Respiratory Medicine (2008) **102**, 1827–1829

available at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/rmed

SHORT COMMUNICATION

# Inspired fraction of carbon dioxide in oxygen supply to chronic pulmonary disease

**Antoine Fremault [a,c], Mitchell Silva [b,c], François Beaucage [a], Daniel Berckmans [b], Marc Decramer [a,*]**

[a] *Department of Pneumology, University Hospital, University of Leuven, Herestraat 49, 3000 Leuven, Belgium*
[b] *Measure, Model and Manage Bio-Responses, Faculty of Applied Bioscience and Engineering , University of Leuven, Herestraat 49, 3000 Leuven, Belgium*

Received 3 July 2008; accepted 26 August 2008
Available online 1 October 2008

**KEYWORDS**
COPD;
Oxygen;
Carbon dioxide;
Retention;
Inspired fraction

**Summary**
Hypoxemic patients with chronic obstructive pulmonary disease (COPD) are at risk of carbon dioxide ($CO_2$) retention during oxygen therapy and hypercapnia in COPD is associated with an ominous prognosis. Rebreathing with oxygen mask is possible in practice and possibly affects $CO_2$ retention due to an increased inspired fraction of $CO_2$. Its effects on arterial partial pressure of $CO_2$ during oxygen supply have, to the best of our knowledge, never been studied. We measured the inspired fraction of $CO_2$ in eighteen non-hypoxemic stable COPD patients with a capnograph during a 5 min trial with two different modes of oxygen supply (oxygen mask without reservoir bag and nasal prongs, respectively at a flow of 10 l/min and 2 l/min). We found no significant increase in inspiratory $CO_2$ concentration. These findings suggest that inspired fraction of $CO_2$ does not increase markedly during controlled oxygen therapy.


## Introduction

Oxygen supply is a life saving treatment and is widely used by medical and paramedical staff. When oxygen is supplied to Chronic Obstructive Pulmonary Disease (COPD) patients, it might occur that carbon dioxide ($CO_2$) retention occurs and causes a respiratory acidosis that may be fatal.[1] Despite extensive research, the mechanism responsible for hypercapnia is not completely understood and $CO_2$ is a major determinant of respiratory stimulation in many COPD patients with acute respiratory failure.[2] Prolonged expiration is typically observed by COPD patients and if expiration is prolonged, the oxygen and $CO_2$ concentrations

* Corresponding author. Tel.: +32 16346807; fax: +32 16346803.
*E-mail address:* marc.decramer@uz.kuleuven.be (M. Decramer).
[c] Contributed equally.

0954-6111/$ - see front matter © 2008 Published by Elsevier Ltd.
doi:10.1016/j.rmed.2008.08.015

in the expired gas approach to those of mixed venous blood.[3] An older study showed that some devices used to administer oxygen therapy could cause appreciable rebreathing.[4] We hypothesized that an increased inspired fraction of $CO_2$ due to this rebreathing could be a possible cause of this $CO_2$ retention during controlled oxygen therapy. We quantified the inspired fraction of $CO_2$ in two different modes of oxygen supply (oxygen mask and nasal prongs, respectively at a flow of 10 l/min and 2 l/min) during a 5 min trial with eighteen non-hypoxemic stable COPD patients. To assess a possible change in $CO_2$ concentration during the test, the $CO_2$ concentration of the first minute was compared with the $CO_2$ concentration of the last minute.

## Methods

Eighteen stable patients with COPD from the Pulmonary Rehabilitation Unit were enrolled: 78 % were men, mean (±SEM) age was $66.9 \pm 1.5$ years, mean (±SEM) forced expiratory volume in 1 s was $1.17 \pm 0.35$ l or $43.3 \pm 15.3$ % predicted with a mean (±SEM) diffusing capacity of the lungs for carbon monoxide of $44.7 \pm 15\%$ predicted. Diagnosis of COPD was based on the GOLD (Global Initiative for Chronic Obstructive Lung Disease) criteria: a smoking history (>10 pack years) and a spirometry with a Tiffeneau index $< 0.7$. Patients had no acute exacerbation of their disease and no symptoms of heart failure or pneumonia at the moment of the examination. All measurements were done under full inhalation therapy according to the GOLD stages but none of the patients had long-term oxygen therapy. Pulmonary function measurements were the latest available in the patient's file. On average, they preceded the examination by a mean (±SEM) of 80.9 (±19.5) days.

Two modes of oxygen supply were assessed: the first was oxygen supply in an oxygen mask (without reservoir bag) with a flow of 10 l/min, the second way was oxygen supply via nasal prongs with a flow of 2 l/min. Two variables were measured: oxygen concentration and carbon dioxide concentration. This was done by calculating the mean $CO_2$ and $O_2$ concentration of the first and the last minute. The subjects were asked to breathe normally in a quiet environment during 5 min with each method of oxygen supply.

A capnograph (Normocap 200, Datex, Finland) was used to record the gas concentrations (measured in %, with a sampling frequency of 100 Hz). Calibration was done before every test in which the $CO_2$ concentration of the ambient air was taken as base line. Respiratory rate was calculated from the $CO_2$ curve as the number of breathing cycles per minute. Out of every breathing curve the percentage of inspiratory $CO_2$ was calculated. Data was processed in Matlab v 7.4.0287 (the Mathworks Inc., Natick, MA, USA). For the comparison between groups one way analysis of variance has been used and $P < 0.05$, two sided was used as the limit of statistical significance.

## Results

For every patient, the mean $CO_2$ concentration at inspiratory level was calculated for the first and last minute of the oxygen mask. The same was done for the nasal prongs. To verify if there was an increase or decrease in $CO_2$ concentration, the ratio between the last minute and first minute was calculated. This was done for both methods of oxygen supply.

For the oxygen mask average $CO_2$% in the first minute was (mean ± SD) $0.0046 \pm 0.0021\%$, for the last minute $0.0037 \pm 0.0020\%$. For the nasal prongs average in the first minute was $0.0044 \pm 0.0020\%$ and in the last minute $0.0043 \pm 0.0020\%$. Differences were not statistically significant: $P = 0.19$ for the oxygen mask and $P = 0.89$ for the nasal prongs (see Fig. 1).

## Discussion

Treatment with oxygen, an available and commonly prescribed drug, has been shown to improve long-term survival in patients with severe chronic bronchitis and emphysema[5] but it is often difficult to adequately oxygenate COPD patients with respiratory failure, the major risk being to precipitate $CO_2$ retention and significant acidosis. Physiological studies have shown that hyperoxia-induced hypercapnia is primarily due to impairment in gas exchange rather than to depression of ventilation.[6,7] Another possible cause of this $CO_2$ retention during controlled oxygen therapy could be an increased inspired fraction of $CO_2$. In our study, we found first, that inspired $CO_2$-fraction was low in all circumstances and second, that there was no significant increase in inspiratory $CO_2$ concentration after a 5 min duration trial done by stable non-hypoxemic COPD patients when comparing the concentrations of the inspiratory $CO_2$ at the first minute to those at the end of the trial, independently of the way of oxygen supply. These findings suggest that the risk of $CO_2$ retention due to an increased inspired fraction of $CO_2$ by non-hypoxemic stable COPD patients is low, that the ventilatory drive in our patients was unaffected by the way of oxygen supply and that there was no increased ventilation-perfusion mismatching. The present study may be, however, limited due to the current limited sample and by the absence of invasive confirmation by blood gas measurements. However, a more detailed study is needed to confirm these results in acutely hypoxemic or mildly hypercapnic patients to

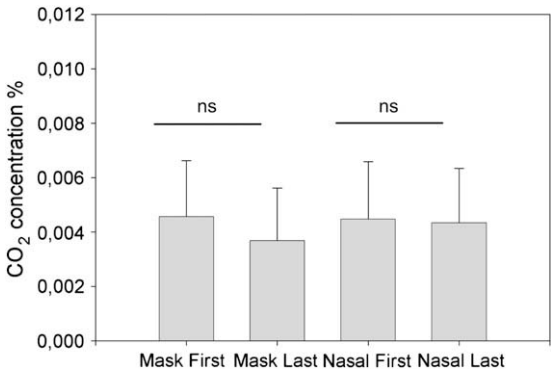


**Figure 1** Mean and standard deviation of inspiratory $CO_2$ concentration for first minute and last minute for a oxygen mask without reservoir at a flow of 10 l/min, and for first minute and last minute for nasal prongs at a flow of 2 l/min.

avoid undertreatment of serious hypoxemia that can result in death and cardiac arrhythmias.[8]

## Conflict of interest

The authors have no conflict of interest.

## References


1. Leach RM, Bateman NT. Acute oxygen therapy. *Br J Hosp Med* 1993 May 5;**49**(9):637—44.
2. Tardif C, Bonmarchand G, Gibon JF, Hellot MF, Leroy J, Pasquis P, et al. Respiratory response to CO2 in patients with chronic obstructive pulmonary disease in acute respiratory failure. *Eur Respir J* 1993 May;**6**(5):619—24.
3. Rahn H, Fenn WO. *A graphical analysis of respiratory gas exchange*. Washington, DC: American Physiological Society; 1955. 2008. Ref Type: Generic.
4. Bethune DW, Collis JM. An evaluation of oxygen therapy equipment. Experimental study of various devices on the human subject. *Thorax* 1967 May;**22**(3):221—5.
5. Long term domiciliary oxygen therapy in chronic hypoxic cor pulmonale complicating chronic bronchitis and emphysema. Report of the Medical Research Council Working Party. *Lancet* 1981 Mar 28;**1**(8222):681—6.
6. Sassoon CS, Hassell KT, Mahutte CK. Hyperoxic-induced hypercapnia in stable chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1987 Apr;**135**(4):907—11.
7. Aubier M, Murciano D, Milic-Emili J, Touaty E, Daghfous J, Pariente R, et al. Effects of the administration of O2 on ventilation and blood gases in patients with chronic obstructive pulmonary disease during acute respiratory failure. *Am Rev Respir Dis* 1980 Nov;**122**(5):747—54.
8. McNicholas WT, Fitzgerald MX. Nocturnal deaths among patients with chronic bronchitis and emphysema. *Br Med J (Clin Res Ed)* 1984 Oct 6;**289**(6449):878.