22. **Lalloo UG,** Malolepszy J, Kozma D, *et al*. Budesonide and formoterol in a single inhaler improves asthma control compared with increasing the dose of corticosteroid in adults with mild-to-moderate asthma. *Chest* 2003;**123**:1480–7.
23. **Löfdahl C-G,** Reiss TF, Leff JA, *et al*. Randomised, placebo controlled trial of effect of a leukotriene receptor antagonist, montelukast, on tapering inhaled corticosteroids in asthmatic patients. *BMJ* 1999;**319**:87–90.
24. **British Thoracic Society, Scottish Intercollegiate Guidelines Network**. British Guideline on the Management of Asthma. *Thorax* 2008;**63**(Suppl IV):iv1–121.
25. **Harrald LR,** Patterson MK, Andrade SE, *et al*. Asthma drug use and the development of Churg–Strauss syndrome (CSS). *Pharmacoepidemiol Drug Saf* 2007;**16**:620–6.
26. **Bili A,** Condemi JJ, Bottone SM, *et al*. Seven cases of complete and incomplete forms of Churg–Strauss syndrome not related to leukotriene receptor antagonists. *J Allergy Clin Immunol* 1999;**104**:1060–5.
27. **Le Gall C,** Pham S, Vignes S, *et al*. Inhaled corticosteroids and Churg–Strauss syndrome: a report of five cases. *Eur Respir J* 2000;**15**:978–81.
28. **Solans R,** Bosch JA, Selva A, *et al*. Montelukast and Churg–Strauss syndrome. *Thorax* 2002;**57**:183–5.
29. **Nathani N,** Little MA, Kunst H, *et al*. Churg–Strauss syndrome and leukotriene antagonist use: a respiratory perspective. *Thorax* 2008;**63**:883–8.
30. **Hauser T,** Mahr A, Metzler C, *et al*. The leukotriene receptor antagonist montelukast and the risk of Churg–Strauss syndrome: a case crossover study. *Thorax* 2008;**63**:677–82.
31. **Weller PF,** Plaut M, Taggart V, *et al*. The relationship of asthma therapy and Churg–Strauss syndrome: NIH workshop summary report. *J Allergy Clin Imunol* 2001;**108**:175–83.

A breath of fresh air for acute oxygen treatment

Peter Calverley

Oxygen therapy given to acutely ill people is one of the commonest interventions used in modern medicine and has become part of the folklore of our times as a sick patient wearing an oxygen mask is pushed through the emergency department, both in real life and on television. Although the principles of oxygen treatment have been established by painstaking quantitative research over the past 60 years, in practice most people learn to use oxygen by following customary practice in their institution rather than considering rationally how it is best employed. A feeling that some oxygen is good, therefore more must be better, can be a dangerous precept to follow, whereas an unnecessary paranoia about inducing carbon dioxide retention can deny some people potentially life saving treatment. These uncertainties make the arrival of the new British Thoracic Society Guideline for Emergency Oxygen Use in Adults[1] particularly welcome. This rather daunting document, which has been extensively endorsed by key professional societies and practitioner groups, provides a comprehensive and at times exhaustive review of the theory and practice of acute oxygen treatment. Its scope ranges from a detailed consideration of how oxygen therapy influences respiratory physiology and tissue oxygen delivery through to which mask should be chosen and how oxygen services should be developed. It is a major resource which answers almost any question about acute oxygen therapy but is best consulted selectively, depending on the interest and needs of the user. The helpful summary of key recommendations contains the essential knowledge for most practitioners while the "summary of the summary" at the beginning of this document is worth committing to memory.

This Guideline represents the views of a wide constituency of oxygen users and this in itself has contributed to its complexity. It has followed a robust methodology with careful consideration of the nature of the evidence available although, as the authors make clear, this evidence is largely grade 3 and grade 4 (ie, based on observational clinical studies or expert opinion). This does not mean it is less important but is simply a reflection of the difficulties of conducting appropriate clinical trials in a setting where informed patient consent is often impossible to obtain and where there is a risk that withholding the intervention might seriously disadvantage the patient. It would be impossible and inappropriate to review all the many recommendations in this editorial but some flavour of the scope of what is covered might be helpful.

The Guideline is quite clear that oxygen is given acutely for the treatment of hypoxaemia. This is not the same as giving oxygen to treat breathlessness, as many hypoxaemic patients are not particularly breathless while many breathless patients are not hypoxaemic. Providing patients with a flow of gas over the face may decrease the perception of breathlessness but this mechanism, if it operates in many of the clinical circumstances reviewed here, is certainly not the same as giving oxygen to improve tissue oxygen delivery. Focusing on this more important and better validated use of oxygen has two immediate consequences. The first is that treating tissue hypoxia involves more than just increasing the oxygen concentration. In some circumstances, oxygen delivery is best improved by increasing cardiac output or correcting anaemia and this integrated approach to care based on a proper diagnosis is central to the Guideline recommendations. The second consideration is more practical. Hypoxaemic patients need to be properly identified and it is unwise to rely too much on a clinical diagnosis of central cyanosis. This is less of a problem than in the past as there is a widespread availability of reliable and relatively artefact free pulse oximeters. The section on using oximeters is well worth reading and particularly the pitfalls that follow when measurement is attempted in patients with poor peripheral circulation or even those still wearing nail varnish! Using pulse oximetry and an assessment of the patient's severity, a reasonable inspired oxygen concentration can be selected with the patient's oxygen saturation targeted to maximise benefit and minimise harm.

There is more enthusiasm for diagnosing hypercapnia on clinical grounds, although a good sensitivity and specificity analysis on any of the clinical signs cited is currently lacking. The hazards of hypercapnia in patients with hypoxaemic chronic obstructive pulmonary disease (but also in those with the rarer conditions, such as neuromuscular disease), are appropriately discussed and the proposed target oxygen saturation of 88–92% in these patients is a sensible recommendation. Even more important is the proposal for a specific oxygen alert card in patients who are at risk of carbon dioxide retention when they receive oxygen or have exhibited this problem in the past. Widespread uptake of this sensible idea would reduce the inadvertent harm done to these patients when transferred to hospital while acutely ill and it is being trialled by many ambulance Trusts in the UK. Although the information contained

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in tables 1–4 appears to be rather complex, they are well worth studying. The clinical setting is related to the likely flow rate required or, in the case of several examples in table 4, to the need for oxygen at all. This is turned into a practical approach to oxygen use in fig 1, which merits wall space in any emergency room, acute medical or respiratory unit. The second figure indicating how oxygen can be given in different environments is likely to be helpful to those less familiar with this practical but relevant skill.

Not only does this Guideline address practical aspects of oxygen delivery in many different settings but it also considers what information might be needed to set up or commission an oxygen service. One key recommendation is the use of an oxygen prescription chart which emphasises that oxygen really is a drug in a hospital setting, even if it does not need a formal prescription as when used in patient transfer. Paying attention to the flow rate of oxygen and how long it needs to be given to the patient will both improve patient safety and decrease inconvenience, for instance when oxygen is delivered for much longer than clinically indicated, hampering patient communication and increasing the worry of those relatives. The Guideline is supported by a wealth of appendices also available online which means that there is literally something here for everyone concerned with acute oxygen care.

The team who worked on this Guideline and especially Drs O'Driscoll, Howard and Davidson, are to be congratulated on their hard work and commitment to deliver a comprehensive but ultimately practical guide as to how we should use one of our most familiar therapies. The challenge now falls to the rest of us to take the advice proffered, customise it to our own circumstances and ensure that the good sense contained with this Guideline translates into even better care for these acutely ill patients.

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Thorax 2008;**63**:849–850. doi:10.1136/thx.2008.105148

REFERENCE

1. **O'Driscoll BR,** Howard LS, Davison AG. BTS guideline for emergency oxygen use in adult patients. *Thorax* 2008;**63**;Suppl VI.

Recent advances in exacerbations of COPD

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The First International Conference on Exacerbations of Airways Disease (ICEAD) brought together experts from both sides of the Atlantic to discuss problems in the management of exacerbations of both asthma and chronic obstructive pulmonary disease (COPD). A brief overview of these discussions on COPD exacerbations follows.

DEFINITIONS AND EPIDEMIOLOGY

Symptom and treatment based definitions

Up to 18 definitions of a COPD exacerbation have been advanced, from less explicit[1] to very explicit symptom based criteria.[2 3] However, large therapeutic trials in COPD have all used treatment based definitions.[4 5] The first consensus definition was treatment based but the current GOLD guidelines accept a symptom based definition.[6 7] Biochemical or physiological markers applied in studies using either definition[8 9] have all shown significant changes, supporting the validity of these approaches to a definition but lack specificity and sensitivity. The healthcare utilisation approach to severity of exacerbation may be more robust as it has been related to mortality[10] but does not allow for detection of untreated exacerbations, which may contribute to poor quality of life.

Health burden

COPD patients have about 0.5–3.5 exacerbations/year, 0.09–2.4 hospitalisations/year and inhospital mortality varies between 10% and 60%, depending on the severity of COPD. Overall, the death rate varies from 5.4 per 1000 person years among normal subjects to 42.9 among subjects with GOLD stage 3 or 4.[11] Thus COPD exacerbations are a significant cause of death, mainly in patients with more severe COPD. This leads to a high cost of COPD care which can be effectively reduced through decreasing hospitalisation.[12]

AETIOLOGY AND SUSCEPTIBILITY

Airway bacterial infection

Bacteria may be detected in up to 60% of exacerbations and viruses in 23–60%.[13 14] A study of hospitalised patients found bacteria in 25%, bacteria and viruses in 25% and viruses alone in 25%, and no infectious agent in another 25%.[14] The acquisition of new strains of bacteria is associated with an increased risk of COPD exacerbation, more inflammation and strain specific immunity.[15 16] However, non-specific reduction of bacterial load during recovery from COPD exacerbation has been associated with resolution of inflammation.[9]

Viruses and coinfection

Respiratory viruses have been associated with higher exacerbation sputum interleukin (IL)-6 levels, with prolonged COPD exacerbations and significant health burden.[13 17 18] Furthermore, bacteria–virus coinfection leads to greater lung impairment and longer inhospital stay.[14]

Susceptibility and inflammation

Frequent COPD exacerbators appear to be a distinct phenotype characterised by a faster decline in lung function, poorer quality of life scores, more viruses at exacerbation, higher mortality, greater airway inflammation and higher airway bacterial load.[3 8 10 13 19] Frequent exacerbators also have smaller reductions in systemic inflammation post-exacerbation further. Interestingly, a high serum C reactive protein concentration post-exacerbation is associated with recurrence.[20]

Susceptibility and other factors

Viruses detected in lower airway samples of patients with stable COPD affect systemic inflammatory processes as well as lower airway bacterial load.[13 21] Thus it is likely that the susceptibility of the frequent exacerbator phenotype is associated with viral colonisation of the lower airway though this has yet to be proved.

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