Transcutaneous Oxygen Saturation and Carbon Dioxide Tension during Meals in Patients with Chronic Obstructive Pulmonary Disease*

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The effect on transcutaneous SaO2 and transcutaneous carbon dioxide tension (PtCO2) of eating was assessed in 44 patients with severe COPD (FEV1<50 percent) The SaO2, PtCO2, and heart rate (HR) were measured every minute before, during, and until 30 minutes after a standardized meal (445 kcal) was consumed. All patients were measured twice on the same day, while eating a meal with high (80 percent) and low (25 percent) carbohydrate content, respectively. The mean meal desaturation (ΔSaO2) was less than 1 percent in normoxemic patients but was −3.2±0.7 percent (p<0.001) in hypoxemic (PaO2<7.3 kPa) patients. Significant differences between hypoxemic patients with a ΔSaO2 greater than 4 percent and less than or equal to 4 percent, respectively, were found in FEV1 (16±3 percent and 29±5 percent; p<0.001), respiratory muscle strength (3.9±1.2 kPa and 5.9±1.2 kPa; p<0.01), HR (112±12 beats per minute and 90±18 beats per minute; p<0.001), body weight (54.9±7.5 kg and 74.7±10.4 kg; p<0.001), and fat-free mass (42.0±6.6 kg and 52.6±5.8 kg; p<0.005) but not in baseline SaO2 and PtCO2. The decrease in SaO2 and the increase in HR were less during the carbohydrate-rich meal. No significant fluctuations in PtCO2 were found after either meal. Meal-related oxygen desaturation cannot explain weight loss in normoxemic patients with COPD but may contribute to a limited dietary intake in a subgroup of hypoxemic patients exhibiting marked oxygen desaturation during meals. A single carbohydrate-rich meal does not have an immediate impact on PtCO2 in stable COPD.

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A negative energy balance commonly occurs in the course of COPD1 and can ultimately lead to cachexia. At present, the cause of progressive weight loss in patients with COPD is not well understood. Several potential mechanisms have been hypothesized,2 which can be divided into two groups: those involving energy input; and those involving energy output. In patients with COPD, an elevated resting metabolic rate relative to predicted needs has been reported by multiple investigators.3-4 Recent data from our group indicated that failure of an adaptive response to undernutrition and an inadequate dietary intake for energy expenditure are two contributing factors to weight loss in COPD. Inadequate dietary intake for energy expenditure was most pronounced in patients suffering from chronic hypoxemia.5

Several studies have measured decreases in SaO2 or in PaO2 during meals in patients with COPD;6,10 however, no systematic study regarding the relative contribution of meal-related oxygen desaturation to a limited dietary intake has been reported.

The adverse effects of excessive carbohydrate calories in the intensive care unit have received considerable attention.11-13 Carbohydrate oxidation yields more CO2 than fat oxidation. Carbohydrate oxidative capacity is limited. Too much carbohydrate (>6 g/kg/24 h) leads to lipogenesis, which also increases CO2 production, resulting in an RQ above 1.0. If the patient is incapable of responding to the increased CO2 production, respiratory failure may result.18-13 It has therefore been suggested to shift from predominantly carbohydrate calories to high-fat diets, resulting in a lower RQ and less CO2 production.13,14 Little is known about the effects of excessive carbohydrate intake in ambulatory patients with COPD.

In this study we addressed two factors related to energy input in patients with severe COPD in a stable clinical condition: (1) the effect of eating on SaO2; and (2) the effect of the carbohydrate content of a meal on carbon dioxide tension.

Materials and Methods

Patients

Forty-four patients with severe airflow obstruction (FEV1 <50 percent of predicted) were admitted to a pulmonary rehabilitation center for physical training and participated in the study. All patients were in stable clinical condition and were not suffering from a lower respiratory tract infection.

Methods

Body height was measured standing barefoot and was determined to the nearest 0.5 cm. Body weight was measured with a beam scale without shoes in light clothing to the nearest 0.1 kg (SECA).

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Table 1 — Caloric Content and Nutrient Composition of Experimental Meals

<table>
<thead>
<tr>
<th>Time</th>
<th>Meal 1*</th>
<th>Meal 2†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>3 PM</td>
<td>5:30 PM</td>
</tr>
<tr>
<td>Energy, kcal</td>
<td>445</td>
<td>445</td>
</tr>
<tr>
<td>Fat, percent of energy</td>
<td>44.0</td>
<td>9.6</td>
</tr>
<tr>
<td>Carbohydrates, percent of energy</td>
<td>25.4</td>
<td>79.6</td>
</tr>
<tr>
<td>Protein, percent of energy</td>
<td>27.6</td>
<td>10.8</td>
</tr>
</tbody>
</table>

*Meal 1: potatoes, cauliflower, meatballs, and gravy.
†Meal 2: three slices of bread, one slice of lean meat, marmalade, banana, and tea.

Body resistance (BIA 101; RJL Systems) was measured as described by Lukaski et al. Fat-free mass (FFM) was calculated from height, weight, and resistance using a patient-specific regression equation.

Inspired vital capacity (IVC) and FEV, were measured with a wet spirometer; the highest value from at least three spirometric measurements was used. The FEV, and IVC were expressed as a percentage of the reference values. Blood was drawn in the end of the morning by puncture of the brachial artery at rest while breathing room air. The PaO, and PaCO, were analyzed on a blood gas analyzer (Radiometer ABL 330). Inspiratory (Pimax) and expiratory (Pemax) muscle strength were assessed by determining maximal inspiratory and expiratory mouth pressures according to the technique described by Black and Hyatt. The best of three determinations was used in subsequent calculations. The SaO, was measured with a pulse oximeter. The electrode was placed around the left little finger and sealed with tape to prevent false light information from outside to reach the sensor. The CO2 sensor was attached to the patient’s skin, at the right underarm, with a double-sided adhesive ring. The sensor was heated to 43°C to increase perfusion at the skin surface. A two-point calibration for CO2 (5 percent/10 percent CO2 in N2) was performed before every measurement. Measurements were done with a combined SaO2 and PtCO2 monitor (Fastrac; Sensor Medics), which is attached to a printer that automatically registers every strength the lowest measured SaO2 value and the highest measured PtCO2 value. Heart rate (HR) was registered with an ECG monitor (Servomed SMS 182).

Design of Study

Three groups of patients were measured: (1) the control group (group 1) consisted of 12 patients with a resting PaO2 of 7.3 kPa or more and a stable body weight; (2) group 2 consisted of 12 patients with a resting PaO2 of 7.3 kPa or more but greater than 10 percent weight loss in the previous year; and (3) group 3 consisted of 20 patients with a resting PaO2 less than 7.3 kPa when breathing room air, 12 of whom used supplemental oxygen via nasal cannula when eating. The SaO2, PtCO2, and HR were measured before, during, and until 30 minutes after a standardized meal.

To study the immediate effect of a carbohydrate load on PtCO2, the patients were measured on the same day in the early evening while eating an isocaloric but carbohydrate-rich meal. The caloric content and nutrient composition of the meals are given in Table 1. The patients were encouraged to eat the entire meal. The exact duration of the meal was recorded. When a patient could not complete the meal, the leftovers were weighed exactly using a digital scale (Soehnle). All patients tolerated the electrodes and the finger oximeter well and were able to eat in their usual manner. Immediately after completion of the meal, the degree of dyspnea was rated (1 = no; 2 = moderate; 3 = severe).

Medication was given 1 h before the beginning of the meal. Maintenance medication included theophylline, β1-adrenergic agonists, inhaled or oral corticosteroids, and diuretics in most patients. Thirty minutes before the meal, the patients were asked to sit relaxed at the dinner table, at which time baseline values of SaO2, PtCO2, and HR were recorded. During the meal, minute values were recorded continuously. Registration proceeded until 30 minutes after completion of the meal. The monitoring was completed in all patients, and there were no complications or untoward events during the study period.

Statistical Analyses

The mean baseline SaO2 (base SaO2) was the average saturation observed over a 10 min baseline period prior to eating. Meal saturation (meal SaO2) was the average saturation observed throughout the meal. Mean meal desaturation (ΔSaO2) was defined as: (base SaO2 - meal SaO2). This index was used to ascertain the time-weighted overall effect of eating on saturation. Mean after-meal saturation was calculated over a 30-minute interval. Similarly, baseline, meal and after-meal values of PtCO2 and HR were defined.

Differences between two groups of patients were tested with the Mann-Whitney U-test. Differences within the individual between the two meals were tested with the Wilcoxon signed-rank test.

Table 2 — Physical and Pulmonary Characteristics of Study Group*

<table>
<thead>
<tr>
<th>Data</th>
<th>Group 1 (n = 12)</th>
<th>Group 2 (n = 12)</th>
<th>Group 3a (n = 8)</th>
<th>Group 3b (n = 12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height, cm</td>
<td>168.8 ± 10.4</td>
<td>174.7 ± 8.4</td>
<td>171.2 ± 8.4</td>
<td>174.0 ± 9.7</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>67.7 ± 9.4</td>
<td>63.0 ± 9.0</td>
<td>54.9 ± 7.5†</td>
<td>74.7 ± 10.4</td>
</tr>
<tr>
<td>FFM, kg</td>
<td>49.0 ± 7.9</td>
<td>47.4 ± 7.2</td>
<td>42.0 ± 6.6‡</td>
<td>52.6 ± 5.8‡</td>
</tr>
<tr>
<td>No. with weight loss</td>
<td>0/12</td>
<td>12/12</td>
<td>5/8</td>
<td>3/12</td>
</tr>
<tr>
<td>IVC, percent</td>
<td>81 ± 15¶¶</td>
<td>62 ± 17</td>
<td>49 ± 12</td>
<td>61 ± 19</td>
</tr>
<tr>
<td>FEV1%, percent</td>
<td>37 ± 6¶</td>
<td>29 ± 10</td>
<td>16 ± 3†</td>
<td>29 ± 8</td>
</tr>
<tr>
<td>Pimax, kPa</td>
<td>7.0 ± 2.2∥∥</td>
<td>4.8 ± 1.4</td>
<td>3.9 ± 1.2†</td>
<td>5.9 ± 1.2∥</td>
</tr>
<tr>
<td>Pemax, kPa</td>
<td>10.2 ± 5.0</td>
<td>7.0 ± 3.0</td>
<td>5.1 ± 1.2†</td>
<td>9.8 ± 2.7</td>
</tr>
<tr>
<td>SaO2, percent</td>
<td>92 ± 11</td>
<td>92 ± 1</td>
<td>87 ± 3</td>
<td>88 ± 4</td>
</tr>
<tr>
<td>PtCO2, kPa</td>
<td>4.5 ± 0.7¶</td>
<td>5.2 ± 0.7</td>
<td>6.3 ± 1.1</td>
<td>5.9 ± 1.0</td>
</tr>
<tr>
<td>HR, beats per min</td>
<td>90 ± 16</td>
<td>88 ± 13</td>
<td>112 ± 12‡</td>
<td>90 ± 18</td>
</tr>
<tr>
<td>No. with supplemental oxygen</td>
<td>. . .</td>
<td>. . .</td>
<td>6/8</td>
<td>6/12</td>
</tr>
</tbody>
</table>

*Table values are means ± SD unless otherwise stated.
†p < 0.001 compared to adjacent group.
‡p < 0.01 compared to adjacent group.
∥p < 0.05 compared to adjacent group.
¶p < 0.001 for group 1 vs group 3.
•p < 0.05 for group 1 vs group 3.
Significance was determined at the 5 percent level. Except in the figures, all results are expressed as means ± SD.

RESULTS

A description of the study group is given in Table 2. The weight-stable and weight-losing groups with a resting PaO₂ of 7.3 kPa or more were significantly different in age and pulmonary function. Even more compromised values for IVC, FEV₁, and Pimax were found in the hypoxemic patients. Mean values of baseline SaO₂, mean SaO₂, and after-meal SaO₂ of meal 1 for the three groups are graphically displayed in Figure 1. Baseline SaO₂ was significantly lower in the hypoxemic relative to the normoxemic patients (p<0.001). The SaO₂ was slightly decreased in weight-stable (-0.8 ± 0.2 percent; p<0.05) and weight-losing (-0.6 ± 0.2 percent; p = 0.05) normoxemic patients. The ΔSaO₂ in the hypoxemic patients was more pronounced (-3.2 ± 0.7 percent; p<0.001) than in the normoxemic patients. The HR increased significantly in all groups during the meal and remained elevated within 30 min after completion of the meal. The average duration of meal 1 amounted to 12.2 ± 2.1 min and was not different among the three groups.

Because a high within-group coefficient of variation of 23 percent was found in the hypoxemic patients, they were divided into two groups according to the criterion used by Brown and colleagues² in an earlier study. Group 3a consisted of eight patients exhibiting a significant drop in SaO₂ during the meal amounting to 4 percent or more; group 3b consisted of 12 patients with a ΔSaO₂ of less than 4 percent (Table 2). The groups did not differ in baseline SaO₂ or baseline PtCO₂, but group 3a had a more compromised pulmonary function, as reflected in significantly lower values for FEV₁ (p<0.001), Pimax (p<0.01), and PEFmax (p<0.001) and a higher baseline HR (p<0.001). Body weight (p<0.001) and FFM were significantly lower (p<0.01) in group 3a, whereas in group 3b, body fat even tended to be relatively increased in relation to FFM.

Five out of eight patients in group 3a experienced moderate to severe dyspnea while eating, relative to 2 of 12 patients in group 3b. To exclude a selection bias of patients using supplemental oxygen, an additional analysis was done only for the patients receiving oxygen therapy while eating; however, results were not different in this subgroup.

Figure 2 displays in more detail the pattern of desaturation for the subgroup 3a, which exhibited a significant desaturation. The graph illustrates that the pattern of desaturation during and immediately after completion of the meal was comparable in all patients: SaO₂ dropped within 5 min after the patients started their meal and remained at a low level throughout the meal, but was restored within a few minutes after the meal. Even eight patients in group 3a experienced moderate to severe dyspnea while eating, relative to 2 of 12 patients in group 3b. To exclude a selection bias of patients using supplemental oxygen, an additional analysis was done only for the patients receiving oxygen therapy while eating; however, results were not different in this subgroup.

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completion of the carbohydrate-rich meal 2.

**Discussion**

Nutritional management of COPD is difficult and controversial. Several factors have been suggested in the literature that may interfere with dietary intake in COPD, such as gastrointestinal disorders, psychosocial factors, and meal-related oxygen desaturation. In this study, we addressed the immediate effect of eating on SaO₂. Furthermore, we were interested in the effect of the carbohydrate content of a meal on PaCO₂ because it has been suggested to patients with COPD to shift from predominantly carbohydrate calories to high-fat diets.

The PaCO₂ and SaO₂ were measured transcutaneously with a combined SaO₂ and PtCO₂ monitor. Several studies demonstrated that PtCO₂ yields an excellent reflection of PaCO₂ as well as of fluctuations in PaCO₂ if the measurement is preceded by a proper calibration. Pulse oximetry, contrary to arterial blood sampling, is a feasible means for long-term monitoring of oxygenation during daily activities; however, the method presents several limitations. Recent studies found for an SaO₂ greater than 85 percent an absolute inaccuracy of ±2.0 to ±5.0 percent for the cutaneous measurement of SaO₂ when compared with arterial blood gas samples analyzed for SaO₂. Differences were larger for an SaO₂ less than 80 percent. Invalid measurements can also occur in conditions which result in decreased cutaneous blood flow, or elevated bilirubin, methemoglobin, or carboxyhemoglobin levels. In this study, patients were therefore grouped according to PaO₂, whereas pulse oximetry was primarily used to identify trends in oxygenation while eating.

We did find a statistically significant decrease in oxygen saturation in patients with a PaO₂ of 7.3 kPa or more, but a decrease of 1 percent can hardly be considered of clinical relevance. No differences in ΔSaO₂ were found between weight-stable and weight-losing patients, which suggests that weight loss in normoxemic patients with COPD is not initiated by meal-related ΔSaO₂.

A substantial amount of individual variability in desaturation was noticed in the hypoxemic patients. We therefore categorized them into two groups: (1) group 3a had a stable desaturation of more than 4 percent throughout the meal; and (2) group 3b had an overall meal desaturation less than 4 percent. Variability in desaturation could not be explained by differences in baseline SaO₂ or PtCO₂, but body weight, FFM, and respiratory muscle strength were lower in hypoxemic patients exhibiting more than 4

![Figure 2](http://journal.publications.chestnet.org/pdflaccess.ashx?url=/data/journals/chest/21636/)

**Figure 2.** Fluctuations in SaO₂ (mean ± SEM) before, during (10 min), and until 15 min after completion of meal 1 for patients exhibiting >4 percent meal desaturation. *p<0.05.
percent desaturation, compared to hypoxemic patients with less desaturation when eating.

Several mechanisms are proposed to explain desaturation during meals. The pattern of desaturation was uniform. Despite the small number of patients involved, significant decreases in the first minutes after beginning the meal and significant increases immediately after completion of the meal were found. The fact that desaturation occurs so early after the beginning of the meal excludes possible metabolic effects of food absorption and digestion on desaturation. This conclusion was confirmed by Brandstetter et al. who could not establish a clinically significant effect of nasogastric feeding on $\text{PaO}_2$ in a group of patients with COPD who were receiving nasogastric bolus or continuous tube feeding.

A second hypothesis is a decrease in minute ventilation as a result of interrupted breathing while chewing and swallowing; however, a decrease in oxygen saturation was not associated with a concomitant increase in $\text{Paco}_2$.

It has been speculated that alterations in ventilation-perfusion ($\text{V/Q}$) relationships may contribute to a fall in $\text{SaO}_2$ if tidal volume or respiratory frequency were altered. Smith et al. recently studied the coordination of eating, drinking, and breathing in seven healthy adults using noninvasive techniques. No change in tidal volume, inspiratory duration, expiratory duration, or minute ventilation was found between the periods of normal breathing, eating, and drinking; however, breathing became more irregular during eating and drinking.

Mean baseline $\text{SaO}_2$ in hypoxemic patients was substantially lower than in the normoxic group. The higher percentage of oxygen desaturation in hypoxemic patients could therefore be attributed to the sigmoidal shape of the oxygen-dissociation curve; however, differences in the degree of desaturation within group 3 cannot be attributed to the shape of the oxygen-dissociation curve. Baseline $\text{SaO}_2$ was not lower in group 3a.

The HR at rest was significantly higher in group 3a, compared to group 3b. In accordance with findings in normal subjects, a significant increase in HR was found in both groups while eating; however, if group 3a exhibits a limitation in stroke volume secondary to a greater loss in pulmonary capillary bed, then the increased HR may have a limited effect in terms of contribution to the cardiac output; ventricular filling after eating may be further compromised by right ventricular preload reduction due to pooling of blood in the vascular intestinal bed. Careful hemodynamic evaluation of the patients will be necessary in further studies. Of interest is the observation that in the hypoxemic group exhibiting significant desaturation, a more modest decrease in $\text{SaO}_2$ was found during the carbohydrate-rich meal, accompanied by a lower increase in HR. This finding is in favor of a hemodynamic explanation for the differences in oxygen desaturation.

The meals did not differ only in carbohydrate content, but also in eating pattern and in protein content. Meal 2 consisted of sandwiches which the patients had to prepare themselves, whereas meal 1 was a hot meal ready for use. The time in between bites was therefore longer for meal 2, which was also reflected in a longer overall meal duration. In addition, the dyspneic sensation was less during meal 2, which suggests that eating this meal constituted less exertion than eating meal 1.

The thermic response to protein is significantly greater than to isonenergetic amounts of carbohydrate or fat. This difference in thermogenic stimuli of carbohydrate and protein is most pronounced approximately one hour after the meal, but probably cannot explain the observed desaturation immediately after beginning the meal.

Another objective was to see if there is evidence for beneficial effects of high-fat supplements on $\text{PtCO}_2$ for ambulatory patients with stable COPD. We found that a single carbohydrate-rich meal did not influence $\text{PtCO}_2$ during or immediately after the meal. This is probably not surprising, since the concept that increasing fat intake aids in reducing the requirement for CO2 excretion assumed importance in the case of sustained carbohydrate loading with a positive energy balance.

We conclude that meal-related $\Delta \text{SaO}_2$ cannot explain weight loss in normoxemic patients with severe COPD but may contribute to a limited dietary intake in a subgroup of hypoxemic patients exhibiting significant desaturation. Increasing oxygen supply in these patients when eating may be advocated.

REFERENCES

7. Schols AMWJ, Soeters PB, Saris WHM, Wouters EF. Weight...
loss in chronic obstructive pulmonary disease (abstract). Clin Nutr 1989; 8:130
11 Askanazi J, Rosenbaum SH, Hyman AI, Silverberg PA, Milic-Emile J, Kirrały JM. Respiratory changes induced by the large glucose loads of total parenteral nutrition. JAMA 1980; 243:1444-47
29 Grollman A. Physiological variations in the cardiac output of man. Am J Physiol 1929; 89:366-70