

# The use of oxygen in acute exacerbations of chronic obstructive pulmonary disease: a prospective audit of pre-hospital and hospital emergency management

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**ABSTRACT** – Treatment with high-flow oxygen in acute exacerbations of chronic obstructive pulmonary disease (AECOPD) can cause or aggravate acute hypercapnic respiratory failure and adversely affect prognosis. National guidelines for the management of COPD recommend an initial fractional inspired oxygen concentration (FiO<sub>2</sub>) of no more than 0.28. However, a prospective audit of 101 consecutive episodes of AECOPD demonstrated that oxygen therapy with an FiO<sub>2</sub> in excess of 0.28 is common, potentially deleterious and predominantly initiated in the ambulance. Patient awareness, aids to disease identification and ambulance protocols are likely to hold the key to improvement in the acute care of these patients.

**KEY WORDS:** chronic obstructive pulmonary disease, hypercapnic respiratory failure, respiratory acidosis

A recent study found that one in five patients admitted to hospital with an acute exacerbation of chronic obstructive pulmonary disease (AECOPD) had a respiratory acidosis<sup>1</sup>. It was reported over 50 years ago that administration of high-flow oxygen to patients with an exacerbation of COPD could worsen respiratory acidosis<sup>2,3</sup>, which in turn is an adverse prognostic factor<sup>4</sup>. The British Thoracic Society recommend that a fractional inspired oxygen concentration (FiO<sub>2</sub>) of no more than 0.28 is given to COPD patients until the result of arterial blood gas analysis is available<sup>5</sup>. However, the need for controlled oxygen therapy is often not recognised in guidelines for ambulance and nursing staff, and may be dismissed by intensivists<sup>6</sup> on the basis that hypoxaemia is more dangerous than hypercapnia for most patients without a diagnosis in the accident and emergency department (A&E).

Patients presenting to the respiratory services with an exacerbation of COPD had been observed to have a high prevalence of acute hypercapnic acidosis with high oxygen tension. The reasons for this iatrogenic problem and its prevalence were investigated in a prospective audit.

## Methods

All consecutive patients presenting between 28 February and 30 April 2000 with a final diagnosis of AECOPD made by a respiratory physician were included in the study. Information on oxygen therapy, arterial blood gas analysis, triage category (a grading for urgency/severity), diagnosis and mortality was obtained from the ambulance and hospital case records and patient interview. A total of 97 patients (mean (standard deviation (SD)) age 69.7 years (9), 55 male, 37 current smokers) were admitted on 101 occasions with an AECOPD.

## Results

### Oxygen administration

At some stage during initial management in the ambulance or the A&E department, 57 (56%) patients received an FiO<sub>2</sub> greater than 0.28 (Table 1). The median (interquartile range) duration from collection by ambulance to triage registration was 30 (16) min, and to first arterial blood gas 1 hour 4 min (1 hour 52 min).

### Respiratory acidosis and mortality

Arterial blood gases were measured in 83 (82%) patients (Table 2). In-hospital mortality was 8/57 (14%) for patients administered an FiO<sub>2</sub> above 0.28, and 1/44 (2%) for those administered an FiO<sub>2</sub>

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**Table 1. Oxygen administration in the ambulance or accident and emergency department (A&E).**

Initial management	Total patients treated	FiO <sub>2</sub> >0.28	
		No.	%
Ambulance personnel	65	52	80
Triage nurse	101	24	24
A&E	65	20	39
Respiratory medical officers	101	11	11

FiO<sub>2</sub> = fractional inspired oxygen concentration.

less than or equal to 0.28 ( $p < 0.05$ , Fisher's exact test). These groups were comparable with respect to triage category, age and smoking history. In-hospital mortality was greatest in the group with a severe respiratory acidosis (4/14 (29%) patients), followed by the group with mild acidosis (2/19 (11%)), those with unknown acid-base status (1/17 (6%)) and the non-acidotic group (2/51 (4%)).

### Recognition of chronic obstructive pulmonary disease

At interview, 35 (35%) patients self-identified their lung disease as COPD or emphysema, whereas 47 (47%) patients described it as asthma. Ambulance crews correctly identified COPD in 21/65 (32%), labelling the disease as asthma in 19/65 (29%). In the ambulance, patients identified as COPD received a mean (SD)  $\text{FiO}_2$  of 0.47 (0.19) versus 0.60 (0.24) if not identified ( $p < 0.05$ , unpaired t-test); if labelled as asthma the mean (SD)  $\text{FiO}_2$  was 0.62 (0.24).

### Discussion

This audit confirmed that the use of uncontrolled oxygen therapy in patients with AECOPD is common, notably in the ambulance and during the early stages of admission, and that it is associated with a higher frequency of severe respiratory acidosis and mortality. This is not due to adverse factors such as more severe exacerbation, more comorbidity or greater age; these factors were similar between the group receiving an  $\text{FiO}_2$  above 0.28 and those receiving an  $\text{FiO}_2$  below or equal to 0.28. Mortality data have hitherto been scarce, with either only limited evidence provided or acidosis used as a surrogate end-point<sup>4</sup>.

Failure to identify correctly the underlying disease is a contributory factor, especially by the emergency services (32%

## Key Points

**National guidelines for the management of acute exacerbations of chronic obstructive pulmonary disease recommend an initial fractional inspired oxygen concentration ( $\text{FiO}_2$ ) of no more than 0.28**

**Oxygen therapy with an  $\text{FiO}_2$  in excess of 0.28 is common; it may cause or aggravate acute hypercapnic respiratory failure and is associated with a higher mortality**

**An  $\text{FiO}_2$  in excess of 0.28 is most often initiated during ambulance transportation but is perpetuated by hospital emergency staff**

**Patient and staff education, aids to disease identification and ambulance protocols are likely to hold the key to improvement in the acute care of these patients**

correct) and the patients (35% correct). It is of note that when COPD was correctly identified, the ambulance crew, on average, administered a lower  $\text{FiO}_2$  but not at the recommended level. Even when identified correctly, a lack of guidelines for ambulance and triage staff may lead to uncertainty. The commonly used Manchester triage protocol does not recognise COPD, having algorithms only for 'shortness of breath' and 'asthma'. The period of exposure to uncontrolled oxygen outside the hospital and in A&E was not particularly short and there were considerable delays before obtaining arterial blood gas analysis.

The significant increase in both severe respiratory acidosis and mortality suggest that the use of controlled oxygen in AECOPD is an important therapeutic issue – which appears, however, in danger of being forgotten in the modern A&E department. The absolute difference in mortality in the two groups, 14% ( $\text{FiO}_2 > 0.28$ ) versus 2% ( $\text{FiO}_2 \leq 0.28$ ), would, if confirmed in larger studies, suggest a 12% excess mortality or a 'numbers needed to harm' of nine patients. A randomised controlled trial of different oxygen concentrations administered to patients with AECOPD is impossible for ethical reasons. The established physiological principles of oxygen therapy bear revisiting<sup>3</sup>, and training in the use of such therapy is desirable for the staff involved in the care of patients with AECOPD. Patient awareness, aids to disease identification and ambulance protocols are likely to hold the key to improvement in the acute care of these vulnerable patients.

We are investigating the use of a 'COPD card' that is held by the patient and contains essential information but is small enough to fit in a wallet. A computerised register of patients at risk of adverse effects from uncontrolled oxygen therapy could be held by ambulance control. Patient and staff education is being carried out in cooperation with the emergency services. Closure of the audit cycle by re-audit (in 2003) will assess whether there has been any improvement.

### References

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**Table 2. Arterial blood gas results (first measurements shown) and mortality in patients with acute exacerbations of chronic obstructive pulmonary disease receiving either high flow or controlled oxygen.**

	$\text{FiO}_2 > 0.28$ (n=57)		$\text{FiO}_2 \leq 0.28$ (n=44)		p value
Arterial blood gas measured (no. (%))	51	89%	32	73%	
H <sup>+</sup> (mean (SD))	48.1	15.7	42.5	7.6	<0.05
pCO <sub>2</sub> (mean (SD))	8.1	3.3	7.2	1.9	NS
pO <sub>2</sub> (mean (SD))	11.3	4.9	10.0	2.3	NS
No acidosis	27	53%	23	72%	NS
Mild acidosis (H <sup>+</sup> >45 and <55 nmol/l)	11	22%	8	25%	NS
Severe acidosis (H <sup>+</sup> 55 nmol/l)	13	25%	1	3%	<0.005
Mortality (no. (%))	8	14%	1	2%	<0.05

$\text{FiO}_2$  = fractional inspired oxygen concentration.

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