



Thoracic Society of Australia and New Zealand

ADULT DOMICILIARY OXYGEN THERAPY

Clinical Practice Guideline

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The purpose of this Clinical Practice Guideline is to provide guidance for prescribers of oxygen therapy in Australia and New Zealand, other health care professionals, government agencies and other providers about domiciliary oxygen therapy.

The development group includes a range of health professionals from allied health, nursing and respiratory medicine backgrounds.

Independent peer review has been provided by the Clinical Care and Resources Subcommittee of the Thoracic Society of Australia and New Zealand (TSANZ).

Dissemination of this Guideline will occur under the auspices of the Clinical Care and Resources Subcommittee and the Executive of the TSANZ.

This document is based on evidence from English language publications up to March 2013 obtained by search of MEDLINE and EMBASE with keywords “domiciliary”, “home”, “long term” and “oxygen”.

Where Levels of evidence are stated these are NHMRC Levels of evidence.

Recommendations are based upon available evidence and the revised GRADE approach to guidelines development was used to categorise *recommendations* into strong, weak or no specific recommendation according to the GRADE levels of evidence (see Appendix 1).

The paper is an update of the Thoracic Society of Australia and New Zealand Adult Domiciliary Oxygen Position Statement published in 2005 and has a currency of five years from the date of publication



KEY POINTS

There is clear evidence for benefit of longterm oxygen therapy in hypoxaemic chronic obstructive pulmonary disease (COPD). GRADE recommendations relate to COPD. Results from studies in COPD have been extrapolated to recommend long term oxygen therapy for all suitable patients with chronic hypoxaemia from any cause

1. Long term continuous oxygen therapy (LTOT), ideally for ≥ 18 hrs/day is indicated to improve longevity when:

- a. Stable daytime PaO₂ is ≤ 55 mmHg (7.3kPA) at rest; or
- b. Stable daytime PaO₂ is 56-59mmHg (7.4-7.8kPa) and there is evidence for hypoxic organ damage (including right heart failure, pulmonary hypertension or polycythaemia)

Flow rate should be set to maintain PaO₂ > 60 mmHg(8kPa) (SpO₂ $> 90\%$) during waking rest.

GRADE: Recommendation: Strong; Evidence: High

There is equivocal evidence for benefit of nocturnal or intermittent oxygen therapy.

2. Nocturnal oxygen therapy may be prescribed for individuals with lung disease who desaturate to SpO₂ $\leq 88\%$ for more than one third of the night, particularly if they suffer sequelae such as pulmonary hypertension or polycythaemia.

GRADE: Recommendation: Strong; Evidence: Low

3. Intermittent oxygen may be prescribed as follows:

- a. In patients commencing LTOT who wish to maximise the number of hours they receive oxygen supplementation during the 24 hour period, through supplementing stationary concentrator use with portable oxygen for physical activities outside the home

GRADE: Recommendation: Strong; Evidence: very low

- b. In occasional cases of chronic lung disease, where patients do not have resting hypoxaemia severe enough to warrant LTOT, and where benefit is demonstrated through a blinded trial of oxygen versus air, assessing outcomes such as exercise capacity or improvement in dyspnoea

GRADE: Recommendation: Weak; Evidence: low

- c. In palliative situations where hypoxaemia co-exists with intractable dyspnoea despite maximal therapy and benefit is demonstrated

GRADE: Recommendation: Strong; Evidence: very low

- d. In maximally treated chronic heart failure with symptomatic central sleep apnoea, in patients intolerant of a continuous positive airway pressure device

GRADE: Recommendation: Strong; Evidence: moderate

- e. During air travel, where a traveller with known lung disease fulfils criteria for requiring LTOT or has a demonstrated fall in SpO₂ to $<85\%$ during an altitude simulation test

GRADE: Recommendation: Strong; Evidence: Low

INTRODUCTION

Supplemental oxygen may benefit patients whose disability is related to a decreased oxygen concentration in arterial blood. This Clinical Practice Guideline summarises current evidence regarding the provision of domiciliary supplemental oxygen therapy, with specific reference to long term continuous oxygen therapy (LTOT), portable oxygen therapy and nocturnal oxygen therapy. It offers guidance with respect to assessment and review of oxygen requirements, the types of devices which may be used to provide such therapy and pitfalls associated with oxygen use.

INDICATIONS

Long term continuous oxygen therapy:

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 (Evidence Level I).^{1,2,3} Long term continuous oxygen therapy (LTOT) aimed at prolonging life should be considered for patients with stable COPD, who have an oxygen partial pressure in arterial blood (PaO_2) consistently less than or equal to 55 mmHg (7.3kPa) at rest when awake and breathing air (Evidence Level 1). At assessment, the patient's condition must be stable and all reversible factors (such as anaemia) should be remediated. As gas exchange may improve substantially on ceasing smoking, assessment should be made at least one month after the patient has stopped smoking.

Polycythaemia (haematocrit > 0.55) and clinical, electrocardiographic or echocardiographic evidence of pulmonary hypertension, as well as episodes of right heart failure, reflect the systemic effects of chronic hypoxaemia and strengthen the case for therapeutic use of oxygen. Stable patients with these complications should be prescribed continuous oxygen if PaO_2 is 56-59 mmHg (7.4 - 7.8kPa) (Evidence Level 1).

In two landmark randomised trials, patients who were prescribed continuous oxygen managed to use it for an average of 18 hours per day.^{1,2} These patients had a greater benefit in mortality compared to those using 15 hours per day or less. Thus, the recommendation is generally that the oxygen be used for as many hours of the day as possible, within reason; ideally a minimum of 18 hours. However, given the potential for LTOT to limit mobility, the benefits of additional hours of LTOT must be weighed against the known beneficial effects of exercise training on quality of life (Evidence Level I).⁴

It is salutary to remember that current recommendations are based on two randomised non-placebo-controlled trials containing fewer than 300 patients. These studies are now over 30 years old. The PaO_2 thresholds for prescription of oxygen therapy were the results of empirical decisions by the trial designers and the studies were performed in COPD populations that would not necessarily be representative of today's COPD patients, many of whom are older and have more co-morbidities than participants in the trials. There have not been any subsequent, high quality randomised controlled studies of long-term oxygen therapy in COPD.

Moreover, to date, there is little evidence to support or oppose the long-term use of oxygen therapy to reduce mortality in patients with chronic respiratory conditions other than COPD. Nonetheless, the results of these early, seminal studies in COPD have been extrapolated to offer long term oxygen therapy to patients



suffering from any illness where chronic hypoxaemia is an important feature: for example, diffuse interstitial lung disease, cystic fibrosis. We believe this recommendation should stand.

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 improving quality of life. There are no placebo-controlled trials examining this question and studies differ in showing either no benefit or a small benefit in health related quality of life in subjects commenced on long term continuous oxygen therapy.^{5,6} **Whether oxygen therapy is worthwhile in the context of a particular individual's management plan must be determined by a comprehensive clinical assessment rather than solely, or mainly, by the increase achieved in PaO₂.**

Nocturnal oxygen therapy

Isolated episodes of hypoxaemia during sleep due to hypoventilation or worsening ventilation-perfusion in patients with obstructive or fibrotic lung diseases should be distinguished from hypoxemia associated with sleep apnoea caused by upper airway obstruction, obesity hypoventilation syndrome or central sleep apnoea. 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 hypoxemia in COPD. Nonetheless, in patients with daytime PaO₂ ≥ 60mmHg (8.0kPa) the clinical consequences of nocturnal hypoxaemia are unknown. A New Zealand study suggested that isolated nocturnal desaturation was very uncommon in the general COPD outpatient population, and patients with nocturnal desaturation had no worse sleep quality, quality of life or daytime somnolence than those without desaturation.⁷ Nocturnal oxygen at 3 L/min over three years was associated in one small study with a smaller rise in pulmonary artery pressure than in a control group receiving supplemental air.⁸ However there was no effect on mortality. A larger two year study of patients with COPD and modest daytime hypoxaemia (PaO₂ 56-59mmHg [7.4-7.8kPa]) who desaturated to SpO₂ < 90% for > 30% of the night found no survival benefit in the group receiving oxygen supplementation and no effect on pulmonary haemodynamics.⁹ Further studies are needed, but the current consensus is that nocturnal oxygen therapy *may* be indicated in patients whose nocturnal arterial oxygen saturation (SpO₂) repeatedly falls below 88% (for > 30% of the night), and who have evidence of hypoxia-related sequelae, provided any contributing factors have been addressed optimally (for example: obstructive sleep apnoea, congestive cardiac failure, airways disease). It should be noted that both the duration and the severity of hypoxaemia upon which prescription is recommended are based on expert opinion.

Intermittent Oxygen therapy

a) Ambulatory

Ambulatory oxygen therapy may be used as part of continuous oxygen therapy, in which case its benefits are those of LTOT. Patients in the "continuous" arm of the Nocturnal Oxygen Therapy Trial, whose survival benefit was greatest, used both stationary and ambulatory systems in order to enable an average usage of 18 hour per day¹. In the absence of need for LTOT, there is no direct evidence that treatment of exercise-induced hypoxaemia retards long-term pulmonary hypertension or prolongs life, and thus its role has in the past been aimed at relieving breathlessness. Despite the observation of small, acute benefits of oxygen therapy during laboratory-based exercise tests in COPD¹⁰ (Evidence Level I), the subject of ambulatory oxygen use in patients who do not fulfil criteria for LTOT remains controversial. A 12-week randomised controlled trial (RCT) compared ambulatory intranasal oxygen with ambulatory air at 6L/minute via portable cylinders in patients with stable COPD and no resting hypoxaemia.¹¹ Although there were within group improvements in dyspnoea and depression scores, these were small and not different between groups. Importantly, there were no differences in the responses of patients who did and did not desaturate on exertion. These results suggest a substantial placebo effect from the administration of intranasal gas, possibly relating to the wearing of nasal cannulae,¹² and are consistent with those of Nonoyama et al¹³, who found an absence of effect on

breathlessness, quality of life or distance walked in a timed walking test with the use of ambulatory oxygen in a majority of COPD subjects who desaturated with exertion (n=27). These recent studies lead us to suggest that supplemental oxygen should not generally be prescribed for patients with COPD who are normoxaemic or only mildly hypoxaemic at rest (Evidence Level II). However as two individuals in the study by Nonoyama et al achieved clinically significant reductions in dyspnoea with the use of supplemental oxygen, occasional, blinded, so-called “n-of-1 trials” may be of use in some individuals with severe breathlessness.¹³

For patients with other lung diseases, for example, interstitial lung diseases, which are often associated with profound oxygen desaturation during exertion, no large RCTs are available and at this time, in the absence of evidence to the contrary, it would appear reasonable to continue previous consensus-based recommendations. These are that: benefit should be established by comparing exercise endurance, oxygen saturation and degree of dyspnoea when breathing oxygen and when breathing air during a walking-based test (that is, 6-minute walk test, incremental or shuttle endurance walk test or treadmill test). Given the strong placebo effect of intranasal gas11 we recommend blinding of gas type during these assessments (Evidence Level II).

b) Other intermittent

Other situations where intermittent oxygen therapy may be indicated include;

- “Palliative” oxygen

Supplemental oxygen may provide symptomatic relief for patients with intractable dyspnoea and significant hypoxaemia ($\text{PaO}_2 < 55\text{mmHg}$ or $\text{SpO}_2 \leq 88\%$) due to terminal illnesses, including late-stage lung disease. However, it is important to bear in mind that hypoxaemia may not necessarily be associated with dyspnoea and relief of hypoxaemia with oxygen therapy may not necessarily relieve dyspnoea. In such cases, if a trial of oxygen therapy is not beneficial, alternate therapies may be appropriate. In the setting of refractory dyspnoea for so called “palliative” use in a population of patients *without* resting hypoxaemia and with a variety of long term cardiopulmonary conditions (predominantly COPD) or lung malignancy, oxygen at 2L/minute delivered via concentrator over 15 hours for seven days did *not* confer a greater benefit than intranasal air at the same flow rate (Evidence Level II).¹⁴

- Oxygen in chronic heart failure

There are no trials of long term oxygen therapy examining functional status or survival in severely hypoxaemic patients with chronic heart failure. Indeed, small studies of patients with chronic heart failure suggest that daytime hypoxaemia is uncommon in this group¹⁵. There is no evidence that use of long-term oxygen therapy reduces breathlessness or clinical events such as hospital admission or mortality in heart failure. Some patients with chronic heart failure have central sleep apnoea. Continuous positive airways pressure (CPAP) therapy may be indicated for such patients after maximisation of their medical therapy. However, for those intolerant of CPAP, there is evidence that oxygen therapy provides small improvements in LVEF and in severity of sleep disordered breathing (Evidence Level II).¹⁶

- Oxygen during air travel

Commercial passenger aircraft operate at cabin pressures similar to ambient pressures experienced at up to 2,500m above sea level. This is analogous to breathing 15% oxygen at sea level. At this “altitude”, the PaO_2 for healthy people falls to around 53–64 mmHg (7.1-8.5kPa), with corresponding oxygen saturations of 85%–91%.¹⁷ As a general rule, supplemental oxygen is unlikely to be required if the resting oxygen saturation is $\geq 95\%$, but is recommended for patients who qualify for continuous oxygen therapy at home ($\text{SpO}_2 \leq 88\%$). In those with a sea-level oxygen saturation between these values, PaO_2 at altitude may be predicted from baseline data. However there is wide variance in these predictions,¹⁷ and laboratory-based altitude simulation provides more accurate estimates of altitude oxygenation.¹⁸ It is recommended that PaO_2 should be maintained above 50mmHg (6.6 kPa) or SpO_2 above 85% during airflight (Evidence Level :consensus).¹⁷



- Oxygen in Pulmonary Rehabilitation

In patients with chronic lung diseases enrolled in pulmonary rehabilitation supplemental oxygen may be used with the aim of enhancing training benefits where symptoms or profound desaturation limit training (Evidence level:consensus), Although there is no evidence to support this recommendation, health personnel supervising exercise training are likely to instruct patients who desaturate significantly to take frequent rests for fear of adverse consequences. In such situations, the clinical benefits of training are likely to be less because of the lower dose of exercise compared to that achieved by avoiding desaturation with supplemental oxygen. Theoretical reasons to support the use of supplemental oxygen during training include the potential for amelioration of exercise-induced elevations in pulmonary arterial pressure^{19,20} and the potential for reductions in minute ventilation and dynamic hyperinflation.²¹

- Patients with acute asthma living in isolated areas or prone to sudden life-threatening episodes may benefit from having oxygen available for use while they are awaiting medical attention (Evidence Level: consensus)

Cognitive Function, Hypoxaemia and Driving

Although cognitive impairment is common in patients with hypoxaemic COPD there is limited evidence for benefit of long term oxygen therapy on cognition.²² Previous guidelines on fitness to drive in Australia recommended patients on long term oxygen therapy should use supplemental oxygen whilst driving a motor vehicle²³. However there is neither evidence for cognitive improvement,²⁴⁻²⁵ nor evidence for improvement in simulated driving performance with acute oxygen therapy in this patient group.²⁶ Thus, there is currently no recommendation for hypoxaemic patients to use portable oxygen therapy whilst driving in Australia and/or New Zealand²⁷

Contraindications to Oxygen Therapy

Oxygen therapy is not indicated for patients:

- With severe airflow limitation whose main complaint is dyspnoea, but who maintain a PaO₂ greater than 60 mmHg (8 kPa) and who show no secondary effects of chronic hypoxia.
- Who continue to smoke cigarettes, because of the increased fire risk and the probability that the poorer prognosis conferred by smoking will offset treatment benefit.²⁸⁻³⁰
- Who have not received adequate therapy for their underlying medical condition(s) responsible for causing hypoxaemia.
- Who are not sufficiently motivated to undertake the discipline required in using oxygen therapy for the prescribed number of hours per day.

Dangers

Pulmonary oxygen toxicity has not been seen at the low rates of flow used for long-term oxygen therapy. Although supplemental oxygen in patients with increased arterial PaCO₂ may theoretically worsen hypercapnia, in LTOT, any increase in arterial PaCO₂ is usually small and well tolerated. Hypercapnia was not a problem in two large trials of long-term oxygen therapy, probably because patients were in a stable condition.^{1,2} 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. Risk appears greater during acute exacerbations of disease and in those 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 receiving oxygen therapy. Where hypercapnia is suspected, its presence needs to be assessed by arterial blood gas analysis.

ASSESSMENT OF LTOT REQUIREMENTS

Arterial Blood Gas (ABG) analysis and Pulse Oximetry

Due to the nature of the oxyhaemoglobin dissociation curve, and the inherent variability and bias of pulse oximeters, spot checks of SpO₂ cannot provide a suitable estimate of PaO₂ to assess ongoing long-term oxygen needs. Room air arterial blood gas (ABG) analysis is required to determine eligibility for long term oxygen therapy. Oximetry or repeat ABG on supplemental oxygen is required to document adequate oxygen delivery (to achieve a SpO₂ > 90% or PaO₂ > 60mmHg [8 kPa]). ABGs are required where hypercapnia is a possibility. Changes in respiratory patterns during arterial sampling can significantly alter measured blood gas values. Sampling should therefore be undertaken by trained operators experienced in the technique with due care in reducing patient apprehension and minimising pain during sampling. The use of topical anaesthetics does not reduce pain associated with radial artery sampling.^{31,32} The use of smaller needles (25 gauge) without anaesthetic is recommended to minimise pain and associated trauma, although patient preference should be considered.

ABG measurement is arguably one of the most reliable of all pathology measurements hence it is essential that care is taken in the pre-analytic phase of the analysis. Sampling of blood in the non-steady-state condition will result in blood gas values that do not reflect the true, resting values and thereby potentially lead to incorrect diagnostic categorisation and clinical decision making. Allowing adequate time for changes in inspired gas concentrations is important, and data indicate that even in severe airways disease a five-minute washout period is adequate.³³ Adequate rest time to allow recovery from exertion prior to blood sampling is also mandatory to allow return to baseline conditions. It would seem reasonable that 5-10 minutes at rest, breathing room air is suitable to achieve steady-state conditions.

Pulse oximetry can be used to provide relatively sensitive monitoring of blood oxygen levels over extended timeframes and so can be used to monitor nocturnal hypoxaemia. Pulse oximeters with onboard memory capable of recording at least eight hours of data are suitable for this purpose with next-day data analysis providing estimates of average oxygenation overnight. These data provide the basis for nocturnal oxygen prescription as detailed above. Such analysis can also provide indicators of desaturation events associated with sleep-related breathing disorders such as sleep apnoea. In these cases further investigation and management of a possible sleep disorder is merited prior to consideration of nocturnal oxygen therapy.³⁴

Reassessment/Review of Oxygen Requirements

There is no evidence supporting the practice of prescribing oxygen at hospital discharge, a common practice which has been described as “short term” oxygen therapy. Nonetheless, over 50% of referrals in a New Zealand oxygen service originated following an inpatient hospital stay.³⁵ If oxygen is prescribed in this situation, it should be discussed prospectively with the patient as a short term treatment during exacerbation recovery that will most likely only be needed for a month or two. The severity of hypoxaemia should be determined 4-8 weeks following discharge, given that up to 38% of patients found to be eligible for long term oxygen therapy at hospital discharge no longer exhibited PaO₂ eligibility for continued oxygen therapy two months thereafter.³⁵ Repeat ABG analysis is important to confirm that the low initial PaO₂ used for initiation of oxygen therapy was not a spurious finding or related to unstable disease status. When discontinuing short term oxygen, a positive emphasis on the patient’s clinical recovery (no longer requiring oxygen as evidenced by ABGs), rather than negative messages about “not qualifying” for oxygen therapy, may lessen anxiety. If ABGs show persistent chronic hypoxaemia, likely benefit versus inconvenience of LTOT can be discussed with the patient. The decision regarding continuation of therapy should be based upon multiple factors including patient acceptance and usage, impact on quality of life as well as whether smoking cessation has been successful.



Subsequent review should be undertaken at least annually, or more often according to clinical need. Given the progressive nature of respiratory disease, repeat ABG assessment and/or overnight oximetry may be advisable at review to ensure that oxygen flow rate is adequate. Improvements in PaO₂ to > 60 mmHg (8.0 kPa) after prolonged periods on LTOT may be seen and have been postulated as representing the reparative effects of oxygen therapy.³⁶ The consensus is that this should not be a rationale for ceasing therapy. A patient having intermittent oxygen therapy should also undergo periodic reassessment, particularly to determine whether they qualify for LTOT.

Patient compliance and quality of service delivery

Overall benefits from oxygen therapy likely depend on appropriate patient selection, smoking cessation and achieving adequate oxygenation with each individual patient's prescribed flow rate. Surveys have shown both patient compliance rates (prescribed hours) and adherence to guidelines vary widely within and between countries³⁷⁻³⁹ with some evidence that an hour of patient education when starting domiciliary oxygen therapy is associated with significantly higher compliance rates.⁴⁰ Oxygen users' beliefs about oxygen were found to strongly influence their pattern of oxygen use, suggesting benefit from discussing their beliefs and concerns when considering a prescription.⁴¹

Monitoring of therapy and compliance with quality indicators and national registries has been shown to improve the quality of care of patients receiving oxygen therapy.⁴² A national oxygen register with data contributed from all domiciliary oxygen therapy services in Australasia would enable strategic planning, benchmarking of costs and outcomes and support the more rational and equitable use of this therapy. Useful information for patients commencing on home oxygen is provided at <http://www.lungfoundation.com.au/lung-information/patient-resources/educational-resources/getting-started-on-home-oxygen/>⁴³

SOURCES OF OXYGEN FOR DOMICILIARY USE

There are three systems for providing oxygen in the home: stationary oxygen concentrators, cylinders, and portable oxygen concentrators (POCs). The conversion of oxygen gas into a liquid is possible; however, liquid oxygen systems have not been adopted for domiciliary use in Australia or New Zealand.

Stationary concentrators

These floor-standing, electrically driven devices entrain room air, extract nitrogen in molecular sieves and deliver a continuous flow of oxygen at the outlet. They are powered by the domestic electricity supply and generally provide an oxygen concentration of 90 to 95% depending on the model.⁴⁴ The percentage of oxygen falls with increasing flow rate and is usually less than 90% with flow rates > 5L/min.⁴⁴ These devices are easy to operate and require minimal maintenance by the patient. Patients may require a back-up oxygen cylinder for use in case of power failure or concentrator failure. To enable movement within the home, lengths of 9 meter extension tubing are used in line to connect the patient to their stationary concentrator. However, if not appropriately secured, the tubing can pose a falls hazard. Further, tubing longer than 30 metres, in combination with flow rates > 3L/min, will lead to a significant fall in the concentration of delivered oxygen and is not recommended.⁴⁵

Cylinders

Cylinders contain compressed pure oxygen in the gaseous state and always deliver 100% oxygen. Patients



must follow procedures for the safe storage of compressed gas cylinders in the home and avoid contact with materials such as oil and grease in order to avoid fire hazard and personal injury. Small portable cylinders (Size C or Traveller, Box 1) are available that allow the patient to leave home for up to several hours. The oxygen is then delivered to the patient either via a flow meter attached to the cylinder or via an oxygen conservation device (OCD). A major drawback of portable oxygen cylinders is the limited amount of time patients are able to leave the home and the cost of refills and delivery.

Patients are usually supplied with either a pull-along trolley or shoulder bag to transport their portable cylinder. The additional weight if carried on the shoulder can offset the benefits of the oxygen so the trolley option (or small suitcase on wheels or shopper trolley) is frequently recommended. For some patients with COPD and very low functional exercise capacity, a wheeled walker (rollator) with cylinder attached may be helpful.⁴⁶ Compressed oxygen cylinders should be stored and carried securely in the home and in vehicles to prevent injury from accidental cylinder movement.

Flow meters and oxygen conservation devices

Flow meters are simple to use but are only able to deliver a continuous flow of oxygen. Oxygen conservation devices (OCDs) deliver oxygen predominantly during inspiration and therefore avoid wastage. ‘Demand flow’ devices, which are the most common, deliver a pre-set volume or bolus of oxygen in early inspiration.⁴⁷ As they are triggered by the patient’s inspiratory effort (sensed as negative pressure at the nares) they may not be triggered if the patient mouth-breathes or if the nares are very large (unless the cannulae are transferred to the mouth). Several models of demand flow OCDs are available and these can be either electronically (generally more efficient) or pneumatically driven.

Some patients find demand flow OCDs difficult to operate and to fit correctly on a cylinder. Training and education are needed to use these devices and to ascertain whether the device is working correctly.

Another type of OCD consists of a nasal cannula set with an integrated pendant shaped reservoir that fills with oxygen during exhalation and delivers a bolus of oxygen during the subsequent inspiration (an older but less preferred “moustache”-style is also available). The efficient timing of oxygen delivery enables the patient to use these devices at a lower flow rate than via conventional nasal cannulae while maintaining equivalent oxygen saturations. This durable device can be used with any continuous flow oxygen source, is simple to use, relatively inexpensive and, unlike most other OCDs, does not require batteries or any controls.

Portable oxygen concentrators

Small, lightweight, portable oxygen concentrators (POCs) are available with multiple power options (household electrical supply, car or rechargeable battery) making them potentially suitable for ambulatory use. These devices give patients freedom to travel and some models have been approved for use by commercial airlines. Two types are available, one that is only capable of delivering oxygen in a pulse-dose mode (generally smaller and lighter), which conserves battery life, and another that gives the option of either pulse-mode or continuous-flow.

When oxygen is delivered using a pulse-dose mode concentrator, the POCs offer a range of pulse settings, generally up to a maximum of six. These settings are not identical to oxygen flow rates in litres per minute. Therefore it is important to use pulse oximetry to titrate oxygen levels when a patient changes from using oxygen supplied by a cylinder to a POC.

The performance specifications of the various makes of POCs differ considerably.⁴⁸ With some POCs, the oxygen concentration of the gas delivered with each breath is relatively constant despite increases in respiratory rate. This is because the device delivers a fixed constant pulse volume of oxygen (per setting) independent of respiratory rate (i.e. fixed pulse volume). Other types of POCs work on the principle of delivering a constant volume of oxygen per minute (fixed oxygen minute volume). With the latter type, if tidal volume remains constant, then the actual concentration of oxygen per breath will decrease when respiratory rate increases, for example during exercise. For patients with high oxygen needs, POCs may not be suitable



for use when exercising and such patients may require a portable oxygen cylinder in order to achieve a sufficient concentration of inspired oxygen at these times.

Comparison between oxygen systems

Box 2 outlines the advantages and disadvantages of the different systems for providing domiciliary oxygen therapy.

Titration of oxygen supply

Oxygen saturation levels will be lower when using a stationary oxygen concentrator as compared to a continuous flow of oxygen (for example: piped hospital oxygen or cylinder).⁴⁹ Further, oxygen titration is required when (i) changing from continuous flow to using a demand flow OCD; (ii) changing between different models of OCDs, and (iii) changing from a cylinder or stationary oxygen concentrator to a POC. Therefore it is important to use pulse oximetry to titrate oxygen levels for each patient using the device(s) they will be using in the domiciliary setting.

Patients require a simple prescription stating what flow rate of oxygen or setting on an OCD or POC should be used for different situations (for example: rest, activity/exercise, overnight) and it is important that all healthcare professionals reinforce the same prescription. Ideally all patients should be provided with a written prescription which should be available for viewing by all health care providers.

Delivery to the patient

All patients should receive careful and detailed instruction on how to operate and obtain optimal benefit from their oxygen equipment. Oxygen supply should be set at the lowest setting needed to maintain a resting $\text{PaO}_2 \geq 60\text{mmHg}$ (8 kPa) or $\text{SpO}_2 > 90\%$ (in practice, most often 1-2 L/min when using continuous flow). Traditionally it has been recommended that patients increase their flow rate empirically by 1L/min nocturnally, during exercise (including everyday physical activities such as showering) and during air travel. In the absence of documented evidence for any adverse effects of this approach, we endorse this recommendation. Nonetheless, we acknowledge evidence from a cross-sectional study in New Zealand suggesting that, in practice, few LTOT patients desaturate significantly overnight even if they have not increased their oxygen flow rate from that recommended during daytime rest.⁵⁰

Humidifiers are not needed at these low flow rates, as the entrainment of ambient air supplies sufficient humidification. Extra soft nasal prongs are recommended for continuous oxygen use, but may become uncomfortable as they deteriorate with prolonged use and at high flow rates (> 3L/min). Anecdotally, some patients find benefit from using nasal spray or drops that contain Vitamin E or denatured sesame oil to counteract the drying of the nasal mucosa and the associated bleeding that may occasionally occur. 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 PaCO_2 , may occur at low flow rates in people with type II respiratory failure, and air-entrainment masks that use the Venturi principle may be necessary in this instance. The appropriate mask should be selected based on arterial blood gas measurements. Both nasal cannulae and masks are acceptable for intermittent oxygen use.

Box 1 Cylinder size and capacity

Size	Volume (m ³)*	Use
G	7.6-8.8	Hospital use only
E	3.8-5.2	Lasts about 30 h (flow rate, 2L/min)
D	1.5	Lasts about 11 h (flow rate, 2L/min)
C	0.55	Lasts about 3 h (flow rate, 2L/min)
Traveller	0.257-0.682	Depends on size

*1m³ = 1000L



Box 2 Advantages and disadvantages of available home oxygen delivery systems*

Advantages	Disadvantages
Stationary concentrators	
<ul style="list-style-type: none"> Low cost to supply Convenient for home use Wide availability Safe Easy to operate 	<ul style="list-style-type: none"> Electricity required (with associated costs for users); back-up cylinders may be required Purity of oxygen declines at higher flow rates Risk of mechanical failure Regular (1-2/yr) maintenance required Produces noise, heat and vibration Oxygen tubing can present a falls hazard
Cylinders	
<ul style="list-style-type: none"> Wide availability High oxygen purity Electricity not required Reliable No background noise 	<ul style="list-style-type: none"> High cost and frequent deliveries required Less convenient compared to stationary concentrators for home use Heavy weight Small capacity Regular changes of cylinders require physical effort and, when used in combination with demand flow OCDs, can be complicated to operate
Portable oxygen concentrators	
<ul style="list-style-type: none"> Powered by standard household electrical supply, car battery or rechargeable battery Small and aesthetically pleasing Enable patient to travel Approved by some airlines Easy to operate 	<ul style="list-style-type: none"> High cost Performance characteristics of available POCs are very variable Not all are capable of producing continuous flow as required to entrain oxygen into CPAP or Non-invasive ventilation (NIV) equipment May not be suitable for patients with high oxygen needs

*Adapted from Kampelmacher et al⁵¹(1994) & Dunne et al⁴⁷ (2009)

Authorisation to prescribe oxygen therapy

Applications to government-funded domiciliary oxygen therapy services in Australia and New Zealand generally require a prescription from a respiratory physician, general physician, cardiologist or paediatrician, with additional approval from a specified respiratory physician required in some jurisdictions. Oncologists, geriatricians, palliative care specialists and nurse practitioners (respiratory) are listed as approved prescribers in some states and in New Zealand. Domiciliary oxygen can be ordered by any registered medical officer if the patient meets the cost or is resident in an aged care facility. Oxygen concentrators provide the stationary source of oxygen in all jurisdictions with back up cylinders provided for use in power outages (eligibility criteria vary widely between jurisdictions) in most states. The level of subsidised ambulatory oxygen provided in addition to a concentrator is extremely variable across services. We recommend that adequate supplies of ambulatory oxygen should be made available to ensure that those patients fulfilling LTOT criteria are enabled to live active lives while maintaining adequate oxygenation.⁵²



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APPENDIX 1

RECOMMENDATIONS AND EVIDENCE BASED UPON GRADE ⁵³

The level of evidence provided by GRADE is:

- High=Further research is very unlikely to change our confidence in the estimate of effect.
- Moderate=Further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate.
- Low=Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate.
- Very low = Any estimate of effect is very uncertain.

The implications of a strong recommendation are:

- For patients—most people in your situation would want the recommended course of action and only a small proportion would not; request discussion if the intervention is not offered.
- For clinicians—most patients should receive the recommended course of action.
- For policy makers—the recommendation can be adopted as a policy in most situations.

The implications of a weak recommendation are:

- For patients—most people in your situation would want the recommended course of action, but many would not
- For clinicians—you should recognise that different choices will be appropriate for different patients and that you must help each patient to arrive at a management decision consistent with her or his values and preferences
- For policy makers—policy making will require substantial debate



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