventilation.

Non-COPD patients at risk of hypercapnic respiratory failure.
- Severe kyphoscoliosis or severe ankylosing spondylitis.
- Severe lung scarring from old TB (especially with thoracoplasty).
- Morbid obesity (Body Mass Index > 40)
- Musculoskeletal disorders with respiratory muscle weakness, especially if on home ventilation.
- Overdose of opiates, benzodiazepines or other respiratory depressant drugs.

8.12.1 COPD exacerbations
There is an extensive literature documenting the effects of high dose oxygen therapy in acute COPD. (24, 38, 48, 62, 64, 83, 103, 133, 142, 187-200, 208, 209, 373) These reports show that the administration of supplemental oxygen to patients with exacerbated COPD causes a rise in PaCO2 for reasons summarised in section 6.6 and the literature is summarised in detail in the review by Murphy et al. (248) There is very little literature describing the effects of oxygen therapy in the other conditions listed above but they are recognised to be at risk of hypercapnic respiratory failure and should be treated in a manner analogous to patients with COPD.

Some patients with previous hypercapnic respiratory failure will have alert cards or an entry in their electronic record to alert the emergency team to the optimal dose of oxygen required during the patient’s previous hospital admissions. (See section 10 of this Guideline). In the absence of such information, it is suggested that a target of 88-92% should be set initially and, if necessary, modified later, based on blood gas results. It is sometimes appropriate to aim at a lower saturation such as 85-90% either based on previous experience with an individual patient or based on initial blood gas results that show significant respiratory acidosis. However, it has been shown that COPD patients with a pH reading below 7.35 despite controlled oxygen therapy are more likely to die and more likely to meet criteria for intubation and ventilation. (38, 48, 142) The best management strategy for persistently acidic COPD patients is a trial of non-invasive ventilation with supplementary oxygen therapy. (48, 325, 360) One of these report (329) also demonstrated that patients with a high arterial oxygen tension on arrival in hospital (> 10.0 kPa or 75 mm Hg) were more likely to meet criteria for ventilation and the severity of acidosis was related to high arterial oxygen tensions. (48) This report was supported by the recent work of Joosten et al which showed that a PaO2 above 74.5 mm Hg (10 kPa) in acute COPD was associated with an increased likelihood of admission to HDU, increased need for NIV and greater length of stay. (373) Unfortunately, many clinical studies have shown that patients with COPD are frequently given very high doses of oxygen, either because of mis-diagnosis or because the risks of hyperoxia in COPD patients have been overlooked. (24, 38, 48, 373) Many patients with COPD are unaware of the diagnosis or mislabelled as having asthma – see section 9.5
Recommendations

49 Patients over 50 years of age who are long-term smokers with a history of exertional breathlessness and no other known cause of breathlessness should be treated as if having COPD for the purposes of this guideline. Grade D

50 Patients with a significant likelihood of severe COPD or other illness that may cause hypercapnic respiratory failure should be triaged as very urgent (Orange Status) on arrival in hospital Emergency Departments (and blood gases should be taken on arrival). Grade D

51 Prior to availability of blood gases, use a 24% Venturi mask at 2 l/min or 28% Venturi mask at 4 l/min and aim for an oxygen saturation of 88-92% for patients with risk factors for hypercapnia but no prior history of type 2 respiratory failure. Grade D

- Patients with a respiratory rate above 30 breaths per minute should have the flow rate set to 50% above the minimum flow rate specified on the Venturi mask and/or packaging. See recommendation 93

52 Aim at a pre-specified target saturation range (if available) in patients with a history of previous respiratory acidosis. In many cases, the ideal target saturation will be specified on patient’s alert card. If no information is available, aim at a saturation of 88-92% pending blood gas results. Grade D

- Patients with previous hypercapnic respiratory failure should have a personalized "Oxygen Alert Card" and this information should be available to primary care staff, ambulance staff and hospital staff. See recommendations 79-82

53 If, following blood gases the pH and PCO2 are normal, aim for oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above unless there is a history of previous hypercapnic respiratory failure. Grade D

54 Recheck blood gases after one hour for all patients with COPD or other risk factors for hypercapnic respiratory failure even if the initial PaCO2 measurement was normal. Grade D

55 If the PaCO2 is raised but pH is satisfactory, the patient has probably got long-standing hypercapnia; maintain target range of 88-92% for these patients. Blood gases should be repeated at one hour to check for rising PaCO2 or falling pH. Grade D

56 If the patient is hypercapnic (PaCO2 > 6.0 kPa or 45 mm Hg) and acidotic (pH < 7.35) consider non-invasive ventilation, especially if the acidosis has persisted for more than 30 minutes despite appropriate therapy. Grade A

- Once patients have stabilized, consider changing from Venturi Mask to nasal cannulae at 1-2 l/min. See recommendation 92

8.12.2 Exacerbation of Cystic Fibrosis.
Patients with breathlessness due to Cystic Fibrosis should be managed in a Cystic Fibrosis Centre unless this is not possible for geographic reasons. If not possible, all cases should be discussed with the Cystic Fibrosis Centre or managed according to a protocol that has been agreed with the regional centre. Patients with advanced Cystic Fibrosis may suffer from exacerbations which are similar to exacerbations of advanced COPD with associated hypoxaemia and hypercapnia. The principles of management are similar to those in acute exacerbations of COPD, including a need to maintain adequate oxygen saturation and avoiding excessive hypercapnia and acidosis. As in
COPD, non-invasive ventilation may be of value in severe cases (306). NIV in CF may also be helpful to reduce symptoms (e.g. work of breathing and dyspnoea) and assist in airway clearance.

It is recommended that patients with acute exacerbations of Cystic Fibrosis should be managed on similar lines to patients with acute exacerbations of COPD with a target oxygen saturation of 88-92% for most patients but recognition that individual patients may need to be managed differently on the basis of previous and current blood gas measurements. It is possible that patients with very high inspiratory flow rates might benefit from a Venturi flow rate set at 6 l/min to minimize the risk of the inspiratory flow rate exceeding the gas flow rate (see table 10.2). One study has shown that patients with a respiratory rate above 30 breaths per minute often have an inspiratory flow-rate above the minimum flow rate specified on the mask packaging. (117) However, there is no direct experimental evidence of the clinical effectiveness of increased flow rates from Venturi devices. Patients with Cystic Fibrosis who have had previous episodes of hypercapnic respiratory failure should be issued with an Oxygen Alert card with recommendations based on previous blood gas measurements. See recommendations 79-82

Recommendation (see Table 3 )

57 Initial therapy of Cystic Fibrosis exacerbations should be similar to the initial therapy of COPD exacerbations (see section 8.12.1). Grade D

8.12.3 Musculo-skeletal and neurological disorders
Hypoxaemia due to musculo-skeletal and neurological disorders is usually sub-acute or chronic but can be acute (e.g. Guillain- Barré Syndrome). For most such patients, non-invasive or invasive ventilatory support is more useful than supplementary oxygen and these patients are at risk of hypercapnic respiratory failure which may be aggravated by high doses of oxygen. For this reason, it is recommended that blood gases should be obtained as early as possible in all such cases. Pending the availability of blood gas results, a saturation target of 88-92% will avoid the risks of severe hypoxaemia or severe hypercapnia.

Recommendation (see Table 3 )

58 In the initial management of musculo-skeletal and neurological disorders with acute respiratory failure, aim at an oxygen saturation of 88-92%. Many such patients will be suitable for Non-invasive ventilation. Grade D

8.12.4 Obesity - hypoventilation syndrome
Patients with the obesity-hypoventilation syndrome often develop chronic hypercapnic respiratory failure and they may decompensate acutely to produce hypercapnic respiratory failure with acidosis. (317) For purposes of oxygen therapy, these patients should be treated in a similar manner to patients with hypercapnic respiratory failure due to an acute exacerbation of COPD (and they clearly do not require bronchodilator and steroid therapy). The initial target saturation will usually be 88 - 92% but, as with COPD, a lower target range may be appropriate for individual patients based on blood gas measurements during a previous exacerbation or due to acute acidosis. Assessment of patients with increasing shortness of breath or worsening oxygen saturation must include blood gases. As in COPD, patients with respiratory acidosis may benefit from non-invasive ventilation.
Recommendations (see Table 3)

59 In the initial management of obesity-hypoventilation syndrome with acute exacerbation, aim at an oxygen saturation of 88-92% Grade D

60 Non-invasive ventilation should be considered for these patients Grade C
# Table 3

## Patients requiring controlled or low-dose oxygen therapy

**Section 8.12**

The initial oxygen therapy is 24% or 28% Venturi mask.

The recommended initial oxygen saturation target range is 88-92% for most at-risk patients pending blood gas results.

Adjust target range to 94-98% (age below 70) or 92-98% (age 70 or above) if the PaCO2 is normal (unless there is a history of previous hypercapnic respiratory failure) and **recheck blood gases after one hour** (recommendations 53-54 and Chart 1).

Change to nasal cannulae when the patient is clinically stable—see recommendation 92.

<table>
<thead>
<tr>
<th>Initial oxygen therapy</th>
<th>Additional Comments</th>
<th>Recommendation number and grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>COPD</td>
<td>24% Venturi at 2 litres/minute or 28% Venturi mask at 4 litres/minute. May need lower range if acidicotic or if known to be very sensitive to oxygen therapy. Ideally use “Alert Cards” to guide therapy based on previous blood gas results. Increase flow by 50% if respiratory rate is above 30, see recommendation 93.</td>
<td>49-56 Grade C</td>
</tr>
<tr>
<td>Exacerbation of Cystic Fibrosis</td>
<td>24% Venturi at 2-4 litres/minute or 28% Venturi mask at 4 litres/minute. Admit to Regional CF centre if possible, if not discuss with regional centre or manage according to protocol agreed with regional CF centre. Ideally use “Alert Cards” to guide therapy. Increase flow by 50% if respiratory rate is above 30, see recommendation 93.</td>
<td>57 Grade D</td>
</tr>
<tr>
<td>Neuro-Muscular Disorders</td>
<td>Depends on severity of hypoxaemia. Usually 24% Venturi at 2 litres/minute or 28% Venturi mask at 4 litres/minute. May require ventilatory support. Risk of hypercapnic respiratory failure.</td>
<td>58 Grade D</td>
</tr>
<tr>
<td>Chest Wall disorders</td>
<td></td>
<td>58 Grade D</td>
</tr>
<tr>
<td>Morbid obesity</td>
<td></td>
<td>59-60 Grade D</td>
</tr>
</tbody>
</table>
8.13 Common medical emergencies for which oxygen therapy is indicated only if hypoxaemia is present (see also Table 4)
There are a number of conditions such as myocardial infarction, angina and stroke for which oxygen was traditionally given to all patients in an attempt to increase oxygen delivery to the heart or brain. However, the administration of supplemental oxygen to normoxaemic patients has very little effect on blood oxygen content but may reduce myocardial and cerebral blood flow. There is no evidence of benefit from the administration of supplemental oxygen to non-hypoxaemic patients with these conditions and there is some evidence of possible harm so it is recommended that oxygen should only be given to patients with these conditions if hypoxaemia is present, usually due to complications such as heart failure or pneumonia. There are no published trials supporting the use of oxygen to relieve breathlessness in non-hypoxaemic patients and there is evidence from randomised studies that oxygen does not relieve breathlessness compared with air in non-hypoxaemic COPD patients who are breathless following exertion. (374)

8.13.1 Acute Myocardial Infarction, suspected myocardial infarction and acute coronary syndromes
Some patients with acute myocardial infarction have heart failure and should be treated accordingly (see section 8.11.5). Most patients with suspected or confirmed myocardial infarction are not hypoxaemic and most are not breathless. In the case of non-hypoxaemic patients it is not known if supplementary oxygen may be beneficial by increasing the amount of oxygen delivered to the hypoxaemic area of myocardium or whether it may actually cause vasoconstriction with increased systemic vascular resistance and reduced myocardial oxygen supply with worsened systolic myocardial performance. (172, 173, 174, 213, 214, 215, 234, 312, 313). A recent study of patients having coronary arteriography found that breathing 100% oxygen reduced coronary blood flow velocity by 20% and increased coronary resistance by 23%. (378) There is also a theoretical possibility that high oxygen levels might exacerbate reperfusion injury to the heart. (312) Despite a multitude of large studies of intervention in myocardial infarction, there has been only one randomised study of oxygen therapy (in 1976) and this study did not identify any benefit from such therapy but found some evidence of potential harm. (69) This trial reported a significantly greater myocardial enzyme rise in the oxygen group, suggesting a greater infarct size. There was a three fold increase in mortality on oxygen therapy that did not reach statistical significance (3 deaths out of 77 patients treated with air versus 9 deaths amongst 80 patients given oxygen at 6 l/min via simple face mask for 24 hours). A systematic review and a historical review of oxygen therapy in acute myocardial ischaemia have both concluded that there was no evidence to support this practice in non-hypoxaemic patients and some evidence of possible harm. (3, 369) One study from 1969 showed that hypoxia did not affect the availability of oxygen for myocardial metabolism in normal subjects until the oxygen saturation fell to about 50% but evidence of myocardial ischaemia was seen at saturations of 70-85% in subjects with coronary artery disease. (370) In these circumstances, it is advised that patients with myocardial infarction or chest pain suspicious of myocardial infarction should be given supplementary oxygen if required to maintain a saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above.
The study of Lal and colleagues in the 1960s (42) has shown that hypoxaemia was present in a high proportion of patients diagnosed with myocardial infarction and could usually be reversed by medium dose oxygen but sometimes required treatment with a reservoir mask to achieve an oxygen tension above 60 mm Hg (8 kPa). The study of Wilson and Channer in 1997 (55) showed that desaturation below 90% was common in patients with myocardial infarction within the first 24 hours of admission to a coronary care unit but these authors may not have been aware that nocturnal desaturation to this level is very common in healthy individuals. (243) Wilson and Channer did not demonstrate any correlation between hypoxaemic events and adverse cardiac events. (55) They did, however, demonstrate that monitoring by oximetry was inadequate in UK coronary care units in the mid 1990s.

There are no UK guidelines for oxygen therapy in acute myocardial infarction. The 1998 European Society for Cardiology / European Resuscitation Council Task Force recommended the use of 3-5 litres per minute of oxygen via face mask to all patients with chest pain of presumed cardiac origin but no evidence was presented to support this advice (239). However, most of the papers that have raised concerns about the effects of oxygen on myocardial blood flow have been published since that date (see preceding paragraph). The European Society of Cardiology published subsequent guidance on the management of ST elevation myocardial infarction in 2003 (238). This revised guidance recommended the use of oxygen at 2-4 litres per minute by mask or nasal cannulae for patients with heart attacks associated with breathlessness or heart failure.

The European Resuscitation Council Guidelines for the management of acute coronary syndromes in 2005 recommended the use of supplementary oxygen at 4-8 litres per minute (device not specified) for patients with arterial oxygen saturation <90% and/or pulmonary congestion (237). The guideline acknowledged the lack of evidence of benefit for non-hypoxaemic patients but recommended supplementary oxygen in case of unrecognised hypoxaemia. This situation would apply in the pre-hospital setting but not in the hospital setting. Therefore, the limited available evidence supports the suggestion that clinicians should aim at a normal or near-normal oxygen saturation in patients with myocardial infarction, acute coronary syndrome and chest pain suspicious of coronary artery disease. A target saturation range of 92-98% will meet all of these goals and further research of this topic should be prioritized because this is such a common medical problem and there is so little existing evidence.

Recommendation (see Table 4)

61 In myocardial infarction and acute coronary syndromes, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above or 88-92% if the patient is at risk of hypercapnic respiratory failure. Grade D

8.13.2 Stroke
In the past, it was customary to give supplementary oxygen to all stroke patients to try to improve cerebral oxygenation. However, there has been only one randomised trial of oxygen therapy in stroke. (235) This trial found no difference in one year survival for the entire cohort of stroke patients and no difference in survival for patients with more severe strokes. However, for patients with minor or moderate strokes, one year mortality was 18% in the group given oxygen and 9% in the group given air (Odds Ration 0.45; 95% CI 0.23 to 0.90 p=0.023). Based largely on the results of this trial, the Royal College of Physicians Stroke Guideline recommends that oxygen saturation
should be maintained in the normal range in stroke patients (314). It is recommended that stroke patients should receive supplementary oxygen only if this treatment is required to achieve an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above (88-92% for patients with co-existing risk COPD or other risk of respiratory acidosis).

There has also been some discussion concerning the optimal body position for the management of patients with stroke and potential hypoxaemia. A systematic review concluded that there was limited evidence that sitting in a chair had a beneficial effect and lying positions had a deleterious effect on oxygen saturation in acute stroke patients with respiratory co-morbidities but acute stroke patients without respiratory co-morbidities can adopt any body position. (53) The authors of this review recommended that people with acute stroke and respiratory co-morbidities should be positioned as upright as possible.

**Recommendation** (see Table 4)

62 In stroke, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above or 88-92% if the patient is at risk of hypercapnic respiratory failure. Grade B

8.13.3 Obstetric emergencies and labour

The use of oxygen has been recommended during many obstetric emergencies and in particular, for collapse related to haemorrhage, pulmonary embolism, eclampsia or amniotic fluid embolism. Severe pre-eclampsia and eclampsia may occasionally present with pulmonary oedema and this can occur in the antenatal or postnatal periods. Medical problems such as pneumonia or acute exacerbations of asthma are not uncommon during pregnancy. Peripartum cardiomyopathy is rare but may present with heart failure in the postnatal period. Major trauma is increasingly common, particularly related to road traffic accidents.

The use of oxygen during pregnancy should follow the same general principles as the use of oxygen for other patients. Pregnant women suffering major trauma or severe hypoxaemia should be started on high dose oxygen via a non-rebreathing reservoir mask and those with milder hypoxaemia can use nasal cannulae or a simple face mask or Venturi mask to achieve an oxygen saturation of 94-98% in most cases. If an undelivered woman is hypoxaemic, she should be managed with left lateral tilt applied. This will improve cardiac output (365) and may also facilitate breathing for mechanical reasons.

Oxygen is commonly given as part of the treatment for many obstetric emergencies. However, it is recommended that when oxygen is administered during pregnancy or labour, clinicians should aim to achieve normoxaemia (saturation 94-98%). There is no randomised trial evidence to suggest that maternal ‘hyperoxaemia’ is beneficial to mother or fetus.

Oxygen is often given when acute fetal compromise is suspected in labour, in the hope of increasing oxygen delivery to the fetus. A Cochrane review found no trials addressing the use of oxygen for fetal compromise. However, two
trials of prophylactic oxygen in labour found a significant increase in the incidence of cord blood acidosis (pH below 7.20) in the oxygenation group [RR 3.5 (95% CI 1.34 to 9.19)]. (316)

In summary, it is recommended that pregnant women with evidence of hypoxaemia should have their blood oxygen saturation maintained in the normal range (94-98%) using supplemental oxygen as necessary to achieve this effect. This applies both before or during labour as well as in the postnatal period. The causes of maternal hypoxaemia may include trauma, pre-existing or de novo medical conditions as well as pregnancy-specific complications. In all of these situations, the aim should be normoxaemia (saturation 94-98%).

**Recommendations**

63 Women who suffer from major trauma, sepsis or acute illness during pregnancy should receive the same oxygen therapy as any other seriously ill patients, with a target oxygen saturation of 94-98%. The same target range should be applied to women with hypoxaemia due to acute complications of pregnancy (e.g. collapse related to amniotic fluid embolus, eclampsia or ante partum or postpartum haemorrhage) Grade D

64 Women with underlying hypoxaemic conditions (e.g. heart failure) should be given supplemental oxygen during labour to achieve an oxygen saturation of 94-98% Grade D

65 All women with evidence of hypoxaemia in late pregnancy should be managed with left lateral tilt to improve cardiac output. Grade B

66 The use of oxygen during labour is widespread, but with evidence this may be harmful to the fetus this is not currently recommended in situations where the mother is not hypoxaemic (except as part of a controlled trial). Grade A

**8.13.4 Anxiety and hyperventilation or dysfunctional breathing.**

Many patients who present to hospital with breathlessness are found to have no cardio-pulmonary problems and many such patients have a specific diagnosis of hyperventilation, dysfunctional breathing, upper airway dysfunction or panic attacks, sometimes in addition to asthma or some other underlying respiratory disorder. (319) Many such patients will have an abnormally high oxygen saturation of 99 or 100% and clearly do not require supplemental oxygen therapy. Many other non-hypoxaemic patients will present to hospital with acute breathlessness of unknown cause and the majority of patients with an elevated respiratory rate are likely to have an organic illness. In some cases, simple investigations will reveal a specific diagnosis such as pneumothorax or pneumonia or pulmonary embolism but many cases remain undiagnosed. A policy of giving supplementary oxygen if the saturation falls below 94% if aged below 70 and below 92% if aged 70 and above will avoid exposing patients with undiagnosed medical illnesses to the risk of hypoxaemia whilst avoiding the un-necessary use of oxygen in patients with behavioural or dysfunctional breathlessness.

Studies in normal volunteers have demonstrated that compensatory desaturation may occur shortly after voluntary hyperventilation. (137) The mean PaO2 of 10 male volunteers increased from 13.7 kPa to 18.6 kPa (103 to 140 mm Hg) during hyperventilation but fell to a nadir of 7.8 kPa (58 mm Hg) about 7 minutes after cessation of hyperventilation and did not normalise until after a total of 17 minutes of observation. It is not known whether or not
this occurs after pathological hyperventilation but this phenomenon could cause considerable confusion if it should occur in an Emergency Department.

A traditional treatment for hyperventilation was to ask the subject to re-breathe from a paper bag to allow the carbon dioxide level in the blood to normalise. However, it has been shown that this practice can cause hypoxaemia with potentially fatal consequences. (95) The average fall in oxygen tension during re-breathing was 26 mm Hg (3.5 kPa) and a maximum fall was 42 mm (5.6 kPa). This guideline does not recommend re-breathing from a paper bag in cases of hyperventilation unless the patient has been shown to have hyperoxia and a low carbon dioxide level and any such treatment should be monitored with continuous oximetry and discontinued if the patient should desaturate.

Recommendations

67 Organic illness must be excluded before making a diagnosis of hyperventilation. Grade C

68 Patients with a definite diagnosis of hyperventilation should have their oxygen saturation monitored. Those with normal or high SpO2 do not require oxygen therapy. Grade B

69 Re-breathing from a paper bag can be dangerous and is NOT recommended as a treatment for hyperventilation. Grade C

8.13.5 Poisonings with substances other than carbon monoxide
Many poisons and drugs can cause respiratory or cardiac depression or direct toxic effects on the lungs. The treatment of individual toxic agents is beyond the scope of this guideline. Specific antidotes such as naloxone should be given if available and oxygen saturation should be monitored closely. Supplementary oxygen should be given to achieve a target saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above pending the results of blood gas analysis (88-92% if at risk of hypercapnic respiratory failure).

Three specific types of lung injury deserve special mention. Oxygen is known to be hazardous to patients with paraquat poisoning (273, 274) and oxygen potentiates bleomycin lung injury and may potentiate lung injury from aspiration of acids. (96, 275, 276) Because of these risks, oxygen should be given to patients with these conditions, only if the oxygen saturation falls below 90%. Some authors have suggested the use of hypoxic ventilation with 14% oxygen as a specific treatment for paraquat poisoning. (277)

Bleomycin lung injury can be potentiated by high-dose oxygen therapy, even if given several years after the initial lung injury. Therefore, it is recommended that high doses of oxygen should be avoided in patients with possible bleomycin-induced lung injury and a lower oxygen saturation target range should be accepted (e.g. 88-92%).

Recommendations (see Table 4)

70 In most poisonings, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above. Grade D

71 In poisoning by paraquat, bleomycin and acid inhalation, aim at a saturation of 88-92% Grade D

8.13.6 Metabolic, endocrine and renal disorders
Many metabolic and renal disorders can cause metabolic acidosis which increases respiratory drive as the body tries to correct the acidosis by increased excretion of carbon dioxide via the lungs. Although these patients have
tachypnoea, they do not usually complain of breathlessness and most have a high oxygen saturation (unless there is a co-existing pulmonary or cardiac problem). Supplementary oxygen is not required for such patients unless the oxygen saturation is reduced, in such cases, oxygen should be given to maintain a saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above.

**Recommendation**  (see Table 4 )

72 In most metabolic and renal disorders, aim at an oxygen saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above. Grade D
Table 4

Conditions for which oxygen therapy is not required unless the patient is hypoxaemic  

Section 8.13

If hypoxaemic, the initial oxygen therapy is nasal cannulae at 2-6 l/min or simple face mask at 5-10 l/min unless stated otherwise (see comments section)

Recommended initial oxygen saturation target range, unless stated otherwise, is 94-98% if aged <70 and 92-98% saturation if aged ≥70

If patients have COPD or other risk factors for hypercapnic respiratory failure, aim at a saturation of 88-92% pending blood gas results but adjust to 94-98% (age below 70) or 92-98% (age 70 or above) if the PaCO2 is normal (unless there is a history of previous hypercapnic respiratory failure) and re-check blood gases after one hour.

<table>
<thead>
<tr>
<th>Additional Comments</th>
<th>Recommendation number and grade</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Myocardial infarction and Acute Coronary Syndromes</strong></td>
<td></td>
</tr>
<tr>
<td>Most patients with acute coronary artery syndromes are not hypoxaemic and the benefits/harms of oxygen therapy are unknown in such cases.</td>
<td>61</td>
</tr>
<tr>
<td>Grade D</td>
<td></td>
</tr>
<tr>
<td><strong>Stroke</strong></td>
<td></td>
</tr>
<tr>
<td>Most stroke patients are not hypoxaemic. Oxygen therapy may be harmful for non-hypoxaemic patients with mild-moderate strokes.</td>
<td>62</td>
</tr>
<tr>
<td>Grade B</td>
<td></td>
</tr>
<tr>
<td><strong>Obstetric Emergencies</strong></td>
<td></td>
</tr>
<tr>
<td>Oxygen therapy may be harmful to the fetus if the mother is not hypoxaemic.</td>
<td>63-66</td>
</tr>
<tr>
<td>Grades A-D</td>
<td></td>
</tr>
<tr>
<td><strong>Hyperventilation or dysfunctional breathing</strong></td>
<td></td>
</tr>
<tr>
<td>Unlikely to require oxygen therapy. Exclude organic illness</td>
<td>67-69</td>
</tr>
<tr>
<td>Grade C</td>
<td></td>
</tr>
<tr>
<td><strong>Most poisonings and drug overdoses</strong></td>
<td></td>
</tr>
<tr>
<td>(See Table 1 for Carbon Monoxide poisoning)</td>
<td></td>
</tr>
<tr>
<td>Hypoxia is more likely with respiratory depressant drugs, give antidote if available.</td>
<td>70</td>
</tr>
<tr>
<td><em>e.g. Naloxone for opiate poisoning</em></td>
<td>Grade D</td>
</tr>
<tr>
<td>Check blood gases to exclude hypercapnia if a respiratory depressant drug has been taken.</td>
<td></td>
</tr>
<tr>
<td><strong>Poisoning with Paraquat Bleomycin or Acid Inhalation</strong></td>
<td></td>
</tr>
<tr>
<td>Patients with Paraquat poisoning, Bleomycin lung injury or acid inhalation may be harmed by supplemental oxygen. Avoid oxygen unless the patient is hypoxaemic. <strong>Target saturation is 88-92%</strong></td>
<td>71</td>
</tr>
<tr>
<td>Grade C</td>
<td></td>
</tr>
<tr>
<td><strong>Metabolic &amp; Renal disorders</strong></td>
<td></td>
</tr>
<tr>
<td>Most do not need oxygen</td>
<td>72</td>
</tr>
<tr>
<td>Grade D</td>
<td></td>
</tr>
</tbody>
</table>
Section 9

Emergency use of oxygen in ambulances, community and pre-hospital settings

- This section applies to a range of clinical settings to include emergency oxygen use in patient’s homes, GP practices or health centres and during emergency ambulance journeys to hospital. Management in some pre-hospital settings such as a Primary Care Centre or in a well equipped paramedic ambulance may be almost identical to hospital management. Readers are referred to section 10 for advice concerning choice of oxygen delivery devices and systems.

- Readers are referred to tables 1 to 4 and charts 1 and 2 for a summary of the key elements of oxygen therapy in common medical emergencies. A brief summary of this section can be downloaded from www.brit-thoracic.org.uk/guidelines.html

9.1 Pulse oximetry and availability of oxygen

It is essential to provide optimal oxygen therapy at the earliest possible opportunity while the acutely breathless patient is being assessed and treated in the community and during transfer to hospital. For most such patients, the main concern is to give sufficient oxygen to support their needs. Hypoxia can lead to cardiac arrhythmias, renal damage and, ultimately, cerebral damage. However, excessive oxygen therapy can also be dangerous for some patients, especially those with advanced COPD. Target saturation should be used, pulse oximetry is necessary to achieve this. Section 10.4.2 provided advice concerning the choice of oxygen cylinders in primary care practices. Emergency Ambulances and Emergency / Fast Response type vehicles and Ambulance Service motorbikes and cycles should be equipped with oxygen and oximeters germane to the mode of transport. Thus Fast Response Cars / Motorbikes and Cycles will require handheld finger oximeter type devices and staff initiating oxygen in the home will need a portable or finger oximeter.

Recommendations

73 Pulse oximetry must be available in all locations where emergency oxygen is being used. Grade D

- (see also the limitations of using pulse oximetry section 7.1.2)

74 Emergency oxygen should be available in primary care medical centres; preferably using oxygen cylinders with integral high-flow regulators. Alternatively, oxygen cylinders fitted with high-flow regulators (delivering over 6 L/min) must be used. Grade D

75 All documents which record oximetry measurements should state whether the patient is breathing air or a specified dose of supplemental oxygen. Grade C

9.2 Clinical assessment by initial response team (GP, nurse or ambulance team).
It is suggested that the first health care professional(s) to encounter an acutely breathless patient should perform an initial “ABC” assessment (Airway, Breathing, Circulation), followed by obtaining a quick history from the patient and/or family or friends. Immediate assessment should include a recording of pulse rate and respiratory rate and pulse oximetry should be recorded.

- **Clinical assessment of a breathless patient starts with ABC (Airway, Breathing, Circulation, See recommendation 10)**

- **A brief history should be taken from the patient or other informant.**

- **Initial assessment should include pulse and respiratory rate in all cases. See recommendation 10**

- **Pulse oximetry should always be measured in patients with breathlessness or suspected hypoxia. See recommendation 8**

- **Disease-specific measurements should also be recorded (e.g. Peak Expiratory Flow in asthma, Blood Pressure in cardiac disease).**

9.3 Immediate management of hypoxaemic patients.

Having ascertained that the airway is clear, the emergency responders should commence oxygen treatment if the oxygen saturation is below the target. The initial oxygen therapy should follow the general principles given in tables 1 to 4 and Charts 1 and 2.

**Recommendations**

- The initial oxygen therapy to be used in the various clinical situations is given in tables 1-4.

- If there is a clear history of asthma or heart failure, or other treatable illness, then appropriate treatment should be instituted in accordance with guidelines or standard management plans for each disease. Grade D

76 The oxygen saturation should be monitored continuously until the patient is stable or arrives at hospital for a full assessment. The oxygen flow should be adjusted upwards or downwards to maintain the target saturation range. Grade D

77 In most emergency situations, oxygen is given to patients immediately without a formal prescription. However, a subsequent written record must be made of what oxygen therapy has been given to every patient (in a similar manner to the recording of all other emergency treatment). Grade D

9.4 Patients with known COPD.

A proportion of breathless patients will have Chronic Obstructive Pulmonary Disease (COPD) (chronic bronchitis and emphysema). Unfortunately, a recent Cochrane review of oxygen therapy for COPD in the pre-hospital setting found no relevant studies. (225)

Audit of emergency admissions to UK hospitals shows that about 25% of breathless medical patients who require hospital admission in the UK have COPD as a main diagnosis. Many of these patients will require carefully controlled oxygen therapy because they are at risk of CO2 retention or respiratory acidosis. In a large UK study (48), 47% of COPD patients had PaCO2 above 6.0 kPa (45 mm Hg) and 20% had respiratory acidosis (pH <7.35) and 4.6% had severe acidosis (pH <7.25). Acidosis was commoner if the blood oxygen was above 10kPa (75 mm Hg) .
The authors recommend that patients with acute COPD should be maintained at 7.3-10kPa (55-75 mm Hg or SaO2 85-92%) to avoid the dangers of hypoxaemia and acidosis.

**Recommendation**

78 Patients with COPD should initially be given oxygen via a Venturi 24% mask at a flow rate of 2 l/min or 28% Venturi mask at a flow rate of 4 l/min and oxygen saturation should be 88-92% in most cases or else an individualised saturation range based on the patient's blood gas measurements during previous exacerbations. Grade C

9.5 Patients who should be assumed to have COPD.

One of the challenges faced by the initial clinical response team is that the diagnosis may be unclear and the patient’s medical records or detailed history may not be available. It has been shown that ambulance teams may be aware of a diagnosis of COPD in only 58% of cases. (252)

The Guidelines group consider that an initial diagnosis of COPD should be assumed if there is no clear history of asthma and the patient is over 50 years of age and a long-term smoker or ex-smoker with a history of longstanding breathlessness on exertion. The diagnosis should be reassessed on arrival at hospital where more information will probably become available.

- Patients over 50 years of age who are long-term smokers with a history of exertional breathlessness and no other known cause of breathlessness should be treated as if having COPD for the purposes of this guideline. See Recommendation 49

9.6 Other patients at risk of hypercapnic respiratory failure.

- Any patient with severe kyphoscoliosis or severe ankylosing spondylitis.
- Severe lung scarring from old TB (especially with thoracoplasty)
- Morbid obesity (Body Mass Index > 40)
- Patients with neuromuscular disorders (especially if muscle weakness has led to wheelchair use)
- Any patient on home mechanical ventilation
- Use of home mechanical ventilation.
- Overdose of opiates, benzodiazepines or other respiratory depressant drugs.

9.7 Oxygen alert cards and 24% or 28% Venturi masks in COPD patients (and others at risk) who have had an episode of hypercapnic respiratory failure.

The administration of high oxygen concentrations in acute COPD and other conditions (see section 8.12) leads to worsening of hypercapnic respiratory failure and respiratory acidosis (48). Patients with COPD and a blood oxygen >10 kPa (75 mm Hg) and a PCO2 > 6.0 kPa (45 mm Hg) may be assumed to have had an episode of oxygen toxicity. This avoidable problem has occurred historically during the transfer to hospital, prior to measurement of arterial blood gases or before a definitive diagnosis is known. Furthermore, ambulance teams are often not informed...
at present of a diagnosis of COPD (252) and may not be aware of the presence of other high-risk conditions such as kyphoscoliosis or respiratory failure due to neuro-muscular conditions. These patients can be issued with an oxygen alert card and a 24% or 28% Venturi mask based on previous blood gas results. The recommended oxygen saturation will be based on the clinical scenario for each individual patient but will usually be 88-92%, occasional 85-88% or 85-90% based on previous blood gas results. Patients should be instructed to show this card to the ambulance crew and Emergency Department staff in order to avoid the use of high oxygen concentrations. This scheme can be successful (224). Ambulance control can also be informed about which patients are issued with oxygen warning cards. (326). The current JRCALC (Joint Royal Colleges Ambulance Liaison Committee) guideline for the use of oxygen in COPD will need to be revised to accommodate these suggestions. (229) An example of an oxygen alert card is shown below (Figure 9.1).

**Recommendations**

79 COPD patients and other patients who have had an episode of hypercapnic respiratory failure should be issued with an oxygen warning card and with a 24% or 28% Venturi mask. They should be instructed to show the card to the ambulance crew and Emergency Department staff in the event of an exacerbation. Grade C

80 The content of the Alert Card should be specified by the physician in charge of the patient’s care, based on previous blood gas results. Grade D

81 The primary care team and ambulance control should also be informed by a responsible clinician that the patient has had an episode of hypercapnic respiratory failure and carries an Oxygen Alert Card. These patients home addresses and ideal oxygen dose or target saturation ranges can be flagged in the ambulance control systems and disseminated to ambulance crews when required. Grade D

82 Out of hours services providing emergency Primary Care services should be informed by a responsible clinician that the patient has had an episode of hypercapnic respiratory failure and carries an Oxygen Alert card. Use of oxygen in these patients will be guided by the instructions on the Alert Card. Grade D

83 During ambulance journeys, oxygen driven nebulisers should be used for patients with asthma and may be used for COPD patients in the absence of an air-driven compressor system. If oxygen is used for patients with known COPD, it should be limited to 6 minutes. This will deliver most of the nebulised drug dose but limit the risk of hypercapnic respiratory failure. (section 10.8.2) Grade D.

**Figure 9.1 Example of oxygen alert card.**

---

**OXYGEN ALERT CARD**

Name: ____________________________

I am at risk of type II respiratory failure with a raised CO₂ level.

Please use my % Venturi mask to achieve an oxygen saturation of ___ % ___ % during exacerbations

Use compressed air to drive nebulisers (with nasal oxygen at 2 l/min).
9.8 Choice of devices in pre-hospital care

The range of oxygen delivery devices is very wide as discussed in section 10. However, most patients can be managed with just 4 types of oxygen delivery device.

**Recommendation**

84 It is recommended that the following delivery devices should be available in pre-hospital settings where oxygen is administered. Grade D See Recommendations 86-93 in section 10.5.4

1. High concentration reservoir mask (non-rebreath mask) for high-dose oxygen therapy.
2. Nasal cannulae (preferably) or simple face mask for medium dose oxygen therapy.
3. 28% Venturi mask for patients with known previous hypercapnic respiratory failure with inappropriately high arterial blood oxygen values (patients who have an oxygen alert card may have their own 24% or 28% Venturi mask)
4. Tracheostomy masks for patients with tracheostomy or previous laryngectomy.
Section 10

Practical aspects of oxygen therapy

Oxygen delivery systems

Oxygen delivery systems can be considered as two components, namely
a) The method of storage and provision of oxygen, *e.g.* cylinders, and
b) The method of delivery to the patient, *e.g.* Venturi mask.

The options available for both will depend on the environment in which it is being used and patient needs.

10.1 Oxygen storage and provision

10.1.1 Cylinders (compressed gas)

Cylinders contain compressed gas held under a very high pressure. They come in an array of sizes and hence capacity, ranging from small portable cylinders for individual patient use to large cylinders suitable for hospital use. These can be used for bedside administration where piped oxygen is not available or can be the supply for a piped system.

Table 10.1 Examples of oxygen cylinder sizes and capacities

<table>
<thead>
<tr>
<th>Oxygen cylinder sizes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Size</td>
</tr>
<tr>
<td>Height</td>
</tr>
<tr>
<td>Capacity (litres)</td>
</tr>
</tbody>
</table>

With recent changes in technology high pressure cylinders are now available i.e. filled to 200 bar rather than 137 bar which can contain 54% more gas for the same size cylinder. It is important for all users of oxygen to be aware that most oxygen cylinders are colour coded (black cylinder with white shoulder). Small light weight cylinders are also available for ambulatory use (these weigh 3.2 Kg when full). All systems containing compressed gases in the UK are subject to the Pressure Systems Safety Regulations 2000 (SI 2000 No 128). These regulations are intended to prevent the risk of injury from pressurised systems.

- Trusts must ensure that they have a policy in place which ensures the safety of patients, staff and contractors in the provision, storage, use and maintenance of compressed gas systems as required by the Health and Safety at Work etc Act 1974

10.1.2 Liquid oxygen
Liquid oxygen is contained in pressure tanks and is obtained from atmospheric oxygen by fractional distillation. It has to be evaporated into a gas before use. Large tanks are often used by hospitals and small tanks can be used domestically. Portable liquid oxygen is also available in small portable containers which can be filled from the larger tanks.

10.1.3 Oxygen Concentrators

Oxygen concentrators are largely used in the domiciliary setting for the provision of long term oxygen therapy and are therefore not used in the acute setting so will not be covered further.

10.2 Patient delivery methods / interfaces

10.2.1 High concentration reservoir mask (non-rebreathing mask)

This type of mask delivers oxygen at concentrations between 60% and 90% when used at a flow rate of 10-15 litres per minute. (151) The concentration is not accurate and will depend on the flow of oxygen and the patients breathing pattern. These masks are most suitable for trauma and emergency use where CO2 retention is unlikely. (Table1)

10.2.2 Simple face mask

This type of mask delivers oxygen concentrations between 40% and 60%. It is sometimes referred to as a MC Mask, Medium Concentration Mask, Mary Catterall Mask or as a "Hudson Mask" but the latter description is discouraged because the Hudson company make many types of mask (including high concentration Reservoir masks). The Guideline Group favours the term "simple face mask". The oxygen supplied to the patient will be of variable concentration depending on the flow of oxygen and the patients breathing pattern. The concentration can be changed by increasing or decreasing the oxygen flows between 5 and 10 litres per minute. However, different brands of simple face mask can deliver a different oxygen concentration at a given flow-rate. Flows of less than 5 litres can
cause increased resistance to breathing and there is a possibility of a build up of CO2 within the mask and rebreathing may occur. (168)

This mask is suitable for patients with hypoxaemic respiratory failure (type 1), but not suitable for patients with hypercapnic (type 2) respiratory failure. This mask may deliver a high concentration of oxygen (above 50%) and is therefore not recommended for patients who require low-dose oxygen therapy because of the risk of CO2 retention. Patients using a simple face mask may have an inspiratory flow rate greater than the gas flow rate from the mask so the simple face mask should not be used at flow rates below 5 l/min. (168) Several publications have shown that patients who require medium dose oxygen therapy tend to prefer nasal cannulae to simple face masks and the cannulae are more likely to be left in position by the patient and less likely to fall off. (175, 253, 254, 255)

10.2.3 Venturi mask

A Venturi mask will give an accurate concentration of oxygen to the patient regardless of oxygen flow rate (the minimum suggested flow rate is written on each Venturi device and the available options are shown in table 10.2). The oxygen concentration remains constant because of the Venturi principle. The gas flow into the mask is diluted with air which is entrained via the cage on the Venturi adaptor. The amount of air sucked into the cage is related to the flow of oxygen into the Venturi system. The higher the flow the more air is sucked in. The proportions remain the same and therefore the Venturi mask delivers the same concentration of oxygen as the flow rate is increased. Venturi masks are available in the following concentrations, 24%, 28%, 35%, 40%, and 60%. They are suitable for all patients needing a known concentration of oxygen but 24% and 28% Venturi masks are particularly suited to those at risk of CO2 retention (e.g. COPD). A further benefit of the Venturi masks is that the flow rate of gas from the mask will usually exceed the inspiratory flow rate of the patient. One study has shown that patients with an respiratory rate above 30 breaths per minute often have an inspiratory flow-rate above the minimum flow rate specified on the mask packaging. (117) Therefore, for patients with a high respiratory rate, it is suggested that the flow-rate for Venturi masks should be set above the MINIMUM flow rate listed on the packaging. The accuracy of oxygen delivery from a Venturi mask is greatly reduced if the mask is not accurately placed on a patient's face. (165)
• Patients with a respiratory rate above 30 breaths per minute often have a flow rate which is above the minimum delivered by the Venturi system as specified by the flow rate recommended for the mask.

Evidence III
Table 10.2  Total gas flow rate from Venturi masks at different oxygen flow-rates.

<table>
<thead>
<tr>
<th>Venturi Values</th>
<th>24% Oxygen</th>
<th>28% Oxygen</th>
<th>35% Oxygen</th>
<th>40% Oxygen</th>
<th>60% Oxygen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen Flow l/Min</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>84</td>
<td>82</td>
<td>30</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>67</td>
<td>50</td>
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<td>10</td>
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<td>8</td>
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<td>4</td>
<td>102</td>
<td>44</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>51</td>
<td></td>
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</tr>
</tbody>
</table>

Venturi masks deliver a constant dose of oxygen but the effect on the patient will depend on the condition being treated and on the breathing pattern and baseline oxygen saturation of the patient. As might be expected from the oxygen dissociation curve, patients with an oxygen saturation that is already in the normal range will have a very small rise in oxygen saturation (although the arterial oxygen tension is likely to rise substantially). However, patients with a very low oxygen saturation will have a marked rise if given even a small dose of oxygen. This is because the oxygen dissociation curve is actually a "rapid escalator" rather than a "slippery slope". (228) This is illustrated in the following figure which uses actual oxygen saturations from references 191 and 192 together with calculated saturations from references 14 (2 different groups) and references 23 and 190.

Figure 10.1  Oxygen saturation response to therapy with 24%, 28% and 35% oxygen
10.2.4 Nasal cannulae

Nasal cannulae can be used to deliver low and medium-dose oxygen concentrations. However, there is wide variation in patients' breathing patterns so the same flow rate of nasal oxygen may have widely different effects on the blood oxygen and carbon dioxide levels of different patients. Nasal cannulae at 1-4 litres per minute can have effects on oxygen saturation approximately equivalent to those seen with 24-40% oxygen from Venturi masks. (90, 182) The oxygen dose continues to rise up to flows of 6L/min (182) but some patients may experience discomfort and nasal dryness at flows above 4 l/min, especially if maintained for several hours. However there is marked individual variation in breathing pattern so the flow rate must be adjusted based on oximetry measurements and, where necessary, blood gas measurements. A crossover comparison of nasal cannulae versus a Venturi mask (both adjusted to give satisfactory initial oxygen saturation) showed that the oxygen saturation of patients with exacerbated COPD fell below 90% for 5.4 hours per day during treatment with a Venturi mask compared with only 3.7 hours per day during treatment with nasal cannulae. (5)
The upper range of oxygen delivery from nasal cannulae is a little lower than the output of a simple face mask but the lower range goes a lot lower than a simple face mask which should not be used below a flow rate of 5 litres per minute (about 40% oxygen). (168) The performance and variation of nasal cannulae for medium concentration oxygen therapy is broadly similar to that of the simple face mask both in laboratory experiments (182) and in clinical practice. (253, 254-257) One study (257) suggested that the saturation was lower with nasal cannulae compared with simple face masks in a sub-group of male patients following abdominal surgery. Further studies are required to see if this was a chance finding or a genuine clinical difference between the devices. Three patient preference studies comparing nasal cannulae with simple face masks in post-operative care found that patient preference was strongly in favour of nasal cannulae with up to 88% of patients preferring cannulae to masks. (253, 254, 255) Another advantage of cannulae over simple face masks is that they are less likely to be removed either accidentally to allow the patient to speak or eat. (176, 254) There are no comparisons of these devices in acute care but there is no reason to believe that the results would be any different for patients requiring medium-dose oxygen therapy.

Advantages of nasal cannulae compared with simple face masks for medium dose oxygen therapy. (Evidence Level III)

1. Comfort (but a minority of patients dislike the flow of oxygen into the nose, especially above 4 l/min)
2. Adjustable flow gives wide oxygen dose range (flow rate of 1-6 L/min gives FIO2 from approximately 24% to approximately 50%)
3. Patient preference
4. No claustrophobic sensation
5. Not taken off to eat or speak and less likely to fall off
6. Less affected by movement of face
7. Less inspiratory resistance than simple face masks
8. No risk of re-breathing of CO2
9. Cheaper

Disadvantages of nasal cannulae:

1. May cause nasal irritation or soreness
2. Will not work if nose is severely congested or blocked

10.2.5 Tracheostomy mask

These devices are designed to allow oxygen to be given via a tracheostomy tube or to patients with previous laryngectomy; i.e. “Neck Breathing Patients”. Adjust the oxygen flow rate to achieve saturation in accordance with
table 1. Oxygen given in this way for prolonged periods needs constant humidification and patients may need suction to remove mucus from the airway.

10.2.6 CPAP (Continuous Positive Airways Pressure) and NIV (Non Invasive Ventilation)
These treatment options are beyond the scope of the present guideline. Readers are referred to the BTS Guideline concerning the use of NIV in patients with exacerbations of COPD. (329)

10.2.7 Flow meters
All oxygen delivery systems must have a method of taking the high pressure/flow of gas and reducing it so it can be administered to the patient at a specific flow depending on the individual’s needs and mask etc being used.
Piped oxygen points have Schrader flow meters and cylinders have pressure and flow regulators. Most oxygen flow meters use a floating ball to indicate the flow rate. The centre of the ball should be aligned with the appropriate flow-rate marking. The example shown below indicates the correct setting to deliver 2 litres per minute.

10.2.8 Oxygen tubing and oxygen wall outlets
Oxygen tubing is needed to connect flow meters and regulators to the patient delivery device. It is important to ensure that all tubing is connected correctly at both ends. The National Patient Safety Agency has reported frequent adverse events related to oxygen use, including four reports of instances where an oxygen mask was connected in error to a compressed air outlet instead of an oxygen outlet. Compressed air outlets are often used to drive nebulisers on in hospitals because they are quieter than electrical compressors. However, the flow meter looks very similar to an oxygen flow meter and is often mounted beside an oxygen flow meter so it is very important to ensure that air flow meters are clearly labelled. Air flow meters are never required in an emergency and should be removed from wall sockets when not in use or else covered by a designated “hood” whilst not in use. The Guideline authors are also aware of some cases where twin oxygen outlets were in use and the wrong one had been turned on or off. For example, one patient tried to turn off the oxygen flow after finishing a nebulised treatment but accidentally turned off the oxygen flow to a neighbouring patient with serious consequences. It is recommended that patients should not be allowed to adjust oxygen flow, especially if there are dual outlets.

Recommendation
85 Trusts should take measures to minimise the risk of oxygen tubing being connected to the incorrect wall oxygen outlet or to outlets that deliver compressed air instead of oxygen. Air flow-meters should be removed
from the wall sockets or covered with a designated air outlet cover when not in use. Special care should be taken if twin oxygen outlets are in use. Grade D

10.3 Oxygen carriage and delivery during patient transport in ambulances etc

Transport of oxygen cylinders in vehicles comes under the transport of dangerous substances act, or the carriage regulations only if 1,000 litres or more (measured by the water capacity of the cylinder) is carried at any one time. Therefore, ambulances are exempt from this. Normal Health and Safety requirements will still apply. (302, 303)

10.3.1 Health and Safety Executive Guidance for safe use of oxygen cylinders: (Reference 302, 303)

- All cylinders must be secured appropriately so they cannot move in transit (includes portable cylinders)
- No smoking in the vicinity of cylinders
- Cylinders must be checked regularly for obvious signs of leakage
- Cylinders must be kept out of direct sunlight
- Green warning triangle ‘compressed gas’ should be displayed on the vehicle
- Cylinders should never be lifted by the neck
- They should only be changed by suitably trained personnel
- Apart from portable cylinders all cylinders should be moved using a cylinder trolley

10.3.2 Oxygen use by UK ambulance services.

Currently within the UK, the Ambulance Service, whether NHS or Private, have a range of vehicles and oxygen delivery systems at their disposal. There is an increasing use of Cycle Response Units, which tend to use the lightweight AZ or C sized cylinder with a capacity of 170 litres. Motorcycle Response Units are generally equipped with the same AZ or C sized cylinders. Fast Response Units based on cars tend to be equipped with at least two of the lightweight CD sized cylinder which holds 460 litres. (The CD cylinder is also the size favoured by Mountain, Cave and Mines Rescue Teams).

Front Line A& E Ambulances are usually equipped with piped oxygen fittings (Schraeder type) and supplied from two HX sized (2,300 litres) cylinders, as well as carrying at least two CD sized cylinders to power a portable oxygen powered resuscitator. The piped supply has several outlet points placed in strategic positions to which are attached standard Schrader flow meters (0 - 15 l/min). This enables oxygen to be given throughout the patient’s journey. The ambulance is also equipped with a portable supply which can be used at the site of an accident, taken into a patient’s
home or can be used when transferring a patient. They carry a range of patient interfaces for delivering the oxygen under the different circumstances encountered.

The portable resuscitators are always capable of supplying free-flow oxygen therapy as well as their resuscitator facility. Again, there are a variety of portable oxygen powered resuscitators and it is beyond the scope of these guidelines to describe each and every one available for pre-hospital care use. It is strongly suggested that those practitioners who need to work closely with the Ambulance Service should become familiar with the equipment used by their local Ambulance Service provider. With the possible exception of the Cycle Response Units all types of Ambulance Service response will have portable resuscitators, bag-valve-mask devices & portable suction as a minimum. Front Line Emergency Department Ambulances will also have vehicle powered suction available.

It is also very common now for Patient Transport Service ambulances to be equipped with an oxygen supply, normally an HX (2,300 litre) cylinder delivering the oxygen via a flow meter attached directly to the cylinder. Such vehicles also tend to carry basic hand held suction devices. The masks available are generally high concentration reservoir masks and are provided specifically for emergency use for patients who might become ill on the vehicle.

- See section 9.8 for advice on which oxygen-delivery devices should be carried in ambulances.

### 10.4 Oxygen carriage in other vehicles and in primary care settings and patients homes.

#### 10.4.1 Oxygen Carriage in private cars (Health and Safety Executive Guidance, Reference 302, 303)

When travelling by car, patients have the freedom to carry their own portable oxygen cylinder. Some General Practitioners in rural areas also carry oxygen in their cars. However, it is advised that certain safety precaution should be followed:

- It is good practice for the car to display a green warning triangle for ‘compressed gas’
- The cylinder should be secure within the car and cannot move during transport or in the event of an accident

#### 10.4.2 Medical centres and primary care practices

The majority of medical centres and practices should have a supply of oxygen for emergency use. Generally, cylinders with integral high flow regulators should be ordered. Otherwise, the cylinder must be fitted with a high-flow regulator capable of delivering a flow of over 6L/min in order to deliver medium and high-dose oxygen therapy. A recommended list of oxygen delivery devices for use in pre-hospital care is given in section 9.8

- Emergency oxygen should be available in primary care medical centres; preferably using oxygen cylinders with integral high-flow regulators. Alternatively, oxygen cylinders fitted with high-flow regulators (delivering over 6 L/min) must be used. See recommendation 74
10.4.3 Patient homes
In patients' homes, oxygen is either provided for long term therapy where an oxygen concentrator is provided (with or without a lightweight cylinder for ambulatory needs) or for short term/short burst therapy. Long term oxygen therapy is covered in other guidelines. This existing home oxygen supply may be used by a patient or GP in an emergency situation prior to the arrival of an ambulance.

The patient/carers should be made aware of the following Health and Safety recommendations (302)

- All cylinders should be stored on a cylinder trolley or suitably secured so they cannot be knocked over
- There should be no trailing oxygen tubing
- A green warning triangle for ‘compressed gas’ should be displayed by the front door (warns emergency services in the event of a fire)
- The minimum number of cylinders should be stored in the house
- There should be no smoking in the vicinity of oxygen cylinders
- Cylinders must be checked regularly for obvious signs of leakage
- Cylinders must be kept out of direct sunlight
- Oxygen must not be used near a naked flame

10.5 Oxygen delivery systems in hospitals.
Most hospitals have piped oxygen systems as described previously, although some wards can still be found where piped oxygen is not available and large compressed gas cylinders are used to supply the oxygen. Acute hospitals can spend up to £100,000 per annum on liquid oxygen so any device that uses lower oxygen flow rates could have significant economic savings for hospitals (e.g. nasal cannulae instead of simple face mask for medium dose oxygen).

10.5.1 Post-operative care on general surgical wards.
Medium concentration masks and nasal cannulae are usually sufficient (target saturation 94-98% if aged below 70 and 92-98% if aged 70 and above) except for patients with known significant COPD who should receive oxygen from a 24% or 28% Venturi mask or 1-2 l/min from nasal cannulae aiming at a saturation range of 88-92%.

10.5.2 Emergency departments
Medium or high concentration oxygen is normally used, (via Nasal Cannulae, Simple face mask or Reservoir Mask) but particular attention should be given to patients who have type II respiratory failure when a 24% or 28% Venturi mask or nasal cannulae at a flow rate of 1-2 l/min would be appropriate.
10.5.3 General wards and respiratory wards

The method of oxygen delivery will depend on the following circumstances:

- Expected duration of treatment.
- Type of respiratory illness.
- Pattern of breathing (high or low respiratory rate and drive).
- Need for humidification.
- Risk of carbon dioxide retention.
- Presence of confusion and its effect on potential compliance.

Nasal cannulae, simple face masks, reservoir masks and Venturi masks should be used where appropriate (see table 1). Nasal cannulae at flow rates or 0.5 to 1 l/min are sometimes used as a substitute for Venturi masks in acute or post-acute COPD patients on Respiratory wards (adjusting flow as necessary to achieve the desired ABG). This practice requires the use of paediatric flow meters to ensure consistent and finely-calibrated oxygen delivery and is not recommended outside of specialist units.

10.5.4 Devices used in emergency oxygen therapy.

Based on the advantages of each delivery system discussed above, the following recommendations are made for delivery of oxygen in medical emergencies. It is likely that additional equipment will be maintained in specialist units but specialist treatment is outside the scope of the present Guideline.

Recommendations

86 Most patients can be managed with one of 4 types of oxygen delivery device. Grade D

1. High concentration reservoir mask (non-rebreath mask) for high-dose oxygen therapy.
2. Nasal cannulae or simple face mask for medium and low dose oxygen therapy. (Nasal cannulae are the preferred option unless there is a specific reason to use a simple face mask)
3. 24% or 28% Venturi mask for patients with known previous hypercapnic respiratory failure with inappropriately high arterial blood oxygen values.
4. Tracheostomy masks for patients with tracheostomy or previous laryngectomy.

87 The high-dose reservoir mask at 10-15 l/min is the preferred means for delivering high dose oxygen to critically ill patients. Grade D

88 Nasal cannula should be used rather than simple face masks in most situations requiring medium-dose oxygen therapy, Nasal cannulae are preferred by patients for reasons of comfort and they and less likely to be removed during meals etc. (see section 10.2.4) There is also a cost saving. Grade C

89 The flow rate from nasal cannulae should be adjusted between 2 and 6 litres per minute to achieve the desired target saturation. Grade C

90 The flow rate from simple face masks should be adjusted between 5 and 10 litres per minute to achieve the desired target saturation. Grade C

91 Venturi Masks are recommended for patients at risk of hypercapnic respiratory failure. Venturi masks can deliver a constant FIO2 of 24% or 28% oxygen with a greater gas-flow than a simple face mask. This achieves a reduced risk of carbon-dioxide rebreathing compared with a simple face mask and less likelihood of dilution of the oxygen stream by room air if the patient’s inspiratory flow rate exceeds the flow rate delivered by the face-mask. Grade D
92 Venturi masks can be substituted with nasal cannulae at low flow rates (1-2 l/min to achieve the same target range) once patients have stabilized. Grade D

93 Patients with a respiratory rate above 30 breaths per minute should have the flow rate set to 50% above the minimum flow rate specified on the Venturi mask and/or packaging. Grade C.

10.6 Use of humidified oxygen

10.6.1 Rationale for use of humidified oxygen
The upper airway normally warms, moistens and filters inspired gases. When these functions are impaired by a pathological process, or when they are bypassed by an artificial airway, it is common practice to provide humidification. However, in the non intubated population there appears to be little scientific evidence of any benefit from humidified oxygen except that single doses of nebulised isotonic saline have been shown to assist sputum clearance (262, 263).

Recommendations

94 Humidification is not required for the short term delivery of low flow oxygen (up to 3 days oxygen). It is not therefore required in pre-hospital care. Grade B

95 In the emergency situation, humidified oxygen use can be confined to patients with tracheostomy or an artificial airway but these patients can be managed without humidification for short periods of time (e.g. ambulance journeys). Grade D

96 Humidification may also be of benefit to patients with viscous secretions causing difficulty with expectoration. This benefit can be achieved using nebulised normal saline. Grade C

10.6.2 Use of bubble humidification systems
Humidified oxygen is widely administered in hospitals across the UK and this is presumed to alleviate nasal and oral discomfort in the non intubated patient. Humidification of supplemental oxygen is commonly delivered by bubbling oxygen through either cold or warm sterile water before it reaches the patient. However, bubbling oxygen through cold water does not increase the humidity of the oxygen, it just distributes some water droplets along the tubing. Their effect on patient comfort is negligible. (147, 251) They do, however, represent an infection hazard and should not be used. (153)

- There is no evidence of clinically significant benefit from "bubble bottle" systems but there is an infection risk. Evidence level III

Recommendation

97 Bubble bottles should not be used because there is no evidence of clinically significant benefit but there is an infection risk. Grade C

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10.6.3 Large volume nebulisation-based humidifiers

If humidification is required, it should ideally deliver the inspired gas at temperature of 32-36ºC. Cold water humidifiers are simple and inexpensive but less efficient than a warm water system (about 50% relative humidity at ambient temperatures). The warm water option is more effective, targeting a relative humidity of 100% but both systems are thought to be a potential infection control risk. Warm water humidifiers are expensive and mostly confined to intensive care units and high dependency units and thus outside the scope of this guideline.

Newer humidifying systems are really a "giant nebuliser" with a one litre reservoir of saline of sterile water and an adjustable Venturi device. These systems are attached directly to the oxygen flow meter and connected to an aerosol mask via Flex tube. They allow delivery of precise oxygen concentrations of 28, 35, 40 and 60% oxygen via their Venturi device. This requires a specific oxygen flow rate as well as adjusting the Venturi nozzle on the device. It is possible to deliver 24% oxygen using a special adaptor. These large volume humidifiers have a high humidification output. The main indication for use is to assist with expectoration of viscous sputum. There are no published randomised studies involving these devices but it has been shown that single doses of nebulised saline can assist sputum production and relieve breathlessness in patients with COPD. (262, 263)

- Patients requiring high flow rates or longer-term oxygen might benefit from a large volume oxygen humidifier device, especially if sputum retention is a clinical problem. Evidence level III
- In the absence of an artificial airway the decision to humidify supplemental oxygen needs to be made on an individual basis but this practice is not evidence-based. Evidence level IV

10.7 Use of oxygen in patients with tracheostomy or laryngectomy.

The number of patients with a tracheostomy being cared for in a ward setting is increasing as critical care colleagues use this as a method of facilitating weaning from mechanical ventilation. In the absence of a pressurised circuit, oxygen is predominantly delivered via tracheostomy mask. This is a variable performance device and delivers concentrations up to 60-70%. If the patient deteriorates and requires an elevated oxygen concentration exceeding the concentration that a variable performance interface can deliver (60-70%), then it will be necessary to seek an alternative delivery system, usually a T-piece device fitted directly to the tracheostomy tube.

With a mask system, the interface will be connected to a humidification system via elephant tubing. As inserting a tracheostomy tube bypasses the patient’s natural mechanisms to warm and moisturise inspired gases, it is essential to humidify any supplemental oxygen being delivered to the tracheostomised patient. This will help maintain a patent tracheostomy tube, reducing the build up of secretions within the inner tube or the tracheostomy itself and minimise any subjective discomfort that the patient may experience.

Recommendation

98 When oxygen is required by patients with prior tracheostomy or laryngectomy, a tracheostomy mask (varying the flow as necessary) should achieve the desired oxygen saturation (tables 1 to 4). An alternative
delivery device, usually a two piece device fitted directly to the tracheostomy tube may be necessary if the patient deteriorates. Grade D
Recommendations

99 For patients with asthma, nebulisers should be driven by piped oxygen or from an oxygen cylinder fitted with a high flow regulator capable of delivering a flow rate over 6L/min. The patient should be changed back to their usual mask when nebuliser therapy is complete. If the cylinder does not produce this flow rate, then an air-driven nebuliser (with electrical compressor) should be used with supplemental oxygen by nasal cannulae at 2-6L/m to maintain an appropriate oxygen saturation level. Grade D

100 When nebulised broncho-dilators are given to hypercapnic acidotic patients, they should be driven by compressed air and, if necessary, supplementary oxygen should be given concurrently by nasal prongs at 2-4 litres per minute to maintain an oxygen saturation of 88-92%. The same precautions should be applied to patients who are at risk of hypercapnic respiratory failure prior to the availability of blood gas results. Grade D

101 Once the nebulised treatment is completed for patients at risk of hypercapnia, controlled oxygen therapy with a fixed concentration (Venturi) device should be re-instituted. Grade D

- During ambulance journeys, oxygen driven nebulisers may be used in the absence of an air-driven compressor system. If oxygen is used, it should be limited to 6 minutes for patients with known COPD. This will deliver most of the nebulised drug dose but limit the risk of hypercapnic respiratory failure. *See Recommendation 83*
Section 11

Prescription, administration and monitoring of oxygen therapy

11.1 Safe prescription and administration of oxygen

11.1.1 Why oxygen needs to be prescribed
Medical oxygen is as a drug. It is prescribed for hypoxaemic patients to increase oxygen tension and decrease the work of breathing necessary to maintain a given arterial oxygen tension. The concentration of oxygen required depends on the condition being treated; an inappropriate concentration may have serious or even lethal effects. Appropriate target saturation ranges for common medical emergencies are given in section 8 of this guideline and in table 1.

11.1.2 Implementing an oxygen prescription policy
Oxygen prescriptions should include starting dose and initial mode of delivery and whether the oxygen therapy should be continuous or as required. The most important aspect of the prescription is to give a target range. The clinicians who administer oxygen (usually nurses or physiotherapists) should be trained and empowered to adjust the oxygen dose upwards and downwards as necessary to maintain the patient in the target saturation range. This will require all hospitals to have an agreed oxygen administration programme with universal access to educational materials about oxygen administration. The clinicians who monitor the patients oxygen saturations (often Health Care Assistants) should be trained to inform those who have been trained to administer oxygen if the oxygen saturations fall above or below the target saturations. Those doing the monitoring should also understand the importance for the patient of keeping in the target range. Implementing this policy will require all hospitals to have training programmes for all clinical staff and regular training programmes in the safe use of oxygen and audit of outcomes.

11.1.3 Administration and monitoring of oxygen therapy
The appropriate device should be used to provide the prescribed oxygen and the effects should be monitored using pulse oximetry, monitoring of respiration rate and close observation of the patient. Arterial or capillary blood gas analysis should be repeated if clinical progress is not satisfactory and in all cases of hypercapnia and acidosis.

11.1.4 Education of health professionals
The clinician or health care professional administering the oxygen therapy should be aware of the hazards of hypoxaemia and hyperoxaemia and the signs and symptoms of inadequate or excessive oxygen delivery.

11.1.5 How to prescribe oxygen effectively
In the past, oxygen was often not prescribed at all or prescribed on a standard hospital drug chart as "Oxygen". It was unusual for the prescription to include full details of what device to use, what flow rate(s) to administer and whether
the prescription was for a fixed dose of oxygen or to aim at a specific oxygen saturation target. (55, 100, 180, 321-324)

It has been shown that a purpose-designed oxygen prescription sheet can improve oxygen prescribing in the short term (321) but experience has shown that free-standing oxygen prescription charts are often forgotten and unused. Recent audit studies by members of the Guideline group (not yet published) have shown improved standards of prescribing with the use of a pre-printed section for oxygen use in all hospital drug charts. This system was further enhanced by setting a desired saturation range for each patient. Suggested target saturation for common medical conditions are given in sections 8 and 9 of this Guideline. It is important that health care professionals where oxygen is administered are familiar with the optimal saturation ranges for common conditions (summary guideline for hospitals; appendix 2) and it is also important that those delivering the oxygen are familiar with the equipment in use and the best types of device to deliver low, medium and high dose oxygen therapy. Chart 3 shows a working example of a pre-printed oxygen section for a hospital prescription chart, and charts 1 and 2 give advice to prescribers and advice to those delivering oxygen on wards.

The safe use of oxygen includes careful consideration of the appropriate delivery device (mask, cannulae etc) together with an appropriate source of oxygen and an appropriate oxygen flow rate.

- For hypoxaemic patients, oxygen therapy should continue during other treatments such as nebulised therapy. See recommendations 100-101

11.1.6 Recommendations for safe prescribing and safe administration of oxygen:

102 Every health care facility should have a standard oxygen prescription document or, preferably, a designated oxygen section on all drug prescribing cards. Grade C

- Oxygen saturation should be measured in all breathless patients and supplemental oxygen should be given to all breathless hypoxaemic patients and to all critically ill patients. Oxygen saturation should be measured under as optimal conditions as possible e.g. nail varnish should be removed. See recommendation 8

- Clinicians should assess the clinical status of the patient prior to prescribing oxygen and the patient's condition should be reassessed frequently during oxygen use. See recommendations 11-12

103 All oxygen should be prescribed except in life-threatening emergencies when it must be started immediately. Grade D

104 Doctors should prescribe oxygen using a target saturation range (Sections 6, 8, 9 and 11) and sign the drug chart. (Chart 3) Grade D

105 In all situations where repeated blood gases are required, they should be measured as soon as possible to determine if the proposed target saturations are appropriate. Grade D

106 The oxygen dose should be increased by staff who have been trained to administer oxygen if the oxygen saturation falls below the pre-specified range and the dose should be reduced if the saturation rises above this range. If the monitoring of oxygen saturations is performed by other
staff (e.g. Health Care Assistants) they should inform staff who are trained to administer oxygen if the oxygen saturation is above or below the target saturation. Grade D Also see recommendations 115, 116, 122, 124 and 136

107 All clinicians prescribing oxygen should have appropriate training and access to written or electronic oxygen prescribing guidelines based on this national guideline. Grade D

108 Every hospital should have a training programme to ensure that clinical staff are familiar with the hospital’s oxygen administration policies. Grade D

11.1.7 How to use the oxygen therapy drug chart (Chart 3)

Recommendations

109 In most emergency situations, oxygen is given to patients immediately without a formal prescription. However, a subsequent written record must be made of what oxygen therapy has been given to every patient (in a similar manner to the recording of all other emergency treatment). Grade D

110 The prescription should be signed by the doctor or other prescribing clinician and the target saturation range circled on the drug chart. Grade D

111 Nurses should sign the drug chart at every drug round and check that that the patient is receiving oxygen therapy. Grade D

112 Most patients are prescribed continuous oxygen. However some patients may be prescribed oxygen PRN (as required). In this scenario if patients are on air at the time of the drug round, nurses should still sign the drug chart but the observation chart should be filled in using the code AX. (see Chart 4) Grade D

11.2 Starting oxygen therapy

Safe prescribing and safe administration of oxygen are closely linked. In emergencies, oxygen therapy should be started immediately and prescribed as soon as possible. In all other situations, oxygen should be prescribed in accordance with the standards described in section 11.1 before administration is commenced. The health care professional who administers the oxygen therapy (usually a nurse or physiotherapist) should be fully trained and should follow local or national protocols as described in section 11.1

Recommendations

- The administering health care professional should note the oxygen saturation prior to commencing oxygen therapy. See recommendation 8

113 The health care professional should commence oxygen therapy using an appropriate delivery system and flow rate as specified in sections 8, 9 and 10 of this guideline. The target oxygen saturation and whether the patient is having continuous oxygen, PRN oxygen or no oxygen therapy should be added on the respiratory section of the observation chart. Grade D

114 Whenever possible, patients should be given an oxygen information sheet (example in appendix 5 of this Guideline) Grade D
11.3 Monitoring oxygen therapy

11.3.1 Pulse oximeters.
Pulse oximetry should be available to all health care professionals managing patients receiving oxygen therapy and they should be trained in their use. (see section 7 for technical and practical information regarding oximeter use). Clinicians should be aware that pulse oximetry gives no information about the PCO$_2$ or pH and most pulse oximeters are unreliable when a patient’s SpO$_2$ falls below about 85%. Pulse oximetry is dependent on pulsatile flow and it may not be possible to achieve a satisfactory oximeter reading in patients with cold hands, especially with severe Raynaud’s phenomenon due to collagen vascular diseases (which may also cause hypoxic lung disease). The readings may also be affected by shock, skin pigmentation, nail varnish etc – see section 7. It is essential to record the oxygen delivery system alongside the oximetry result.

- All measurements of oxygen saturation should be recorded in the observation chart along with the code for the oxygen delivery system that is being used (including the various codes if the patient is breathing air) Chart 4. See recommendation 8.

11.3.2 Recommendations for use of Arterial or arterioles capillary blood gases

- Arterial or arterioles capillary blood gases should be measured and the inspired oxygen concentration noted on arrival at hospital (or at the time when oxygen therapy becomes necessary) for most patients requiring emergency oxygen therapy. See recommendation 25

- Blood gases should be repeated in all critically ill patients and in many other cases according to response to treatment. See recommendation 25

11.3.3 Physiological monitoring;

Early Warning Scoring systems (EWS or mEWS) are useful for monitoring patients. Evidence Level III

- Tachypnoea is a sensitive indicator of deteriorating respiratory function. Evidence Level III

- All acutely ill patients should have physiological monitoring using Early Warning Scores or a similar physiological assessment system in addition to pulse oximetry. See recommendation 11

11.3.4 Monitoring during the first hour of oxygen therapy

Recommendations

115 All patients should have their oxygen saturation observed for the first five minutes after starting oxygen therapy. Grade D
116 If the oxygen saturation should fall below the target saturation and the patient is unstable medical advice should be sought. Grade D

117 If the oxygen saturation is above the target saturation range and the patient is stable, the delivery system and oxygen flow rate should be reduced accordingly. Grade D

118 Patients who have a target saturation of 88-92% should have their blood gases measured within 30-60 minutes. This is to ensure that the carbon-dioxide level is not rising. This recommendation also applies to those who are at risk of developing hypercapnic respiratory failure but who have a normal PaCO2 on the initial blood gas measurement. Grade D

119 Stable patients whose oxygen saturation is within their target saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above do not need repeat blood gases within 30-60 minutes if there is no risk of hypercapnia and acidosis and may not need any further blood gas measurements. Grade D

11.3.5 Subsequent monitoring
The exact requirements for monitoring will depend on the clinical condition of each patient. Saturations are usually monitored after one hour of oxygen therapy then four hourly subsequently. However, critically ill patients will require continuous monitoring of oxygen saturation and other physiological measurements.

Recommendations

120 Stable patients on oxygen treatment should have SpO2 and mEWS measured four times-a-day. Grade D

121 In those who are unstable, oxygen saturation should be monitored continuously and the patient should ideally be managed in a high-dependency area. Grade D

122 If the patient is clinically stable and the oxygen saturation is within the target range treatment should be continued (or eventually lowered) depending on the clinical situation. Grade D

123 Any sudden fall in oxygen saturation should lead to clinical evaluation of the patient and, in most cases, measurement of blood gases. Grade D

124 Oxygen therapy should be increased if the saturation is below the desired range and decreased if the saturation is above the desired range (and eventually discontinued as the patient recovers). Grade D

125 Monitor the saturation continuously for 5 minutes after any increase or decrease in oxygen dose to ensure that the patient achieves the desired saturation range. Grade D

126 Record the new saturation (and the new delivery system) on the patient's observation chart after 5 minutes of therapy at the new oxygen dose. Each change should be recorded by the clinician trained to administer oxygen by signing the observation chart (only changes should be signed for). Grade D

127 Repeat blood gases are not required for stable patients who require a reduced dose of oxygen (or cessation of oxygen therapy) to maintain the desired target saturation. Grade D

128 Patients with no risk of hypercapnia do not always need repeat blood gases after an increase in oxygen dose but should have clinical review to determine why the oxygen saturation has fallen. Grade D

129 Patients at risk of hypercapnia (usually those with a target range of 88-92%; see Table 3) require repeat blood gas estimation 30-60 minutes after an increase in oxygen therapy. Grade D

130 For patients with no risk of hypercapnia, monitoring by pulse oximeter is sufficient (repeated blood gases not required) provided the saturation remains in the desired range, usually 94-98% if aged below 70 and 92-98% if aged 70 and above. Grade D
11.3.6 When to increase oxygen therapy

In most instances, failure to achieve the desired oxygen saturation is due to the severity of the patient's illness but it is worth checking that the oxygen delivery device and the oxygen flow rate is correct. If the oxygen is being delivered from a cylinder, clinicians should check the labelling of the cylinder to ensure that it is an oxygen cylinder and checks should be made to ensure that the cylinder is not empty or near-empty.

Recommendations

131 If a patient's oxygen saturation is lower than the prescribed target range, first check all aspects of the oxygen delivery system for faults or errors. Grade D

132 If a patient's oxygen saturation is consistently lower than the prescribed target range, there should usually be a medical review and the oxygen therapy should be increased according to an agreed written protocol. Grade D

133 The patient should be observed for five minutes after oxygen therapy has been increased. Grade D

134 If the oxygen saturation fails to rise following 5-10 minutes of increased oxygen therapy or if there is clinical concern following medical review, then blood gases should be repeated. Grade D

135 If the target saturation is between 88-92% range, blood gases should be repeated at 30-60 minutes after any increase in oxygen therapy to ensure that the carbon dioxide level is not rising. Grade D

11.3.7 When to lower oxygen therapy

Most conditions which require supplemental oxygen therapy will improve with treatment and it will then be possible to reduce the amount of oxygen administered to the patient. Improvement will usually be confirmed by observing an improving oxygen saturation and a reduction in the physiological score on the mEWS observation chart as discussed in section 7.

Recommendations

136 Lower the oxygen dose if the target saturation is higher than the prescribed range. Grade D

137 Lower the oxygen dose if the patient is clinically stable and the oxygen saturation has been in the upper zone of the target range for some time (usually 4-8 hours). Grade D

138 Saturations should be observed for five minutes following a change of oxygen therapy. Grade D

139 If the target saturation is maintained, the new delivery system and flow should be continued. Repeat blood gases are not required. If the patient is stable the process can be repeated and the patient can eventually be weaned off oxygen-see section 12. Grade D
Section 12

Weaning and discontinuation of oxygen therapy.

In most acute illnesses, oxygen therapy will be reduced gradually as the patient recovers and oxygen therapy can be discontinued once the patient can maintain a saturation of 94-98% if aged below 70 and 92-98% if aged 70 and above whilst breathing air (or the patient’s baseline oxygen saturation level if known). However, some patients may be continued on oxygen therapy to palliate breathlessness, often on a “PRN” Basis (as required, not continuous). Some patients may have episodic hypoxaemia during recovery from acute illness (e.g. COPD patients with intermittent mucus plugging) and some convalescent patients may be comfortable at rest with a normal oxygen saturation but may desaturate and become breathless when they start to mobilise. There is no evidence that oxygen, either before or after exercise, is of benefit to non-hypoxaemic patients either by relieving breathlessness or by shortening length of stay in hospital. More research in this area is needed.

Some patients with chronic lung diseases will already be established on long-term oxygen therapy and should be tapered slowly to their usual maintenance dose of oxygen.

A small number of patients who have suffered major respiratory or cardiac injury may require a prescription for home oxygen to permit safe discharge from hospital. However, many patients with COPD exacerbations may have a low PaO2 on discharge from hospital but a reasonable PaO2 at a subsequent clinic visit so decisions about long-term oxygen should not be made on the basis of blood gas measurements made during acute exacerbations of COPD, see Royal College of Physicians clinical guideline for domiciliary oxygen services. (301)

12.1 How to discontinue oxygen therapy for stable patients

Recommendations

- Reduce oxygen therapy gradually for stable patients. See section 11.3.7 and recommendations 136-139

140 The lowest dose of oxygen for most stable convalescent patients will be 2 litres per minute via nasal cannulae and 1 litre per minute via nasal cannulae or a 24% Venturi mask for patients at risk of hypercapnic respiratory failure. Grade D

141 Stop oxygen therapy once a patient is clinically stable on low-dose oxygen and the oxygen saturation is within the desired range on 2 consecutive observations. Oxygen should also be stopped if the patient is on a written protocol of timed oxygen e.g post-operatively. Grade D

142 Monitor the oxygen saturation on room air for 5 minutes after stopping oxygen therapy. If it remains in the desired range, recheck at one hour. Grade D

143 If the oxygen saturation and mEWS is satisfactory at one hour, the patient has safely discontinued oxygen therapy but continue to monitor saturation and mEWS on a regular basis according to the patient’s underlying clinical condition. Grade D

144 If the saturation falls on stopping oxygen therapy, recommence the lowest dose that maintained the patient in the target range and monitor for 5 minutes. If this restores the saturation into the target range, continue oxygen therapy at this level and attempt discontinuation of oxygen therapy again at a later date provided the patient remains clinically stable. Grade D

145 If a patient requires oxygen therapy to be restarted at a higher dose than before to maintain the same target saturation range, then the patient should have clinical review to establish the cause for this deterioration. Grade D

146 Some patients may have episodic hypoxaemia (e.g. after minor exertion) after they have safely discontinued continuous oxygen therapy. If these patients require intermittent oxygen therapy, they should have a prescription for oxygen as required (“PRN”). Grade D
Cross oxygen off the drug chart when oxygen discontinued (and sign to confirm discontinuation).

Grade C
Section 13

Outcomes and audit

13.1 Audit

One year after publication of this guideline, the Emergency Oxygen Guideline Committee will organize a UK-wide audit to establish how many hospital, PCT providers and ambulances services have introduced this guideline. Audit of oxygen use within individual trusts will also be encouraged strongly.

13.2 Audit of compliance with guidelines.

It is recommended that all users of oxygen will audit their own practice against the suggested optimal practice suggested in this Guideline. This applies especially to high-frequency users such as Ambulance services, Emergency Departments, and medical wards.

“On the job audit”

Regular reviews of the drug card and observation chart on medical rounds at the pilot sites have been very important in the successful introduction of this policy. This has provided instant feedback for doctors, nurses and health care assistants and has produced successful change.

Whole hospital and ward audits

An audit tool has been developed at the pilot sites (Appendix 7). This has been successfully used hospital wide and on individual wards. It is suggested that audits are done soon after the introduction of the policy.

Other audit questions could include the following.

- Does the Trust have an oxygen prescribing policy based on this Guideline?
- Is this policy known to all staff and available at all times on the Trust Intranet?
- Was oxygen prescribed for every patient who was using oxygen at the time of the audit?
- Did every patient have a target oxygen saturation specified (and was this target appropriate)?
- Was oxygen given in accordance with the present guideline?
Section 14

Dissemination and implementation of the Guideline

14.1 Dissemination

Dissemination of this guideline will be encouraged and supported strongly by the Societies involved in the production of the Guideline. It is hoped that each specialist Society or College will alert members to the key recommendations in this Guideline. Copies will be sent to the Chief Executives, Medical Directors and Head Nurses of all Hospital and Primary Care Trusts and Ambulance Authorities in the UK and also to the directors of education at Nursing Schools, Medical Schools and at Education for Health (National Respiratory Training Centre).

14.2 Local guidelines

It is recognised that many Trusts tend to modify national guidelines for local use. Short, "user-friendly" versions of the Guideline website for acute hospitals, ambulance services and general practice are shown in Appendix 2 and 4 on the BTS. (www.brit-thoracic.org.uk/guidelines.html)

Educational materials will be made available on the BTS Website, to include Guideline Summaries and Flow Charts in addition to teaching slides. It is hoped that the shortened version of this guideline (or a customised local version) will be made available on the website of every NHS Trust.

14.3 Local oxygen policy

It is usual for a new policy to be presented to the local policy committee. A specimen example of a local policy is available in Appendix 3 to help with the production of this policy in individual Trusts.

14.4 New prescription chart

The introduction of the guideline will require a new “oxygen section” in the prescription chart in all hospitals. A specimen example is available in Chart 3. It is recommended, after experience at the two pilot sites, that oxygen should be at the start of the prescription chart because so many patients are prescribed oxygen. The oxygen prescription may be missed if it is placed in another part of the drug chart.

14.5 Staff education

Medical staff education will be required before the introduction of the guideline and regularly subsequently. Teaching slides are available on the BTS website. It is thought that these would be suitable for FY1, FY2 and Specialty Training lectures and other educational material. They are also suitable for undergraduate medical education. Nursing staff and nursing students will also require education. Lecture studies have also been produced for this purpose and are available on the BTS website. It is suggested that small groups of 5-10 nurses from wards are taught in sessions lasting 30 minutes each day before the introduction of the guideline locally. This has been found to be more successful than relying on training days. We believe that this would take too long to train enough staff adequately. Slides are available for ambulance staff and primary care staff on the BTS website.

14.6 Local champions

The guideline committee has tried to identify local champions in hospitals, PCT providers and ambulance services who will help introduce these guidelines. The local champions are listed in Appendix 13 on the BTS website. It is hoped that the champions will help organise the introduction of a local guideline and oxygen policy, a new prescription chart and help organise staff education.
Clinical Governance Leads will also need to become committed to the implementation of the Emergency Oxygen Guideline and audit of this process.

**14.7. Benefits of nationwide implementation**

One major benefit of nationwide implementation will be that when staff transfer between organisations that they will be familiar with the oxygen prescription and administration system.
Section 15.
Areas requiring further research

Because of the life-and-death nature of many conditions for which emergency oxygen therapy is used, it seems that clinicians have been wary of conducting controlled trials of oxygen therapy for most of the commoner indications. It is worrying that the limited number of existing trials of oxygen therapy given to non-hypoxaemic patients in common conditions such as heart attacks, strokes, and difficult labour have failed to show benefit and there have been suggestions of possible harm in these trials despite the near-universal use of oxygen for such conditions in the past.

Further research is required in many areas including:

- Use of oxygen in myocardial infarction
- Use of oxygen in unstable coronary syndromes
- Use of oxygen in chest pain of presumed cardiac origin
- Use of oxygen in obstetric emergencies
- Benefits of alert cards and personalised oxygen masks for patients with prior hypercapnic respiratory failure
- Clinical outcomes of patients exposed to hyperoxia
- Studies to determine if different types of oxygen mask can affect clinical outcomes
- Comparisons of nasal cannulae and simple face masks after abdominal surgery
- Studies to determine if implementation of this Guideline improves patient outcomes
- Prospective studies to establish the ideal target saturation range in patients with exacerbated COPD
- Prospective studies of the effect of oxygen in non-hypoxaemic patients with major trauma and head injury
- Audit studies of survival outcomes in patients given oxygen therapy.
- Oxygen levels and outcomes in a wide range of conditions.
- Use of oxygen “PRN” or as required for non-hypoxaemic patients
Section 16

Membership of working party.

**PHYSICIANS IN GENERAL/RESPIRATORY MEDICINE**
Ronan O’Driscoll, Salford, Co-Chair, British Thoracic Society
Tony Davison, Southend, Co-Chair, British Thoracic Society
Mark Elliott, Leeds, Royal College of Physicians

**RESPIRATORY PHYSICIANS**
Wisia Wedzicha, London, British Thoracic Society and Editor Thorax

**EMERGENCY MEDICINE**
Prof Kevin Mackway-Jones, Manchester, British Association for Emergency Medicine

**EMERGENCY PHYSICIAN (EAU)**
Paul Jenkins, Norwich, Society for Acute Medicine (SAMUK)

**INTENSIVE CARE**
Roop Kishen, Salford, Intensive Care Society

**GENERAL PRACTITIONER**
Mark Levy, London, Royal College of General Practitioners and GP in Airways Group (GPIAG).

**NURSE**
Susan Perrott, Cambridge, Royal College of Nursing

**PHYSIOTHERAPIST**
Leigh Mansfield, RVI Newcastle, Chartered Society of Physiotherapy

**ARTP REPRESENTATIVE**
Angela Evans, North Staffs, Association for Respiratory Technology and Physiology

**PATIENT / LAY REPRESENTATIVE**
Sarah Panizzo, London, Nominated by Royal College of Physicians

**AMBULANCE SERVICE REPRESENTATIVES**
Fionna Moore, London, Joint Royal Colleges Ambulance Liaison Committee

David Whitmore, London, British Paramedic Association
Simon Gibbs, London, Senior Clinical Advisor to the Medical Director, LAS

**CARDIOLOGIST**
Simon Gibbs, London, British Cardiovascular Society

**ANAESTETIST**
Bruce Martin, London, Royal College of Anaesthetists

**OBSTETRICIAN**
Kim Hinshaw, Sunderland, Royal College of Obstetricians and Gynecologists
Section 17

Acknowledgements

All societies who helped but are not formally affiliated to the guideline
All individuals who helped us formally or informally
Peer Reviewers

This section to be completed once guidelines are almost ready for publication
**Chart 3**

**Working example for oxygen section in hospital prescription charts**

*(Two panels are required on the prescription chart because oxygen may change from continuous to PRN as a patient improves)*

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**OXYGEN** The method and rate of oxygen delivery should be altered by nurses or other health care professionals in order to achieve the target saturation range as per hospital guideline. For most conditions, oxygen should be prescribed to achieve a target saturation of 94-98% for patients aged below 70 and 92-98% for those aged 70 or above or 88-92% for those at risk of hypercapnic respiratory failure. The nurse should sign this prescription chart on every drug round. The delivery device and flow rate should be recorded alongside the oxygen saturation on the bedside observation chart or mEWS (Early Warning Score) chart.

*Saturation is indicated in almost all cases except for palliative terminal care.

### Drug Oxygen

<table>
<thead>
<tr>
<th>Saturation Range</th>
<th>Date Administered</th>
</tr>
</thead>
<tbody>
<tr>
<td>88-92%</td>
<td>06</td>
</tr>
<tr>
<td>92-98%</td>
<td>09</td>
</tr>
<tr>
<td>94-98%</td>
<td>14</td>
</tr>
<tr>
<td>Other</td>
<td>18</td>
</tr>
<tr>
<td>Other</td>
<td>22</td>
</tr>
</tbody>
</table>

**Circle target oxygen saturation**

- 88-92%
- 92-98%
- 94-98%
- Other

**Starting device/flow rate**

PRN / Continuous (Refer to O2 Guideline)

Tick here if saturation not indicated *

Date and Signature

PRINT NAME

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### Drug Oxygen

<table>
<thead>
<tr>
<th>Saturation Range</th>
<th>Date Administered</th>
</tr>
</thead>
<tbody>
<tr>
<td>88-92%</td>
<td>06</td>
</tr>
<tr>
<td>92-98%</td>
<td>09</td>
</tr>
<tr>
<td>94-98%</td>
<td>14</td>
</tr>
<tr>
<td>Other</td>
<td>18</td>
</tr>
<tr>
<td>Other</td>
<td>22</td>
</tr>
</tbody>
</table>

**Circle target oxygen saturation**

- 88-92%
- 92-98%
- 94-98%
- Other

**Starting device/flow rate**

PRN / Continuous (Refer to O2 Guideline)

Tick here if saturation not indicated *

Date and Signature

PRINT NAME

---
**Chart 4**

**Working example for respiratory section of observation chart**

<table>
<thead>
<tr>
<th>Respiratory Rate</th>
<th>Example 20</th>
<th>PRN</th>
<th>Not on oxygen therapy</th>
<th>Target range: 88-92%</th>
<th>92-98%</th>
<th>94-98%</th>
<th>Other_____</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen Saturation %</td>
<td>94%</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oxygen Device or Air</td>
<td>N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oxygen flow rate L/min</td>
<td>4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Your Initials* LW

*All changes to oxygen delivery systems must be initialled by a registered nurse or equivalent.*

*If the patient is medically stable and in the target range on two consecutive rounds, report to a registered nurse to consider weaning off oxygen.*

**Codes for recording oxygen delivery on observation chart**

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Air - patient not requiring oxygen therapy</td>
</tr>
<tr>
<td>AX</td>
<td>Measurement on air for a patient on PRN Oxygen therapy</td>
</tr>
<tr>
<td>AW</td>
<td>Measurement on air for a patient who is being weaned off oxygen but not yet discontinued on chart.</td>
</tr>
<tr>
<td>N</td>
<td>Nasal Cannulae</td>
</tr>
<tr>
<td>SM</td>
<td>Simple mask</td>
</tr>
<tr>
<td>V24</td>
<td>Venturi 24%</td>
</tr>
<tr>
<td>V28</td>
<td>Venturi 28%</td>
</tr>
<tr>
<td>V35</td>
<td>Venturi 35%</td>
</tr>
<tr>
<td>V40</td>
<td>Venturi 40%</td>
</tr>
<tr>
<td>V60</td>
<td>Venturi 60%</td>
</tr>
<tr>
<td>H28</td>
<td>Humidified oxygen at 28% (&quot;Quatro&quot; or similar device)</td>
</tr>
<tr>
<td>H35</td>
<td>For humidified oxygen at 35%</td>
</tr>
<tr>
<td>H40</td>
<td>For humidified oxygen at 40%</td>
</tr>
<tr>
<td>H60</td>
<td>For humidified oxygen at 60%</td>
</tr>
<tr>
<td>RM</td>
<td>Reservoir Mask</td>
</tr>
<tr>
<td>TM</td>
<td>Tracheostomy Mask</td>
</tr>
<tr>
<td>CP</td>
<td>Patient on CPAP system</td>
</tr>
<tr>
<td>NIV</td>
<td>Patient on NIV system</td>
</tr>
<tr>
<td>OTH</td>
<td>Other device: __________________________ (Specify which)</td>
</tr>
</tbody>
</table>
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(References are still in “random order” but will be strictly ordered by a publishing professional in the near future)

(End-Note or Reference Manager format)

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The Appendices that are shown in the attached file will not appear in the printed document, they will be available on the BTS website as a Web-only resource.