

CASE REPORT

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Carbon dioxide narcosis due to inappropriate oxygen delivery: a case report

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Abstract

Background: Oxygen delivery to patients with chronic obstructive pulmonary disease may be challenging because of their potential hypoxic ventilatory drive. However, some oxygen delivery systems such as non-rebreathing face masks with an oxygen reservoir bag require high oxygen flow for adequate oxygenation and to avoid carbon dioxide rebreathing.

Case presentation: A 72-year-old Caucasian man with severe chronic obstructive pulmonary disease was admitted to the emergency department because of worsening dyspnea and an oxygen saturation of 81% measured by pulse oximetry. Oxygen was administered using a non-rebreathing mask with an oxygen reservoir bag attached. For fear of removing the hypoxic stimulus to respiration the oxygen flow was inappropriately limited to 4L/minute. The patient developed carbon dioxide narcosis and had to be intubated and mechanically ventilated.

Conclusions: Non-rebreathing masks with oxygen reservoir bags must be fed with an oxygen flow exceeding the patient's minute ventilation (>6–10 L/minute). If not, the amount of oxygen delivered will be too small to effectively increase the arterial oxygen saturation. Moreover, the risk of carbon dioxide rebreathing dramatically increases if the flow of oxygen to a non-rebreathing mask is lower than the minute ventilation, especially in patients with chronic obstructive pulmonary disease and low tidal volumes. Non-rebreathing masks (with oxygen reservoir bags) must be used cautiously by experienced medical staff and with an appropriately high oxygen flow of 10–15 L/minute. Nevertheless, arterial blood gases must be analyzed regularly for early detection of a rise in partial pressure of carbon dioxide in arterial blood in patients with chronic obstructive pulmonary disease and a hypoxic ventilatory drive. These patients are more safely managed using a nasal cannula with an oxygen flow of 1–2L/minute or a simple face mask with an oxygen flow of 5L/minute.

Keywords: Non-rebreathing mask, Oxygen inhalation therapy, Obstructive lung diseases, Hypercapnia, Respiratory acidosis

Background

In 2011, chronic obstructive pulmonary disease (COPD) had a global prevalence of 12% and was the third leading cause of death in the USA [1]. Oxygen (O₂) delivery to COPD patients with an acute disease exacerbation remains challenging. High inspired O₂ concentrations should be used with caution, because COPD patients may breathe with a hypoxic drive [2]. To avoid carbon

dioxide (CO₂) narcosis, O₂ must be provided in a controlled fashion with a target saturation of only 88–92% [3]. This recommendation was studied in a prehospital care setting of 405 patients with COPD exacerbations: When O₂ was delivered via nasal prongs with a target percentage of oxygen saturation of arterial blood of 88–92%, measured by pulse oximetry (SpO₂), a significantly lower in-hospital mortality (4% versus 9%, $p = 0.02$) was observed when compared with standard O₂ delivery using a non-rebreathing mask [4]. In addition, there were fewer hypercapnic and acidotic episodes in the group with O₂ delivered via a nasal cannula. Accordingly, the British Thoracic Society recommends three

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options for oxygen delivery to COPD patients at risk for hypercapnic respiratory failure: (i) Venturi mask 28% at 4 L/min. or Venturi mask 24% at 2–4 L/min. with a target SpO₂ of 88–92%. (ii) If the SpO₂ falls below 88%, O₂ delivery should be changed to a nasal cannula with an O₂ flow 2–6 L/min. or a simple face mask with an O₂ flow of 5 L/min. (iii) Increases in partial pressure of carbon dioxide in arterial blood (P_aCO₂) or progressive acidotic pH require noninvasive ventilation or intubation and mechanical ventilation [5]. Non-rebreathing masks may be used in severely hypoxic patients. It is essential to set the O₂ flow of non-rebreathing masks to 10–15 L/min. in order to avoid CO₂ rebreathing. Because of the risk of hypercapnic respiratory failure, blood gases must be analyzed every 30–60 minutes. We report the case of a patient with COPD who developed CO₂ narcosis because of inadequate use of the non-rebreathing mask.

Case presentation

A 72-year-old, cachectic (height 170 cm, weight 50 kg, BMI 17.3 kg/m²) Caucasian man with COPD and a history of smoking had increasing shortness of breath. Pulmonary function testing performed 9 years prior showed a decreased forced expiratory volume in 1 second (FEV₁) [38% of predicted, Global Initiative for Chronic Obstructive Lung Disease (GOLD) stage 3], which did not increase after albuterol inhalation. The high total lung capacity and residual volume were consistent with emphysema. On arrival of the emergency medical team at his home, our patient was dyspneic but alert. His vital signs were: blood pressure 150/100 mmHg, heart rate 103 bpm, and SpO₂ 81%. High-flow O₂ was supplied at 10 L/min. using a non-rebreathing mask with an O₂ reservoir bag (Fig. 1). The SpO₂ increased to 99% within 20 minutes, and our patient was transported to the emergency department. As it was unknown whether our patient had a hypoxic respiratory drive, O₂ flow was erroneously limited to 4 L/min. He got increasingly irritated, had no headache, but was no longer oriented to time, place, and person, and became unconscious, gasping for air 2 hours later. His blood pressure was 110/80 mmHg, and the heart rate was 80 bpm. Breath sounds were distant, and his tongue was cyanotic. An arterial blood gas analysis taken shortly after starting bag-valve-mask ventilation showed marked hypercapnia with respiratory acidosis (Table 1A). Noninvasive ventilation was not a possible option [6]. Our patient was intubated, mechanically ventilated, and received albuterol and ipratropium bromide by inhalation. Methylprednisolone, amoxicillin clavulante plus clarithromycin (for an infiltrate in the right paracardiac region), and low-dose theophylline were administered intravenously. Four hours later, our patient was extubated (Table 1B), and was later transferred to a medical ward. The high P_aCO₂ was explained by low

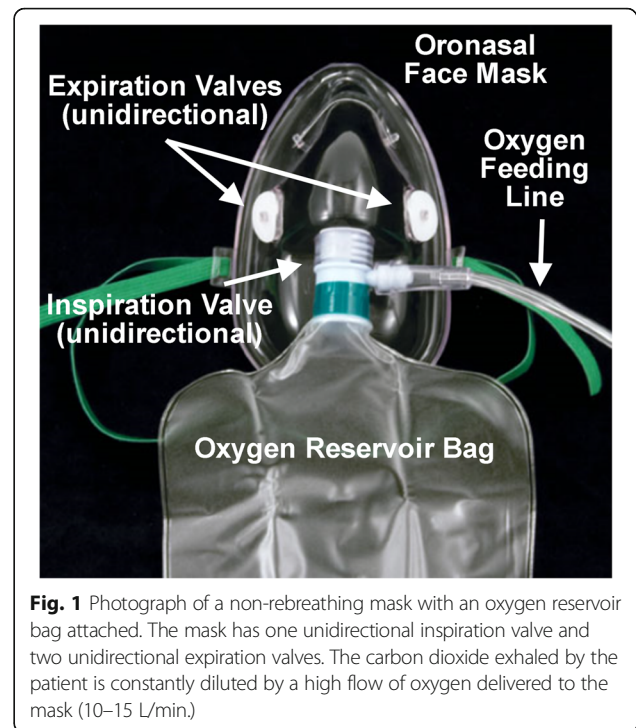


Fig. 1 Photograph of a non-rebreathing mask with an oxygen reservoir bag attached. The mask has one unidirectional inspiration valve and two unidirectional expiration valves. The carbon dioxide exhaled by the patient is constantly diluted by a high flow of oxygen delivered to the mask (10–15 L/min.)

tidal volumes and a probably hypoxic ventilatory drive (Table 1C). Because of our patient's worsening dyspnea, he mistakenly received O₂ at 2 L/min. by the same non-rebreathing mask (Fig. 1). After a few hours, intensive care unit (ICU) admission was required due to hypotension (75/50 mmHg) and bradypnea (Table 1D). He was re-intubated and mechanically ventilated for 24 hours. Norepinephrine was given to stabilize the blood pressure. An electrocardiogram (ECG) showed a sinus rhythm with right bundle branch block and right ventricular hypertrophy, and the echocardiography documented a chronic cor pulmonale with pulmonary arterial hypertension. Respiratory acidosis improved, and the patient was temporarily extubated (Table 1E). However, aspiration of a pea (removed by bronchoscopy, Fig. 2) with atelectasis of the right lung again necessitated mechanical ventilation and a tracheostomy. One month after admission, the exhausted patient died of CO₂ narcosis (Table 1F). An autopsy was not performed.

Discussion

The first two episodes of CO₂ narcosis (Table 1A, D) were due to near asphyxia and CO₂ rebreathing because of incorrect use of the non-rebreathing mask by providing insufficient (2–4 L/min.) O₂ supply. The third episode was due to probable hypoventilation and severe exhaustion (Table 1F).

Assuming a reduced tidal volume [7] of 200 mL (normal 7 mL/kg body weight = 350 mL) and a respiratory rate of 30/min., our patient's estimated minute ventilation was 6

Table 1 Arterial blood gas analyses at different time points during the hospital stay

Time point (see text)	A, day 1	B, day 1	C, day 3	D, day 4	E, day 5	F, day 30
O ₂ flow rate; mode of administration	4 L/min.; non-rebreathing mask	2 L/min.; nasal cannula	2 L/min.; nasal cannula	2 L/min.; non-rebreathing mask	2 L/min.; nasal cannula	2 L/min.; tracheal cannula
pH (7.3–7.4)	7.1	7.3	7.4	7.2	7.4	7.1
P _a O ₂ (kPa ^a [11.1–14.4])	10.9	10.4	6.8	6.0	8.1	10.9
P _a CO ₂ (kPa ^a [4.7–6.4])	20.0	9.3	7.8	15.8	8.3	20.7
Bicarbonate (mmol/L [24–31])	41.0	37.5	35.4	42.7	36.7	49.8
Base excess (-1.5 to +3.0)	3.0	8.2	9.1	9.4	10.5	17.7
S _a O ₂ (% [95.0–99.9])	89.4	92.5	84.8	70.3	88.6	94
Respiratory rate (L/min. [12–16])	ND	30	24	Apneic	30	ND

ND not determined, O₂ oxygen, P_aO₂ partial pressure of oxygen in arterial blood, P_aCO₂ partial pressure of carbon dioxide in arterial blood, pH power of hydrogen; negative logarithm of the hydrogen ion concentration, S_aO₂ percentage of oxygen saturation of arterial blood

^aTo convert kPa to mmHg, multiply by 7.5

L/minute (respiratory minute volume [mL] = tidal volume [mL] × respiratory rate [1/min.]. The respiratory minute volume [mL] is also the sum of the alveolar ventilation [mL] + the dead space ventilation [mL]). In high-concentration reservoir masks only an O₂ flow that exceeds the minute ventilation guarantees a sufficient air supply. Consequently, the reservoir bag of the non-rebreathing mask must not be allowed to deflate by more than one third during inspiration. Our patient's respiratory dead space was 100 mL (2 mL/kg body weight), and the dead space of the mask was 50 mL. The increased dead space (150 versus 100 mL) was relevant for gas exchange; the alveolar ventilation decreased from 3 L/min. to 1.5 L/min., which substantially compromised gas exchange. Furthermore, due to insufficient washout by the low O₂ flow

to the face mask [8], our patient rebreathed a toxic amount of CO₂. Our patient was unable to increase his low tidal volume, and an increase in the respiratory rate was insufficient, given the considerable ventilatory dead space.

Jensen *et al.* examined the effect of different oxygen flow rates on ventilation parameters and gas exchange in ten healthy volunteers wearing a simple oronasal face mask (Hudson[®]) without a reservoir bag. Lowering the oxygen supply from 5 to 0 L/min. led to an increase in minute ventilation from 4.8 to 7.5 L/min. due to an increase in tidal volume from 380 to 540 mL with almost no change in respiratory rate. The P_aO₂ decreased from 38 to 13 kPa, the S_aO₂ from 100% to 96%, while the P_aCO₂ remained unchanged. The authors inferred from their results that CO₂ retention will occur in patients with COPD wearing a simple oronasal face mask with an O₂ supply set to < 5L/minute, because these patients cannot increase their tidal volumes [8].

In severely hypoxemic patients with COPD, O₂ may be delivered using a non-rebreathing mask with a target O₂ flow rate of 10–15 L/min. Arterial blood gases must be analyzed regularly. Approximately 13% of patients with COPD admitted with an exacerbation of their disease will develop CO₂ retention during controlled O₂ therapy [2]. In this case, it is important not to stop the O₂ flow completely because of the risk of rebound hypoxemia [9]. For a target S_aO₂ of 88–92%, an O₂ supply via nasal cannula with an O₂ flow rate of 1–2 L/minute is usually sufficient [5]. If not, either noninvasive or mechanical ventilation must be considered [6]. Paramedics may safely use low-inspired O₂ flow (≤4 L/min.) by nasal cannula initially in patients with COPD exacerbations [10].

Conclusions

Non-rebreathing masks with O₂ reservoir bags must be fed with an O₂ flow exceeding the patient's minute ventilation (>6–10 L/min.). If not, the amount of oxygen



Fig. 2 Photograph made during bronchoscopy on day 10. A pea is visible in the bronchus to the right laterobasal pulmonary segment

delivered will be too small to effectively increase the arterial O₂ saturation. Moreover, the risk of CO₂ re-breathing dramatically increases if the flow of oxygen to a non-rebreathing mask is lower than the minute ventilation, especially in patients with COPD and low tidal volumes. As a consequence, CO₂ narcosis may develop. Non-rebreathing masks (with O₂ reservoir bags) must be used cautiously by experienced medical staff and correctly with an appropriately high O₂ flow of 10–15 L/min. [5, 11]. Nevertheless, arterial blood gases must be analyzed regularly for early detection of a rise in P_aCO₂ in patients with COPD and a hypoxic ventilatory drive. These patients are more safely managed using a nasal cannula with an O₂ flow of 1–2L/min. or a simple face mask with an O₂ flow of 5L/min. [5].

Abbreviations

BMI: Body mass index; CO₂: Carbon dioxide; COPD: Chronic obstructive pulmonary disease; FEV₁: Forced expiratory volume in 1 second; GOLD: Global Initiative for Chronic Obstructive Lung Disease; O₂: Oxygen; P_aCO₂: Partial pressure of carbon dioxide in arterial blood; P_aO₂: Partial pressure of oxygen in arterial blood; S_aO₂: Percentage of oxygen saturation of arterial blood; S_pO₂: Percentage of oxygen saturation of arterial blood, measured by pulse oximetry

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Availability of data and materials

Not applicable.

Authors' contributions

TH drafted the manuscript and created the figures. EA performed the bronchoscopy and had an important role in writing and reviewing the manuscript. T Hegi was in charge of the patient during his stay in the intensive care unit. He had a role in writing and reviewing the manuscript. AR critically reviewed the manuscript. MS conceived of the case report, and assisted in drafting the manuscript. All authors read and approved the final manuscript.

Authors' information

MS is President of the Verein Komplikationenliste. Under the auspices of the Swiss Society of General Internal Medicine the Verein Komplikationenliste uses a program for the anonymous, systematic and prospective registration of iatrogenic complications of medical interventions in hospitalized patients in Switzerland. The patient reported was part of this registry.

Ethics approval and consent to participate

Involvement of the ethics committee of the Canton of Zurich was not considered necessary, since the writing of a case report was not based on a study protocol, and is not classified as research by the Swiss Federal Act on Research on Human Beings.

Consent for publication

Written informed consent was obtained from the patient's next-of-kin for publication of this case report and all accompanying images. A copy of the written consent is available for review by the editor of this journal.

Competing interests

The authors declare that they have no competing interests.

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