Oxygen Therapy: When, What and Why

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Oxygen: A medicine to treat hypoxia
What I will cover

- Indications for oxygen therapy
- COPD as a guide
- LTOT, ambulatory & SBOT
- Oxygen and smoking
- Palliative care setting
- When to be worried
What I won't cover

- Children
- Travel
- Neuromuscular weakness
- Emergency oxygen
Respiratory failure

Type 1 RF:
Low PaO$_2$ (<8.0kPa, 60mmHg)
Normal PaCO$_2$

Type 2 RF:
Low PaO$_2$ (<8.0kPa, 60mmHg)
High PaCO$_2$ (>6.5kPa, 50mmHg)
Causes of respiratory failure

High altitude

V/Q mismatch:
- COPD
- Asthma
- PH
- ILD
- CF
- PE
- CCF

Cardiac or intrapulmonary shunts

V/Q mismatch:
- COPD
- Asthma
- PH
- ILD
- CF
- PE
- CCF

Cardiac or intrapulmonary shunts
Signs and Symptoms of Respiratory Failure

- Tachypnoea
- Dyspnoea
- Tachycardia
- Tachyarrhythmias
- Polycythaemia

Restlessness, disorientation, lethargy

Signs of cor pulmonale

(poor prognosis; 5 year survival <50%)
Who to assess

- Very severe airflow obstruction (FEV1 <30% predicted)
- Cyanosis (SaO2 ≤92% on air)
- Polycythaemia
- Peripheral oedema
- Signs of cor pulmonale
Some history

Oxygen discovered by JB Priestley in 1773

Usual composition of room air
Nitrogen 78%
Oxygen 21% (FiO2= fraction inspired oxygen)
Other 1%
Oxygen therapy: administration of oxygen at concentrations higher than those noted in room air with aim of:

- Reducing hypoxaemia
- Improving survival
- Decreasing ventilatory load
- Decreasing pH & myocardial load
- Reducing arrhythmias
- Reducing secondary polycythaemia
- Improving sleep quality
- Reducing disability
- Improving neuropsychological health
Domiciliary Oxygen Therapy

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  - Reducing secondary polycythaemia
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  - Reducing disability
  - Improving neuropsychological health

3 major kinds of home oxygen therapy

1. Long-term O2 therapy (LTOT) 
   (use ≥15 hours/day)
2. Short-burst O2 therapy (SBOT)
3. Ambulatory oxygen therapy
Indications for LTOT (revised BTS guidance 2015)

COPD/Cystic Fibrosis/ILD/Advanced Cardiac Failure

PaO2 \leq 7.3kPa (55mmHg); or
PaO2 \leq 8kPa (60mmHg) when stable with 1 or more of:
Secondary polycythaemia (HCT>0.55)
Peripheral oedema
Pulmonary hypertension

Pulmonary Hypertension (including idiopathic)
PaO2 \leq 8kPa (60mmHg)

Neuromuscular/chest wall disease
NIV +/- supplemental oxygen
Indications for NOT (revised BTS guidance 2015)

**COPD/CF/ILD:**
Is not indicated if criteria for LTOT not met

**Advanced cardiac failure:**
Can be used if nocturnal hypoxia plus evidence of sleep disordered breathing
Assess response, check for hypercapnia

**Neuromuscular/chest wall disease/OSA/OHS**
Is not indicated alone; can be used with ventilatory support
Oxygen Assessment

- 2 ABGs at least 3/52 apart - confident diagnosis, receiving optimum medical management & when stable (minimum 8/52)
- Titrate – 1LPM oxygen, 20 minute intervals
- Aim should be PaO2 ≥8.0kPa &/or SaO2≥90% without rise in PaCO2
- If PaCO2 rises >1kPa - ? unstable, reassess
- If PaCo2 rises >1kPa repeatedly – LTOT plus NIV
- DO NOT guide simply on SaO2
- If not meeting criteria for LTOT but borderline repeat again in 3/12
- Home visit by oxygen specialist within 4 weeks - education, risk assessment
- Follow up 6-12/12 after initial follow up, annual
ABG or CBG?

Meta-analyses
Consent for radial ABG needed
Earlobe CBG predicts arterial PaO2
(adjusted r² = 0.88, mean bias = 0.5 kPa/ 3.8mmHg cf. arterial)
May underestimate PaO2 by mean 0.17–0.32 kPa
Fingertip sampling not adequate
Can use ELBG or capnography for titration process
What about after hospital admission?

- Ideally should reassess after 8/52 when stable

- If symptomatic hypoxaemia (sats<92%), can provide temporary home oxygen, ensuring patient is aware this may be removed

- Must assess safety

- Must assess ongoing need at 8/52
Where’s the evidence?


- 3 UK centres
- Prospective randomised study
- 87 patients (all <70 yrs)
- Evidence of hypoxaemia (PaO2 40-60mmHg/ 5.3-8.0kPa), CO2 retention & CCF- cor pulmonale
- Randomised to nocturnal O2 or no O2
- Usually 2LO2/min via nasal cannulae for minimum 15 hours/day
- 19/42 O2 patients died in 5Y vs. 30/45 controls
NHLBI NOTT (Nocturnal Oxygen Therapy Trial) (1980)

- 6 centres
- 203 patients, hypoxaemic COPD (PaO2 < 55mmHg/ 7.3kPa or < 59mmHg/ 7.9kPa with signs of PH)
- Allocated to continuous O2 or 12h nocturnal O2
- F/u 19 months
- Mortality of nocturnal O2 group x1.94 that of continuous O2 group
- Included normocapnic patients (moderate/severe COPD)
Minimum 15 hours/day, benefit extends to 20 hours/day
Most desaturation occurs nocturnally so majority of use should be at night

In those with only nocturnal desaturation no survival benefit shown with nocturnal O2
How does it work

Effects on Pulmonary Arterial Pressure

- NOTT trial extension - survival after 8y related to decrease in mean PAP during 1st 6/12 therapy, improved PVR and SV

- MRC trial LTOT prevented a rise in PAP of 3mmHg (seen in controls) but did not reduce PAP

Less polycythaemia
1. Very small studies by modern standards
   Done at time when overlap COPD/OSA or COPD/OHS not recognised, and NIV not available

2. Survival benefit not seen until nearly 2 years
   Survival related to number of hours used
   So patients will only get benefit if use oxygen correctly and for long enough
   Treating moderate hypoxaemia (PO2>7.5) does not prolong survival – this is related to airway obstruction

SO LTOT PRESCRIPTION IS RARELY AN EMERGENCY
What’s the problem?

• Cost of provision of oxygen cylinders and concentrators

• Variable prescribing habits

• Poor guideline adherence

• Lack of follow-up & monitoring

• If reassess many patients ineligible after 3/12 stability

• Harm and waste in patients who smoke
Considering the cost…

85,000 LTOT patients (Engl)
£100 million/yr

11,000 users in London
£12 million/yr
Home Oxygen therapy and smoking: a dangerous practice

Annals Burns and Fire Disasters 2008

14-51% LTOT users smoke
86 cases of home oxygen burns
8% skin grafting
LOS 4.6 days, 12% died

25% of all O2 related domestic fires result in death, 30% result in serious injury
• LTOT studies we base our practice on did not control for smoking status (43% MRC and 38% NOTT smokers!)

• In later twelve year follow up study of patients with hypoxic cor pulmonale given domiciliary oxygen therapy, 51% continued to smoke, and 10 year survival was 26% (Cooper 1987)

• Degree of airflow obstruction is more important determinant of survival than hypoxaemia

• Smoking accelerates lung function decline

• Smoking increases the severity of secondary polycythaemia in patients with hypoxaemic COPD and prevents its correction by oxygen therapy

Support patients to stop smoking BEFORE LTOT is issued

Have and use a CO monitor within your team to confirm abstinence
Oxygen delivered by equipment carried by patient

Consider if:

- Evidence of exercise desaturation (≥4% to <90%) & motivated to use O2 (COPD, ILD, CF, chest wall, NM disorders but not CCF)

- Improvement demonstrated with oxygen therapy (improvement either walking distance or dyspnoea score with cylinder O2 vs. air)

- Exercise desaturation does not necessarily indicate need for ambulatory O2

- 12/52 double-blind, randomised, cross-over study 4L O2/min vs. cylinder compressed air for activities induce dyspnoea
- 39 subjects completed study
- Dyspnoeic but not chronically hypoxic COPD patients
- Exertional desaturation ≤88%
- Improved CRQ & HADS in O2 vs. air group
<table>
<thead>
<tr>
<th></th>
<th>Room air</th>
<th>Cylinder oxygen</th>
<th>Cylinder air</th>
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<tbody>
<tr>
<td>Subjects n</td>
<td>41</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>67.1 (9.3)</td>
<td></td>
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<tr>
<td>Male %</td>
<td>70</td>
<td></td>
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<tr>
<td>Body mass index</td>
<td>23.7 (4.4)</td>
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<tr>
<td>FEV1 % pred</td>
<td>25.9 (8.0)</td>
<td></td>
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<tr>
<td>Resting PaO2 kPa</td>
<td>9.2 (1.0)</td>
<td>377* (94)</td>
<td>337* (113)</td>
</tr>
<tr>
<td>Resting PaCO2 kPa</td>
<td>5.8 (0.7)</td>
<td></td>
<td></td>
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<tr>
<td>6MWD m</td>
<td>358 (93)</td>
<td>96 (1.7)</td>
<td>94 (1.9)</td>
</tr>
<tr>
<td>Pre-6MW SaO2</td>
<td>94 (1.9)</td>
<td></td>
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</tr>
<tr>
<td>Post-2MW SaO2</td>
<td>87 (2.9)</td>
<td></td>
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</tr>
<tr>
<td>Post-6MW SaO2</td>
<td>82 (5.4)</td>
<td>90 (4.2)</td>
<td>83 (4.2)</td>
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<tr>
<td>Pre-6MW Borg dyspnoea</td>
<td>0.7 (1.0)</td>
<td>0.7 (0.9)</td>
<td>0.7 (0.9)</td>
</tr>
<tr>
<td>Post-6MW Borg dyspnoea</td>
<td>4.7 (1.6)</td>
<td>4.1* (1.8)</td>
<td>4.8* (1.5)</td>
</tr>
</tbody>
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Data are presented as mean (SD). FEV1: forced expiratory volume in one second; PaO2: oxygen tension in arterial blood; PaCO2: carbon dioxide tension in arterial blood; 6MWD: 6-min walk distance; SaO2: arterial oxygen saturation. *: p=0.0001; *: p=0.005.
• Improved CRQ, HADS and SF-36 in O2 vs. air group

• Correction of exercise desaturation only achieved in 54%

• Acute response not good indicator of long-term response

• 41% acute or short-term responders did not wish to continue Rx
31 RCTs
534 subjects

Oxygen improved all pooled outcomes for
• endurance exercise capacity and
• maximal exercise capacity
All patients being considered for ambulatory O2 should be assessed by a specialist

- Determine extent of desaturation
- Improvement with supplemental O2
- Flow rate required for correction

AOT should not be offered routinely

- LTOT patients only eligible if mobile outdoors
- ILD patients not fulfilling criteria for LTOT may benefit
- 6/12 follow up
Don’t forget PULMONARY REHABILITATION
What’s more important?

Don’t forget acceptability & *ongoing* acceptability
Short burst O2 therapy (SBOT)

Intermittent use of supplemental O2

No evidence for use to relieve dyspnoea
Cluster Headache

= only condition with evidence base for use of SBOT (Grade A)

= only condition with evidence base for use of oxygen in absence of hypoxia

• 12-15LPM via non rebreathe mask for acute attacks
• Static plus portable cylinders
• Use for 15 minutes at onset of attack – stop if no effect, mop up if effective
• Infrequent attacks – GP to keep a copy of HOOF
• Care in smokers and those who may have undiagnosed COPD
Not indicated if SaO2 >92%

- Fan therapy effective due to cooling effect stimulating facial nerves
- Psychological dependence
- Barrier
- Restriction of activities
- Drying effect on mucous membranes
- Monitoring gases not appropriate
- Consider pharmacological therapies (BZDs, opiates) & role of psychology input
- How do you withdraw something that shouldn’t have been started?
Effect of palliative oxygen versus room air in relief of breathlessness in patients with refractory dyspnoea: a double-blind, randomised controlled trial

Amy P A Bernethy, Christine F McDonald, Peter A Frith, Katherine Clark, James E Herndon II, Jennifer Marcella, Iven H Young, Janet Bull, Andrew Wilcock, Sara Booth, Jane L Wheeler, James A Talsky, Alan J Crockett, David C Carrow

9 sites Australia, USA & UK

Double-blind RCT

Refractory dyspnoea but PaO2>7.3kPa

1:1 assignment to O2 at 2LO2/min or medical air via concentrator for 7/7 for 15 h/day
Mean breathlessness score (points)

- O₂: 0.9 vs Air: 0.7 points (am)
- 0.3 vs 0.5 (pm)

NO DIFFERENCE in symptoms or quality of life

“Since oxygen provides no additional symptomatic benefit for relief of refractory dyspnoea in patients with life-limiting illness ….try other strategies…..”
Oxygen is a medicine (medical gas)

- Inappropriate oxygen therapy may cause respiratory depression
- Inadequate monitoring and follow-up - at least annual review including pulse oximetry
- Value proposition and risks with smoking
Beware if...

NM disorders
Obesity
OSA with CPAP requirement
Spinal/chest wall disease
Overlap disorders

Must be assessed by expert in ventilation as likely to require ventilatory support (+/- supplemental O2)
Obesity: a growing problem
Warning signs:

- Morning headache
- Drowsiness
- Altered mood, irritability, concentration problems
- All patients and carers should be aware of what to look out for and who to contact
Oxygen alert cards and Patient Specific Protocols

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 ____% to ____% during exacerbations

Use compressed air to drive nebulisers (with nasal oxygen at 2 l/min).
If compressed air not available, limit oxygen-driven nebulisers to 6 minutes.

London Ambulance Service NHS Trust
Patient Specific Protocol

This protocol has been specifically prepared for the patient named below and details the treatment to be given in specified circumstances.

Patient's Name: ______________________
Date of Birth: ______________________

Address: ______________________

Reason for protocol: ______________________

Specific Treatment / Instructions:

If required please transport to the nearest A&E Department
All other aspects of clinical care remain unchanged.
If required contact EOC and ask for the Clinical Support Desk OR the On Call Clinical Advisor

Name of Referring Clinician:
Fiona Moore BSc, FRCS, FRCSEd, FCEM, FIMC (Ed)
Medical Director
London Ambulance Service NHS Trust
Date of Issue: ______________________

Page 1 of 1
LTOT is useful if used appropriately
Consider relative value and risk of harm in smokers
Strict criteria for LTOT
Assess & tailor for ambulatory O2 and ensure ongoing acceptance
Avoid SBOT but be aware of cluster headache use
Consider evidence base and better options in palliative care
Reassess, reassess, reassess!