Domiciliary oxygen therapy is an effective but potentially expensive and inconvenient intervention. It should be prescribed only for patients in whom there is evidence of benefit, such as those whose disability relates to a chronic reduced arterial oxygen concentration (chronic hypoxaemia).

The most common cause of chronic hypoxaemia in Australia and New Zealand is chronic obstructive pulmonary disease (COPD). In hypoxaemic COPD, domiciliary oxygen is the only therapy (apart from smoking cessation) that reduces mortality. There is also evidence that it alleviates right heart failure caused by cor pulmonale, enhances neuropsychological function, and improves exercise performance and capacity to undertake the activities of daily living (E2).7

Although long-term oxygen therapy has been best studied in COPD, other possible indications include hypoxaemia associated with cyanotic congenital heart disease, severe congestive cardiac failure, diffuse interstitial lung disease, advanced lung cancer or cystic fibrosis,8 and, in general, any illness of which chronic hypoxaemia is an important feature. In the absence of hypoxaemia, oxygen therapy is unlikely to contribute usefully to relief of dyspnoea. Home oxygen therapy for patients with chronic heart failure and/or angina is not well supported by evidence of efficacy, and reduced mortality with this therapy has not been verified. An inspired-oxygen concentration of 50% may modestly improve exercise duration in heart failure,9 but concentrations this high are difficult to attain with current home delivery systems.

Indications for oxygen therapy

Continuous oxygen therapy (at least 15 hours/day)

Long-term continuous oxygen therapy should be considered for patients with stable chronic lung disease, particularly COPD, who have an arterial partial pressure of oxygen (PaO2) consistently ≤ 55 mmHg (7.3 kPa) when at rest, awake and breathing air. At assessment (see Investigations), the patient’s condition must be stable, and all reversible factors (such as anaemia) should be remediated.10 Because gas exchange may improve substantially on ceasing smoking, assessment should be made at least a month after the patient has stopped smoking.

Polycythaemia (haematocrit > 0.55), clinical or electrocardiographic evidence of pulmonary hypertension and/or episodes of right heart failure reflect the systemic effects of chronic hypoxaemia and strengthen the case for therapeutic oxygen use. Patients with these complications should be prescribed continuous oxygen therapy if their stable PaO2 is 56–59 mmHg (7.4–7.8 kPa). In COPD, continuous oxygen therapy is of most benefit for patients with increased arterial partial pressure of carbon dioxide (PaCO2) (> 45 mmHg or 6 kPa).4,11

As benefit has been shown to increase with increasing daily use of oxygen,9 patients should be advised to use oxygen whenever the physical restriction imposed by the therapy is not onerous. In two landmark randomised controlled trials, patients who were prescribed continuous oxygen therapy managed to use it for an average of 18 hours a day.5,4 These patients had reduced mortality compared with those using the oxygen 15 hours a day or less. Thus, the recommendation is generally that the oxygen be used for as many hours out of 24 as possible, within reason. The benefit of daytime oxygen use, which may restrict mobility, must be weighed against the benefit of exercise, which can improve quality of life (E1).12
Position on continuous oxygen therapy
Continuous oxygen therapy is indicated to improve longevity and quality of life when
• stable daytime PaO2 ≤ 55 mmHg (7.3 kPa) at rest (E1); or
• stable daytime PaO2 is 56–59 mmHg (7.4–7.8 kPa) and there is evidence of hypoxic organ damage (including right heart failure, pulmonary hypertension or polycythaemia) (E1).
Flow rate should be set to maintain PaO2 > 60 mmHg (8 kPa) (oxygen saturation level, measured by pulse oximetry [SpO2] > 90%) during waking rest. This will usually need to be increased by 1 L/min during sleep, exertion or air travel (E4).

Intermittent oxygen therapy
Intermittent or ambulatory oxygen therapy may be used as part of continuous oxygen therapy, in which case its benefits are those of long-term oxygen therapy. The known benefits of pulmonary rehabilitation on exercise capacity and quality of life in COPD patients (E1) support the use of ambulatory oxygen therapy in all mobile patients on long-term oxygen therapy to allow them to achieve their full potential in terms of these benefits as well as the reduced mortality from continuous oxygen therapy.

The subject of ambulatory oxygen use in patients who do not fulfill criteria for continuous oxygen therapy is controversial. A recent Cochrane review of the subject found that no firm conclusions could be made from available evidence about the effectiveness of this intervention in patients with COPD and only mild hypoxaemia. Nonetheless, it is known that ambulatory oxygen therapy may rapidly improve exercise capacity in patients with fibrotic or obstructive lung diseases, and benefit may occur irrespective of resting or exercise-induced hypoxaemia. Although there is no direct evidence that treatment of exercise-induced hypoxaemia retards long-term pulmonary hypertension or prolongs life, it may improve quality of life in patients who experience significant arterial oxygen desaturation during exercise (SpO2 < 88%).
A recent randomised crossover trial suggested that modest benefits in terms of emotional and social function, anxiety and depression levels and dyspnoea could be achieved in mildly hypoxaemic COPD patients who experienced desaturation with exertion (E2). In that trial, rapid beneficial response to supplemental oxygen did not predict longer-term benefits in quality of life, and our consensus view, in the absence of further data, is that evidence of short-term benefit should be demonstrated before this therapy is recommended. Our view is supported by the consensus statements of both the American Thoracic Society and the Royal College of Physicians, UK. Benefit should be established by comparing exercise endurance and degree of dyspnoea when breathing oxygen and when breathing air (using a 6-minute walk test, treadmill, stationary bicycle, or similar endurance test). Room air is probably adequate for this comparison, as there appears to be no difference in exercise endurance between breathing room air and cylinder air.

Ambulatory oxygen therapy may be particularly useful for patients in rehabilitation programs, including those awaiting lung transplantation or lung reduction surgery, to maintain an increased level of fitness and thus improve their prognosis.

Other patients for whom intermittent oxygen therapy is indicated include:

Position on intermittent oxygen therapy
• Ambulatory oxygen therapy may improve quality of life in patients who experience significant arterial oxygen desaturation during exercise (SpO2 < 88%) (E2).
• It is recommended that rapid improvements in dyspnoea or exercise capacity in response to oxygen therapy be demonstrated before this treatment is prescribed (E4).
• Ambulatory oxygen therapy may be useful for patients in rehabilitation programs, including those awaiting lung transplantation or lung reduction surgery, to maintain an increased level of fitness and thus improve their prognosis (E4).
• A small cylinder of oxygen for emergency use by the patient with severe asthma who is prone to sudden life-threatening episodes is recommended (E4).
• Home oxygen therapy may be appropriate to relieve symptoms in terminally ill patients, who will usually have a life expectancy of less than 3 months (E4).

Nocturnal oxygen therapy
Isolated episodes of hypoxaemia during sleep due to hypoventilation or worsening ventilation–perfusion inequality in patients with obstructive or fibrotic lung diseases should be distinguished from hypoxaemia associated with sleep apnoea caused by upper airway obstruction or obesity hypoventilation syndrome. Apnoea syndromes are diagnosed by overnight polysomnography, and generally require other forms of therapy (such as continuous positive airway pressure or nocturnal ventilation) rather than supplemental oxygen. In the absence of a high clinical pre-test probability of obstructive sleep apnoea or obesity hypoventilation syndrome, simple nocturnal oximetry is probably adequate to confirm isolated nocturnal hypoxaemia in COPD. Such isolated nocturnal hypoxaemia should be...
considered in patients whose arterial oxygen tension while awake is too high to prescribe continuous oxygen therapy but who have daytime somnolence, daytime hypercapnia, polycythaemia, pulmonary hypertension or right heart failure. The clinical importance of isolated nocturnal hypoxaemia in COPD is unclear. Although extrapolation from continuous oxygen therapy studies might suggest that such patients would benefit from nocturnal oxygen supplementation, results of studies have been conflicting. In one small study, nocturnal oxygen therapy at 3 L/min over 3 years was associated with a smaller rise in pulmonary artery pressure than in a control group receiving supplemental air. However, there was no effect on mortality. A larger 2-year study of patients with COPD and modest daytime hypoxaemia (PaO₂ 56–69 mm Hg [7.4–9.2 kPa]) who experienced oxygen desaturation to SpO₂ < 90% for over 30% of the night found no survival benefit in the group receiving oxygen supplementation and no effect on pulmonary haemodynamics. The overall mortality rate in the latter study was too small to draw any definite conclusion, and the treatment group did not consist entirely of patients with evidence for hypoxic damage. Although data are insufficient to make rigorous recommendations for this group, and further studies are needed, the current consensus is that nocturnal oxygen therapy may be indicated in patients whose nocturnal arterial oxygen saturation falls below 88% and who have evidence of hypoxia-related sequelae. Other patients whose oxygen saturation repeatedly falls below 88% for over a third of the night may also benefit (E4).

Position on nocturnal oxygen therapy

- Nocturnal oxygen therapy may be indicated to relieve demonstrated oxygen desaturation during sleep to SpO₂ ≤ 88% (PaO₂ < 55 mm Hg or 7.3 kPa) for more than a third of the night or in the presence of hypoxia-related sequelae (E4).
- The role of continuous positive airway pressure or other ventilatory support needs to be considered and may replace or complement oxygen therapy. If oxygen therapy is indicated, a concentrator will be the least expensive mode of delivery. It can also be used, at negligible extra cost, to provide oxygen during daytime exertion.

Contraindications for oxygen therapy

Oxygen therapy is not indicated for patients
- with severe airflow limitation whose main complaint is dyspnoea but who maintain a PaO₂ > 60 mm Hg (8 kPa) and who show no secondary effects of chronic hypoxia;
- who continue to smoke cigarettes (owing to the increased fire risk and the probability that the poorer prognosis conferred by smoking will offset treatment benefit);
- who have not received adequate therapy of other kinds (eg, inhaled and oral bronchodilators and corticosteroids, treatment for right ventricular failure or for any respiratory infection); and
- who are not sufficiently motivated to undertake the discipline required for oxygen therapy.

Investigations

The following investigations should be carried out to determine suitability for domiciliary oxygen therapy:
- Establish the nature and severity of the pulmonary disorder responsible for hypoxaemia by appropriate testing, including objective tests of pulmonary function.
- Undertake appropriate clinical, electrocardiographic, echocardiographic and radiological assessment of right heart failure and pulmonary hypertension.
- Measure haematocrit. Polycythaemia, the usual response to chronic hypoxaemia in otherwise healthy people, is not always seen in people with hypoxaemia of chronic lung disease. The degree to which it is adaptive or adds to the burden of disordered function through increased blood viscosity is controversial. Anaemia is always a burden and should be investigated and corrected.
- Undertake other appropriate tests, according to clinical findings, for other major diseases that might be expected to seriously limit survival. It is appropriate to prescribe oxygen therapy for symptomatic relief in patients with a very limited prognosis.

Initiation of oxygen therapy

Before introducing oxygen therapy, undertake optimal treatment of the pulmonary disorder while monitoring improvement with objective tests such as FEV₁ and vital capacity. Treatment may include maximum therapy for airway obstruction, attention to nutrition and bodyweight, an exercise rehabilitation program, control of infection, and treatment of cor pulmonale. When drug treatment and other therapy has been optimised and the patient's condition has stabilised over about 4 weeks, measure PaO₂ at rest and, when indicated, arterial oxygen saturation during sleep. It is recommended that, before initiating therapy, the goals of treatment be defined (ie, reversal or prevention of pulmonary hypertension, improved longevity, symptomatic alleviation of dyspnoea, improved quality of life) and clearly discussed with the patient.

In patients selected for oxygen therapy, assess the adequacy of relief of hypoxaemia (PaO₂ > 60 mm Hg [8 kPa], SpO₂ > 90%) and/or improvement in exercise capacity or nocturnal arterial oxygen saturation. If hypercapnia is present at baseline, it will be necessary to repeat arterial blood gas measurements to ensure there is minimal rise in PaCO₂ and fall in pH in response to supplemental oxygen.

Reassessment

Patients should be reassessed 1–2 months after starting continuous or nocturnal oxygen therapy, both clinically and by measurement of PaO₂ and PaCO₂. It should then be decided whether the treatment has been properly applied and whether it is worthwhile or should be abandoned. Indices that may be used in weighing the benefits of continuing therapy include whether the patient is refraining from smoking, whether the patient is finding the therapy helpful and easily managed, or whether the treatment is having a negative impact on his or her quality of life. This 1–2-month review is particularly important to confirm that the low initial PaO₂ was not spurious because the patient was unstable at the time of sampling. Many patients are prescribed oxygen therapy because they are hypoxaemic at discharge from hospital after exacerbation of an underlying respiratory disease, despite an absence of data to support short-term or longer-term benefits of oxygen therapy for the individual patients concerned. A New Zealand study found that 38% of such patients did not fulfil criteria for long-term oxygen therapy at their 2-month review. Reassessment is especially important in this group.

Subsequent review should be undertaken at least annually, or more often, according to the clinical situation. Arterial blood gas measurements may be advisable at review to ensure that oxygen...
flow rate is adequate, given the progressive nature of respiratory disease. Some patients may show a sustained rise in PaO₂ to over 60 mmHg (8 kPa) when breathing air. It has been hypothesised that this represents the reparative effects of supplementary oxygen therapy, and the consensus is that this should not be a rationale for stopping therapy.

Patients having intermittent oxygen therapy should also undergo periodic reassessment, particularly to determine whether they qualify for continuous oxygen therapy. However, this may be unnecessary and undesirably disruptive for those with a limited prognosis.

Quality of life

With the potential restriction of movement imposed by long-term continuous oxygen therapy, it is possible that the treatment may only prolong suffering rather than improve quality of life. However, for patients who qualify for continuous oxygen therapy according to the above criteria, the improvement in quality of life will mostly outweigh the restriction imposed. There is some evidence that women experience more improvement than men in several quality-of-life dimensions. Whether oxygen therapy is worthwhile for a particular individual must be determined by a comprehensive clinical assessment rather than solely, or mainly, by the increase achieved in PaO₂.

Dangers

Pulmonary oxygen toxicity has not been seen at the low rates of flow used for long-term oxygen therapy. Although supplementary oxygen in patients with increased PaCO₂ may theoretically worsen hypercapnia, any increase in PaCO₂ in patients receiving long-term oxygen therapy is usually small and well tolerated. In two large trials of long-term oxygen therapy, hypercapnia was not a problem — probably because patients were in a stable condition. However, serious hypercapnia may occasionally develop, making further investigation and consideration of non-invasive ventilation appropriate. The development of hypercapnia is suggested by an obvious decrease in respiratory rate and depth, as well as the development of somnolence and disorientation. The risk appears greater during acute exacerbations of disease and in patients who are generally more hypoxaemic. Sedatives, narcotics, alcohol and other drugs, which impair the central regulation of breathing, should not be used in unstable patients with hypercapnia who are receiving oxygen therapy.

Methods of oxygen delivery

There are three methods of oxygen delivery for the home: oxygen concentrators, cylinders and liquid oxygen systems.

### Concentrators

Oxygen concentrators are floor-standing, electrically driven devices that entrain room air, extract nitrogen in molecular sieves and deliver oxygen at the outlet. They run off the domestic electricity supply, delivering 92% ± 3% oxygen when operating at a flow rate of ≤ 4 L/min. The percentage falls with increasing flow rate (to 90% ± 3% oxygen at ≥ 5 L/min), depending on the model of concentrator used.

Small, portable, lightweight, battery-driven oxygen concentrators generating up to 5 L of oxygen per minute are now available and are potentially suitable for ambulatory use. The currently available model uses a 12 V battery, and may therefore be run from a car battery.

### Cylinders

Cylinders contain compressed pure oxygen gas and deliver 100% oxygen. Sizes and contents vary (Box 1). Several portable lightweight cylinders are available that allow the patient to leave home for several hours.

### Liquid oxygen systems

At very low temperatures, gaseous oxygen may be converted to a liquid. Liquid oxygen systems conserve space by storing oxygen in liquid form. The oxygen is delivered through coils, where it vaporises. Two tanks are needed: a large storage tank, which is filled by the supplier as required, and a portable unit, filled from the larger tank for ambulatory use.

### Comparison between delivery methods

At the relatively low flow rates employed, there is no significant difference in the quality of oxygen delivered by the three different methods. Advantages and disadvantages of each are compared in Box 2. For most patients receiving continuous or nocturnal oxygen therapy, concentrators are favoured.

### Conservation devices

Conservation devices are designed to maximise the effectiveness of the delivered oxygen. “Demand flow” devices, which are the most common, use an electronic sensor to initiate oxygen flow only during inspiration, ensuring that oxygen is not wasted during expiration. They are useful cost- and time-conserving devices for cylinders and liquid oxygen systems, especially portable units. As these devices switch on flow by sensing negative pressure at the nares via the nasal cannula, they may not be triggered if the patient mouth-breathes (unless the cannula is transferred to the mouth). Many breathless patients become mouth-breathers when they are more distressed.

### Delivery to the patient

All patients should receive careful and detailed instruction on how to operate and obtain optimal benefit from their oxygen equipment. Flow should be set at the lowest rate needed to maintain a resting PaO₂ of 60 mmHg (in practice, most often 2 L/min). It should be increased by 1 L/min during exercise and sleep.

Humidifiers are not needed at these low flow rates, as ambient air entrainment supplies sufficient humidification. Extra soft nasal prongs are recommended for continuous oxygen use, but may become uncomfortable at flow rates over 2–3 L/min and in the

---

**Table: Cylinder size and capacity**

<table>
<thead>
<tr>
<th>Size</th>
<th>Volume (m³)*</th>
<th>Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>G</td>
<td>7.6–8.8</td>
<td>Hospital use only</td>
</tr>
<tr>
<td>E</td>
<td>3.8–5.2</td>
<td>Lasts about 30 h (flow rate, 2 L/min)</td>
</tr>
<tr>
<td>D</td>
<td>1.5</td>
<td>Lasts about 11 h (flow rate, 2 L/min)</td>
</tr>
<tr>
<td>C</td>
<td>0.55</td>
<td>Lasts about 3 h (flow rate, 2 L/min)</td>
</tr>
<tr>
<td>Traveller</td>
<td>0.257–0.682</td>
<td>Depends on size</td>
</tr>
</tbody>
</table>

*1 m³ = 1000 L.
long term. Facemasks may be preferred for at least some of the time. Although simple masks may be adequate for many patients, significant rebreathing, with resulting elevation of \( \text{PaCO}_2 \), may occur at low flow rates in people with type II respiratory failure, and Venturi masks may be necessary in this instance. The appropriate mask should be selected based on arterial blood gas measurements. Both nasal prongs and masks are also acceptable for intermittent oxygen use.

In selected patients needing continuous high-flow oxygen therapy, transtracheal delivery systems may have advantages. These allow substantially lower flow rates, as the tracheal cannula fills the tracheal and upper airway dead space with oxygen during each expiration. However, care of this relatively invasive appliance is demanding, only a few patients find it an attractive alternative, and transtracheal delivery is not currently offered routinely in Australia or New Zealand.

### Authorisation of oxygen therapy

Current guidelines for prescription through state-based financial support programs in Australia and New Zealand usually specify respiratory physicians and cardiologists as authorised prescribers. It could be argued that other groups should be authorised, as long as the guidelines are followed. At present, any registered medical practitioner may order home oxygen therapy if the patient meets the costs. State-based funding for concentrator oxygen therapy is generally provided throughout Australia for patients fulfilling criteria for long-term continuous oxygen therapy. Some states also fund ambulatory oxygen therapy to a limited degree, but access and costs to the patient vary considerably between states. In New Zealand, the situation is analogous: government-funded concentrator oxygen therapy can be authorised only by physicians designated by the local District Health Board, and ambulatory oxygen therapy is only occasionally available.

### Competing interests

None identified.

### References


18 Fletcher EC, Luckett RA, Goodnight-White S, et al. A double-blind trial of nocturnal supplemental oxygen for sleep desaturation in patients with...


(Received 29 Nov 2004, accepted 21 Mar 2005)