

Full Length Research Paper

Arterial oxygen saturation and heart rate during a meal in chronic obstructive pulmonary disease

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Patients with chronic pulmonary diseases may have difficulties with breathing during meals. Our objective in this investigation was to evaluate oxygen saturation and heart rate during a meal in patients with chronic obstructive pulmonary disease. We evaluated 16 patients with a diagnosis of chronic obstructive pulmonary disease in a stable phase of the disease and 15 healthy volunteers. The oxygen saturation and heart rate were measured by pulse oximetry for 10 minutes before the ingestion of a solid meal with 1800 calories, during the ingestion, and 10 minutes after the ingestion. Mean oxygen saturation was always lower in patients than in the normal volunteers. Healthy volunteers had no significant change in mean oxygen saturation during the meal, but oxygen saturation decreased after the meal ($p < 0.001$). In patients, mean oxygen saturation decreased during and after the meal compared to the results before the meal ($p < 0.01$). Heart rate was always higher in patients compared with volunteers. In both groups, heart rate was higher during and after the meal compared with the values before the meal ($p < 0.01$), with the same increase in patients and volunteers. We conclude that eat causes a decrease in oxygen saturation in patients with chronic obstructive pulmonary disease.

Key words: Chronic obstructive pulmonary disease, oxygen saturation, heart rate, swallowing, digestion, respiration

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is an important cause of morbidity and mortality throughout the world. It is a clinical syndrome characterized by chronic obstruction of air flow which is not fully reversible, varying little over time and progressing slowly. COPD is associated with an abnormal inflammatory response of the lungs to the inhalation of environmental particles or harmful gases, in particular cigarette smoke (GOLD, 2008; Paggiaro and Vagaggini, 2003). The term obstructive refers to limited expiratory flow, which is

compensated for by increased work of inspiratory muscles (Pride, 1995). The patients present with hypoxemia which may be related to disorders of the ventilation-perfusion ratio, to reduced ability to diffuse, to an increase in dead space, and to fatigue of the respiratory muscles (Gunen and Kosar, 2001). Some studies have shown that, when eating, patients with COPD have a small increase (Colodny, 2001) or a reduction in arterial oxygen saturation levels (SaO_2) (Schols et al., 1991; Brown et al., 1983; Schenkel et al., 1996). The reduction was less than 1 percent in normoxemic patients and of 3.2 ± 0.7 percent in hypoxemic (Schols et al., 1991). The decrease in SaO_2 during a meal ingestion occur mainly in patients already hypoxemic at the rest (Schenkel et al., 1996). There are few references in the literature to the occurrence of changes in heart rate (HR) in these patients during a meal and to their relation to SaO_2 . It is suggested that the HR change during a meal is related to the decrease in

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Abbreviations

COPD, Chronic obstructive pulmonary disease; SaO_2 , Arterial oxygen saturation; HR, Heart Rate

SaO₂ (Schols et al., 1991). The objective of the present study was to assess the changes in blood SaO₂ and in HR during a solid meal ingestion in patients with COPD. The possible decrease in SaO₂ and the increase in HR may cause discomfort during the meal ingestion and the impossibility to ingest the desirable amount of food. Our hypothesis was that patients with COPD may experience a decrease in SaO₂ associated with an increase in HR during a meal.

MATERIALS AND METHODS

We studied 16 patients with COPD and 15 asymptomatic volunteers (controls). Control subjects, 3 women (20%) and 12 men (80%) aged 57 to 73 years (mean: 65 years), were recruited from the community. The results of their pulmonary function tests were normal. All were healthy individuals who had never smoked.

COPD patients were recruited at the outpatient clinics of the Section of Pulmonology, University Hospital of Ribeirão Preto. COPD was diagnosed clinically and confirmed by spirometry according to Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria (GOLD, 2008). They were volunteers for the investigation and selected consecutively if they were able to participate. All subjects were in a stable phase of the disease, were able to have a meal without assistance, had an independent life and reported a relevant past history of smoking, although they had abandoned the habit for at least 6 months. Exclusion criteria for both groups were evidence of other associated pulmonary diseases, such as bronchiectasis and sequels of tuberculosis, and other significant diseases such as severe cardiopathies, cancer, diabetes, severe arterial hypertension, and stroke.

Sixteen patients aged 56 to 77 years (mean: 68 years) were selected, one woman (6%) and 15 men (94%). Two patients had moderate COPD and 14 exhibited severe disease (GOLD, 2008). All COPD patients reported fatigue when walking, five reported dyspnea, and four reported cough related to the act of eating.

Spirometry and gasometry tests used for diagnosis were performed in ambient air within 3 months before the date of evaluation of SaO₂ during a meal. Gasometries were performed only in COPD patients.

Spirometry was performed with a Pulmonet III spirometer (Sensormedics, Anaheim, CA, USA). During the exam, the subjects remained in the sitting position, coupled to the mouthpiece of the instrument and wearing a nose pinch to prevent air escape.

Forced volume capacity (FVC), forced expiratory volume in 1 second (FEV₁), and the FEV₁/FCV ratio were determined. Using the same instrument and the technique of helium dilution, residual volume was calculated and used to determine total pulmonary

capacity. The spirometric results were expressed according to the percentage estimated for age, height and gender (Pereira et al., 1991).

Arterial gasometry was determined using blood samples obtained by puncture of the radial artery of one of the upper limbs. Heparinized blood samples were promptly analyzed with a Corning 178 gas analyzer.

On the day of evaluation the subjects consumed a meal at 12:30 PM after a 30 minute rest and a 3 hour fast. The subjects remained in the sitting position, keeping their trunk erect and their head straight, with the feet resting on the floor and without help, while being monitored with a pulse oximeter. The subjects received a standardized solid meal of 1800 calories [carbohydrate: 259 g (57%), protein: 70 g (15%), lipid: 56 g (28%)], prepared by the Nutrition and Diet Division of the University Hospital of Ribeirão Preto. The food was standardized in terms of nutritional value and consistency and included rice, beans, meat, legumes, vegetables, and 200 mL of water.

The following items were observed during the meal:

- a) meal time: period of time (in minutes) needed by the individuals to complete the meal;
- b) complete or incomplete ingestion
- c) presence or absence of cough before, during and after the meal.
- d) degree of dyspnea before, during, and immediately after the meal, estimated by means of a numerical scale from 0 to 10 (0 indicating absence of shortness of breath and 10 indicating a sensation of intense shortness of breath).

Transcutaneous measurement of blood SaO₂ and of HR was performed using a pulse oximeter (Oxpleth DX2405, Dixtal Biomédica, Manaus AM, Brazil) placed on the index finger of the subject on the side contralateral to the hand used to eat. SaO₂ and HR were continuously recorded transcutaneously at three different times: (a) for 10 minutes before the meal, (b) during the meal, whose duration differed from an individual to another, and (c) for 10 minutes after the end of the meal. The result for each individual was the mean of the results recorded every minute during the periods before, during and after the meal.

The investigation was approved by the Human Research Committee of the University Hospital of Ribeirão Preto and written informed consent was given by all subjects.

The statistical analysis was performed at the Center of Quantitative Methods (CEMEQ) of the Faculty of Medicine of Ribeirão Preto, USP, using a fixed, random and mixed effects model in which the responses of the same individual are grouped and the assumption of independence between observations in the same group is not adequate (Schall, 1991). Fixed effects were time (before, during and after the meal) and group (patients and controls), with the individual being the random effect. In order to use this model, its residues must have normal

Table 1. Clinical features and respiratory parameters of patients with COPD (n = 16) and controls (n = 15).

	CONTROLS		COPD	
	Range	Mean (SD)	Range	Mean (SD)
Age (years)	57-73	65.3 (7.4)	56-77	68.4 (6.2)
Weight (kg)	62-85	76.2 (8.2)	53-93	71.4 (11.3)
Height(cm)	152-180	166.3 (8.9)	151-180	164.1 (7.9)
Body mass index (kg/m ²)	23.0-34.6	27.7 (3.0)	20.7-37.5	26.8 (5.0)
Meal duration (min)	5-19	10.9 (4.1)	3-15	9.8 (3.5)
Dyspnea score				
Before the meal	0-0	0.0 (0.0)	0-7	2.6 (2.5)
During the meal	0-0	0.0 (0.0)	0-8	3.5 (3.0)
After the meal	0-0	0.0 (0.0)	0-9	4.0 (2.7)
FEV ₁ (%)	77.9-126.9	98.5 (12.7)	24.6-55.2	40.2 (8.1)
FVC (%)	74.9-129.0	98.7 (12.4)	21.6-101.2	69.5 (19.5)
FEV1/FVC	70.0-90.5	76.9 (5.8)	29.3-62.8	43.1 (10.1)
pH	-	-	7.39-7.50	7.43 (0.03)
PaO ₂ (mmHg)	-	-	64.0-84.7	71.1 (7.8)
PaCO ₂ (mmHg)	-	-	33.8-45.1	38.1 (2.9)

FEV₁ - forced expiratory volume in 1 second

FVC - forced volume capacity

PaO₂ - arterial oxygen pressure

PaCO₂ - arterial CO₂ pressure

distribution with a mean of zero and constant variance. To satisfy this assumption, transformation of the response variable were necessary. The model was adjusted using the PROC MIXED procedure of the SAS software, version 9 (Littell et al., 1996). The results are shown as mean and standard deviation.

RESULTS

The age, weight, height and body mass index (BMI) of patients with COPD and controls were similar ($p > 0.05$, Table 1). Obesity (BMI > 30 kg/m²) was seen in three subjects of the control group (20%) and in 4 subjects with COPD (25%). There was no subject with BMI < 20 kg/m². Although the meal duration was the same in both groups, 11 (73.3%) of the healthy subjects ingested the entire meal volume and 4 (26.7%) were unable to eat half of the meal volume, compared with only 2 (12.5%) of the subjects with COPD who ingested the entire volume. Nine patients (56.2%) were unable to eat half of the meal and 5 (31.3%) ingested two thirds of the meal. In patients, dyspnea intensity increased from before the meal to during and after the meal. The results of the pulmonary function tests and blood gases obtained are shown in Table 1. No subjects in either group coughed before, during or after meal ingestion.

Mean SaO₂ values were lower in the COPD group compared to controls before (controls: $96.2 \pm 1.1\%$, COPD: $95.3 \pm 1.2\%$, $p = 0.02$), during (controls: $96.1 \pm 1.3\%$,

COPD: $94.9 \pm 1.6\%$, $p < 0.01$), and after (controls: $95.9 \pm 1.4\%$, COPD: $95.0 \pm 1.5\%$, $p = 0.02$) the meal. In addition, in the COPD group the mean SaO₂ values during and after the meal were lower than those obtained before the meal. In the control group, mean SaO₂ was similar before and during the meal, and was reduced after the meal. SaO₂ showed reductions in most patients with COPD (68.9% of subjects during the meal and 62.5% after the meal) and controls (73.3% of subjects during the meal and 73.3% after the meal). Among the patients with COPD, the reduction of SaO₂ between the time periods analyzed did not exceed, on average, 1%, but this reduction, although small, was significant and more intense than that observed in the controls (Figure 1, $p = 0.04$).

At all three time points studied, before (controls: 65.8 ± 9.8 beats/minute- bpm, COPD: 77.4 ± 10.4 bpm), during (controls: 76.3 ± 9.9 bpm, COPD: 86.9 ± 8.1 bpm) and after (controls: 70.7 ± 10.8 bpm, COPD: 80.5 ± 11.3 bpm) the meal, HR remained higher in COPD patients compared to the controls ($p < 0.01$). Both groups showed an increase in HR during and after the meal compared to the period before it (Figure 2), with the increases being more intense during than after the meal. The increases in HR were not related to the change in SaO₂. Among the subjects exhibiting falls in SaO₂ during the meals, the mean SaO₂ decrease was more intense in COPD patients ($n = 11$; $-1.0 \pm 0.9\%$) than in controls ($n = 11$; $-0.3 \pm 0.2\%$, $p = 0.04$). HR showed similar increases in these subsets of patients (9.5 ± 4.7 bpm) and controls

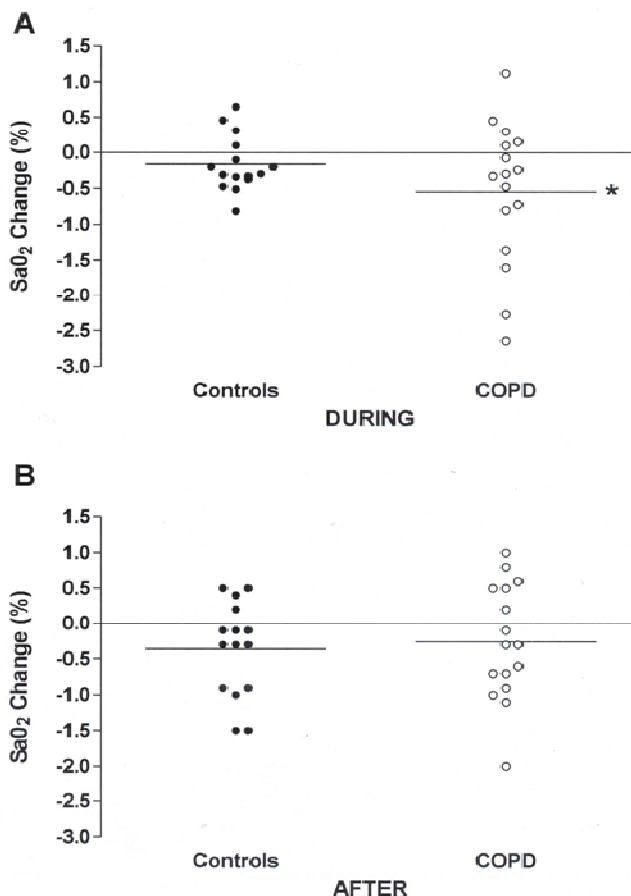


Figure 1. Change in arterial oxygen saturation (SaO₂) (%) during (A) and after (B) a meal, compared with the values before the meal, in patients with COPD and controls. The horizontal bar represents the mean. *p = 0.04 vs controls

(10.1±2.4 bpm). In five COPD patients whose SaO₂ increased (0.4±0.4%), HR increased 8.7±5.1 bpm, whereas in the controls (n=4) whose SaO₂ increased (0.4±0.2%), HR increased 9.6±2.1 bpm. The COPD severity was not a determinant of the change in SaO₂ or HR.

DISCUSSION

In the present investigation, a pulse oximeter was used to assess the effect of a meal on blood SaO₂ levels and on HR in COPD patients and in normal individuals. This method has been used by investigators to identify swallowing and aspiration (GOLD, 2008; Zaid et al., 1995) and can be considered as an alternative instrumental exam to the videofluoroscopic and endoscopic study of swallowing, providing objective values (Colodny, 2000; Sellars et al., 1998). It has the advantages of noninvasiveness, easy operation, quick response time and accuracy of data, but may be influenced by the circulatory status of the subjects, temperature,

peripheral vascular status, and movement (Colodny, 2001). The mean results obtained indicated that the difference in SaO₂ between the time before the meal and the time during the meal, although small, was significant for the patient group but not for the control group, as also observed in previous studies (Sellars et al., 1998; Chan and Lo, 2009). The act of eating is one of the activities of daily life that provokes a fall in SaO₂ in individuals with moderate to severe COPD who have normal basal values under resting conditions (Schenkel et al., 1996). The mean percent fall in SaO₂ in the COPD group did not exceed 1% at any of the times studied and the basal values of these patients did not characterize substantial oxygen desaturation. A mean SaO₂ decay of less than 1% was observed in normoxemic patients with COPD, while the mean SaO₂ desaturation in the blood of hypoxemic COPD patients was more than 1% (Colodny, 2001). Other studies have shown decay values of more than 1% in patients with lower basal values (Brown et al., 1983; Schenkel et al., 1996).

Factors such as swallowing, coughing and posture may

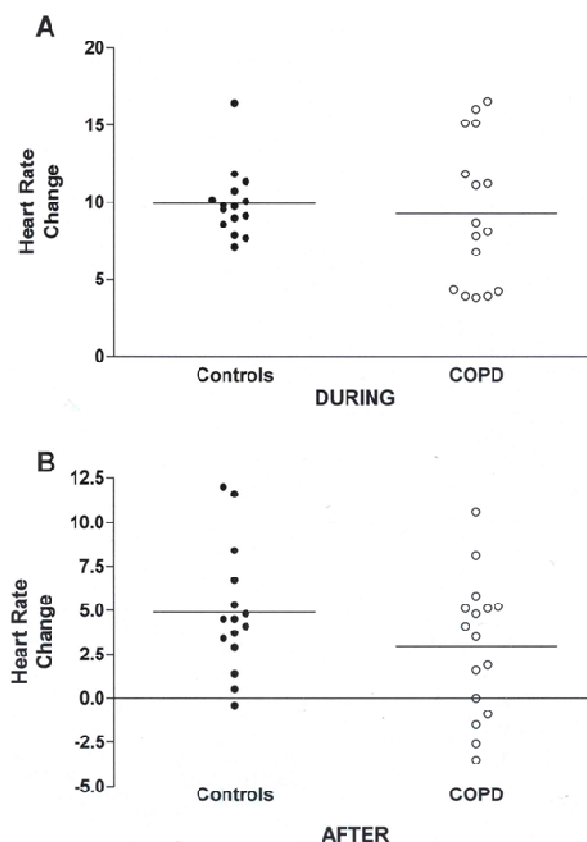


Figure 2. Heart rate change (beats/minute) during (A) and after (B) a meal, compared with the values before the meal, in patients with COPD and controls. The horizontal bar represents the mean.

influence SaO_2 levels (Leder, 2000). Studies have shown that the longer period of apnea of COPD patients during swallowing may render them more susceptible to the swallowing factors that disturb respiratory function compared to normal subjects (Shaker et al., 1992; Smith et al., 1989; Higo et al., 2003). Another study suggested that factors that may contribute to a reduction of SaO_2 are the various moments of brief apnea during swallowing (Teramoto et al., 1996). A study conducted on young individuals revealed that breathing is affected by chewing and by the initial consistency of solid food (Palmer and Hiimeae, 2003). The present results indicate that COPD itself may be associated with the occurrence of oxygen desaturation during a meal, since the two groups studied did not present signs suggestive of the occurrence of penetration and/or aspiration during the evaluation of the meal. In addition, no patient coughed at any time during the meal. This suggests that the presence of COPD was an important factor for the occurrence of the difference in saturation observed during the meal.

In the COPD group, the dyspnea scored on a subjective scale showed higher values during the meal

compared to the values before the meal, being reported by five individuals (31%) with severe disease. Although the cause of dyspnea is multifactorial (Demir et al., 2003), the present study permits us to assume that the severity of the disease may have been one of the causes of the sensation of dyspnea due to the act of eating in these COPD patients. This explanation is based on a previous study that reported that the act of eating causes an increase in dyspnea in subjects with severe COPD (Wolkove et al., 1998). During eating, the breathing pattern is more irregular in terms of volume, flow and time, and the minute volume is reached at the lowest energy expenditure. However, in COPD patients this deviation in the breathing pattern, which is associated with the increased work involved in breathing in order to maintain a normal ventilatory minute volume, may cause a sensation of dyspnea while eating (Smith et al., 1989). The movements that occur during the act of eating may also explain the presence of dyspnea, since the muscles of the scapular belt are activated (Velloso et al., 2003). Individuals with COPD and a tracheostomy who depend on mechanical ventilation, i.e., with more severe respirat-

ory alteration, present a rapid occurrence of fatigue while eating, the same occurring during other activities of daily life such as bathing and getting dressed (Coelho, 1987).

The presence of dyspnea in severe COPD may be related to the digestive process itself. When a volume is introduced into the stomach, its accommodation involves an increase in the dimension of the rib cage and of the abdomen, with a reduction of lung size. This would scarcely affect breathing in normal subjects, but would involve respiratory effort in subjects with severe COPD during a meal as a consequence of the reduction of residual functional capacity (Gilrov et al., 1985). Probably, for most of the COPD patients who did not report dyspnea while eating, the effort was not strong enough to cause this sensation.

The increased dyspnea score reported by seven COPD patients (48%) from before to after the meal could explain the smaller amount of food ingested by this group. However, it has been demonstrated that this increase is not a condition responsible for reduced food intake among subjects with severe COPD during the stable phase of the disease (Gray-Donald et al., 1998). The partial ingestion of food may be better explained by another study which found that early satiety in COPD may be related to pulmonary hyperinflation and to the position of the diaphragm (Akrabawi et al., 1996). HR increased in both COPD patients and control subjects during and after the meal. This increase in HR should be considered as a normal physiological response (Colodny, 2000). HR is higher in COPD possibly in response to changes in arterial blood gases, respiratory mechanics, pulmonary hypertension, systemic inflammation or effect of drugs used to treat the disease. In this study the meal-related increase in HR was not more prominent in COPD patients than in controls, or in subjects who exhibited reductions of SaO_2 compared to those who showed increases in saturation.

We conclude that SaO_2 is reduced during a meal in patients with moderate to severe COPD but the HR response to meal ingestion is similar as that of normal volunteers.

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