BTS Guideline for Home Oxygen use in adults
Key Questions - PICO
10 December 2012

Evidence base for Home Oxygen therapy in COPD, non-COPD respiratory disease and non-respiratory disease

1. SBOT

1.1 Use of SBOT in normoxic patients

1.1.1 Does SBOT improve symptoms of breathlessness, quality of life, exercise capacity, recovery time or reduce health care utilisation in patients with normal resting oxygen saturations quicker and better than non-pharmacological measures (including fans, CBT and breathing air)?

1.2 Use of SBOT in hypoxic patients, above LTOT threshold

1.2.1 Does SBOT improve symptoms of breathlessness, quality of life, exercise capacity, or reduce health care utilisation in patients whose resting oxygen saturations are permanently or intermittently below normal, but above the threshold for LTOT quicker and better than non-pharmacological measures (including breathing air)?

1.3 Use of SBOT in Sickle cell disease

1.3.1 Does use of SBOT in patients with sickle cell disease reduce the severity, duration and healthcare utilisation associated with sickle cell attack/crisis quicker and better than non-pharmacological measures (including breathing air)?

1.4 Use of SBOT in Cluster headache

1.4.1 Does use of SBOT in patients with cluster headache syndrome reduce the severity, duration and healthcare utilisation associated with cluster headache quicker and better than non-pharmacological measures (including breathing air)?

2. LTOT

2.1 What benefits are there for using LTOT in COPD and non-COPD patients?

2.1.1 Does LTOT lead to improved life expectancy, symptoms, quality of life, pulmonary hypertension, and healthcare utilisation in COPD patients compared with similar patients who have never received LTOT?

2.1.2 Does LTOT lead to improved life expectancy, symptoms, quality of life, pulmonary hypertension and healthcare utilisation in non-COPD patients (pulmonary hypertension, pulmonary vascular disease, cystic fibrosis, bronchiectasis, interstitial lung disease, chest wall disease, neuromuscular disease, obesity hypoventilation, cardiac disease including congestive cardiac failure and adult congenital heart disease) compared to no LTOT?

2.2 What evidence is there for using current arterial blood gas parameters for prescribing LTOT?

2.2.1 Does LTOT lead to improved life expectancy, symptoms, quality of life, pulmonary hypertension and healthcare utilisation in patients with an arterial PaO2 less than 7.3kPa compared with no LTOT?

2.2.2 Does LTOT lead to improved life expectancy, symptoms, quality of life, pulmonary hypertension and healthcare utilisation in patients with an arterial PaO2 greater than or equal to 7.3kPa compared to no LTOT?

2.2.3 Does LTOT lead to improved life expectancy, symptoms, quality of life, and healthcare utilisation in patients with an arterial PaO2 7.3 – 8kPa who have evidence of pulmonary hypertension, polycythaemia, or nocturnal desaturation compared with no LTOT?

2.2.4 Does LTOT lead to improved life expectancy, symptoms, quality of life, and healthcare utilisation in patients with an arterial PaO2 7.3 – 8kPa who have no evidence of pulmonary hypertension, polycythaemia, or nocturnal desaturation compared with no LTOT?

2.3 What evidence is there for current prescribing of LTOT for 16 hours per day?

2.3.1 Does use of LTOT for over and above 16 hours per day lead to improved life expectancy, symptoms, quality of life, pulmonary hypertension and reduce healthcare utilisation compared to LTOT used for less than 16 hours per day.

2.4 What evidence of benefit is there for prescribing LTOT to non-smokers?

2.4.1 Does LTOT lead to improved life expectancy symptoms, quality of life and healthcare utilisation in patients who are non-smokers (self-reported or proven by exhaled CO monitoring or urinary cotinine) over similar patients who have never received LTOT?

2.5 What evidence of benefit is there for prescribing LTOT to smokers?

2.5.1 Does LTOT lead to improved life expectancy symptoms, quality of life and healthcare utilisation in smokers with COPD, non-COPD respiratory disease and non-respiratory disease over similar patients who have never received LTOT?

3. AOT

3.1 Use of ambulatory oxygen during exercise in non-LTOT patients

3.1.1 Does use of ambulatory oxygen by patients whose resting oxygen saturations are above 92%, and become breathless and/or desaturate on exercise (to less than 90% or who experience a 4% fall in oxygen saturations from baseline) lead to reduced breathlessness, increased exercise capacity, reduced recovery time from exercise induced breathlessness and improved quality of life (including ability to perform activities of daily living) compared to breathing air (including fans and compressed air) in similar patients?

3.1.2 Does the provision of ambulatory oxygen to patients who desaturate on exercise to less than 90% or who experience a 4% fall in oxygen saturations from baseline on exercise reduce morbidity, mortality or healthcare utilisation compared to breathing air on exercise?

3.2 Use of ambulatory oxygen during exercise in LTOT patients

3.2.1 Does use of ambulatory oxygen in patients who are receiving LTOT/ fulfil criteria for LTOT lead to reduced breathlessness, increased exercise capacity, reduced recovery time from exercise induced breathlessness, improved quality of life (including ability to perform activities of daily living) and improved compliance with LTOT therapy compared to breathing air (including fans and compressed air) in similar patients?

4. Palliative oxygen therapy

4.1 When is the use of oxygen beneficial in providing palliative care to patients?

4.1.1 Does oxygen therapy help improve symptoms and quality of life in patients with terminal illness/cancer or end-stage cardio-respiratory disease who are breathless but normoxic at rest, compared to non-pharmacological treatments?

4.1.2 Does oxygen therapy help improve symptoms and quality of life and health care utilisation in patients with terminal illness/ cancer or end-stage cardio-respiratory disease who are breathless and hypoxic at rest, compared to non-pharmacological treatments?

5. Nocturnal oxygen therapy

5.1 Is nocturnal oxygen therapy beneficial in patients with nocturnal desaturation?

5.1.1 Does treatment with overnight oxygen compared to no overnight oxygen lead to improved health status, health care utilisation, mortality, pulmonary hypertension and sleep quality in normoxic COPD patients with nocturnal desaturation?

5.1.2. Does treatment with nocturnal oxygen therapy compared to no nocturnal oxygen therapy lead to improved health status, health care utilisation, mortality, pulmonary hypertension and sleep quality in normoxic patients with nocturnal desaturation and other respiratory diseases (Interstitial lung disease, bronchiectasis, pulmonary hypertension, pulmonary vascular disease, cystic fibrosis, chest wall disease, neuromuscular disease, obesity hypoventilation, cardiac disease including congestive cardiac failure and adult congenital heart disease)?

Referral, assessment, follow-up and withdrawal of home oxygen in non-palliative care patients

6. Referral

6.1 Use of information at the time of referral for home oxygen assessment

6.1.1 Does provision of written information or a telephone consultation to patients at the time of referral for home oxygen assessment decrease the number of patients who decline or fail to attend HOS assessment compared with patients not given written information or a telephone conversation?

6.2 Timing of oxygen assessment in relation to exacerbation of underlying cardio-respiratory disease

6.2.1 Does performing an oxygen assessment in patients less than 5 weeks following an acute exacerbation of cardiorespiratory disease result in unnecessary oxygen prescriptions compared to assessment performed at an interval of greater than 5 weeks?

6.3 Use of oximetry as a screening tool for home oxygen referral

6.3.1 Does the referral of patients for home oxygen assessment with a resting oxygen saturation of less than 92% rather than patients with a resting oxygen saturation of greater than or equal to 92% result in patients more patients being eligible for home oxygen therapy?

7. Assessment

7.1 Use of oximetry, arterial blood gases and capillary blood gases in assessment for home oxygen

7.1.1 Does measurement of pulse oximetry lead to the same number of patients being prescribed home oxygen therapy by clinicians, the same patient experience satisfaction and same adherence to treatment as measurement of arterial blood gases?

7.1.2 Does measurement of capillary blood gases lead to the same number of patients being prescribed long term oxygen therapy by clinicians, the same patient experience satisfaction and same adherence to treatment as measurement of arterial blood gases?

7.1.3 Does a single measure of oxygenation lead to the same number of patients being prescribed long term oxygen therapy by clinicians as two repeated measures at an interval?

7.2 Patients who become hypercapnic during home oxygen assessment

7.2.1 Do patients who develop an increase in PaCO2 of >1kPa during an oxygen assessment have an increased morbidity and mortality compared with patients who do not?

7.2.2 Do patients suitable for LTOT who demonstrate an increase in PaCO2 by >1kPa during an oxygen assessment benefit (reduced morbidity, mortality and improved quality of life) from nocturnal NIV?

7.2.3 Do patients who develop respiratory acidosis during an oxygen assessment have increased morbidity and mortality compared with patients who do not?

7.2.4 Do patients suitable for LTOT who develop respiratory acidosis during an oxygen assessment benefit (reduced morbidity, mortality and improved quality of life)from nocturnal NIV?

8. Follow-up

8.1 Reassessment of home oxygen patients

8.1.1 Does follow-up for home oxygen patients including home visits, clinic visits or telephone calls lead to improved compliance and improved clinical outcomes with home oxygen prescription compared with no follow-up?

8.2 Patients discharged from hospital

8.2.1 Does the discharge of patients from hospital with resting oxygen saturations of less than 92% with home oxygen result in less morbidity, mortality and healthcare utilisation than discharge without home oxygen?

9. Withdrawal of home oxygen

9.1 Does withdrawal of home oxygen therapy from patients whose oxygen levels have improved to above treatment threshold on follow up result in increased morbidity, mortality and healthcare utilisation within the following 12 months compared with not withdrawing oxygen therapy?

9.2 Does the provision of patient education or non-pharmacological strategies result in reduced anxiety and symptoms in patients from whom home oxygen is withdrawn compared to no education or other strategies?

Equipment used for Home oxygen therapy

10. What equipment should be used for homes oxygen assessment?

10.1 Does assessing patients for home oxygen on the same equipment and flows that they are prescribed lead to increased adherence, fewer subsequent enquiries with regards to equipment use, fewer equipment changes and reduced admissions than assessment on standardised equipment ?

10.2 Does humidification of home oxygen in patients with difficult secretions (tracheostomy patients, cystic fibrosis, bronchiectasis) who are receiving home oxygen at a flow rate of 4l/min or more lead to fewer chest infections or exacerbations than provision of oxygen which is not humidified?

10.3 Do ambulatory devices used by patients at rest and on exertion lead to the same reduction in mortality and hospital admissions and improvement in symptoms compared to LTOT?

Safety and Home oxygen therapy

11. Smoking and home oxygen

11.1 Do patients who smoke at home who are provided with home oxygen therapy, have an increased risk of personal injury and damage to property than smoking patients who are not prescribed home oxygen?

11.2 Does monitoring of smoking status by patient self-report, urinary cotinine or CO monitors improve safety in patients who smoke at home who are provided with home oxygen therapy compared with no monitoring of smoking status?

12. Hypercapnia and home oxygen

12.1 Does provision of LTOT to patients with known hypercapnia cause increased morbidity, mortality and healthcare utilisation compared to hypercapnic patients not prescribed HO?

12.2 Does the provision of HO other than LTOT to patients with known hypercapnia cause increased morbidity, mortality and healthcare utilisation when they exacerbate compared to hypercapnic patients not prescribed HO?

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