

Does Active Eating Influence Dyspnea and Pulmonary Function Tests in COPD?

Çağla Özgören^{1*}, Osman Hacıömeroğlu², Rasmi Muammer³

¹Institute of Health Sciences, Department of Physiotherapy and Rehabilitation, Istanbul Medipol University, Istanbul, Turkey

²Süreyyapaşa Chest Diseases and Thoracic Surgery Education and Research Hospital, Istanbul, Turkey

³Department of Physiotherapy and Rehabilitation, Faculty of Health Science, Yeditepe University, Istanbul, Turkey

ABSTRACT

Active eating is considered an effortful activity for patients with chronic obstructive pulmonary disease (COPD). Aim of study was to determine the effect of active eating on pulmonary functional capacity, dyspnea level and oxygen saturation (SpO₂) in COPD patients. 40 clinically stable COPD patients who were hospitalized in Süreyyapaşa Chest Diseases and Thoracic Surgery Education and Research Hospital were enrolled. All patients were in group D according to 2016 GOLD guide. Demographic data, smoking status, body mass index, medications and additional diseases of patients were noted. The degree of dyspnea was assessed using the Modified BORG scale before and after active eating for all participants and was also measured in obese and non-obese subgroups. Spirometric parameters and SpO₂ were analyzed one hour before and after active eating. In general, there was no statistically significant difference in FVC, FEV₁, FEV₁/FVC and SpO₂ values compared to initial measurements (p>0.05). However, significant difference was observed in BORG scale evaluations for all participants (p<0.05). According to subgroups, there was no significant difference in FEV₁/FVC and SpO₂ results (p<0.05), but difference was found in FEV₁, FVC and BORG values in non-obese group (p<0.05). In obese group, we found no difference in any of evaluated parameters (p <0.05). In conclusion, we observed no change in spirometric parameters and SpO₂ level in total but there was a change in FEV₁, FVC and dypnea level in non-obese group.

Keywords: Chronic obstructive pulmonary disease, Active eating, Spirometry, Pulmonary capacity

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is an important cause of morbidity and mortality worldwide, characterized by chronic inflammation of the respiratory system and chronic non-fully reversible airflow limitation (1). COPD is associated with an abnormal inflammatory response of the lungs to the inhalation of harmful environmental gases and particles, particularly tobacco smoking. Common symptoms of COPD include progressive shortness of breath, expectoration, dyspnea, and fatigue (2). Patients with COPD often experience exercise restriction due to muscle weakness and shortness of breath, resulting in physical inactivity and reduced quality of life. Hypoxemia may be seen in patients due to deterioration in ventilation-perfusion ratio and fatigue of respiratory muscles (3,4).

Categorisation and guiding optimal treatment of COPD patients are based on Global Initiative for Chronic Obstructive Lung Disease (GOLD)

guidelines. Understanding the impact of COPD on the patient is possible by combining the patient's symptomatic assessment using the spirometric classification and the number of exacerbations. A post-bronchodilator spirometric measurement is performed to determine airflow limitation. In addition, dyspnea assessment is performed using the Modified Medical Research Council Dyspnea Scale (mMRC) or symptom assessment is performed using CAT (COPD Assessment Test). Finally, patients are grouped as A, B, C, D based on their exacerbation history and numbers of previous annual hospitalizations (5,6). Dyspnea is defined by the American Thoracic Society (ATS) as “an experience of respiratory discomfort that can vary from patient to patient, consisting of qualitatively different sensations of varying intensity” (7). It is one of the most important symptoms of COPD, which occurs when the respiratory or circulatory systems are insufficient to meet the body's needs (7,8). Activity-related dyspnea is a common problem in

*Corresponding Author: Çağla Özgören, Istanbul Medipol University, Department of Physiotherapy and Rehabilitation, Istanbul, Turkey
E-mail: cagla.ozgoren@medipol.edu.tr, Phone: 0 90 444 85 44, Fax: 0212 521 23 77

ORCID ID: Çağla Özgören: 0000-0002-8698-7672, Osman Hacıömeroğlu: 0000-0001-9273-4930, Rasmi Muammer: 0000-0001-6764-4167

patients with COPD and can cause oxygen desaturation. There are many reasons for the increased level of dyspnea in patients during eating. The first reason is that there is a decrease in the minute ventilation thanks to interruption of breathing during chewing and swallowing. Another reason is that although active eating is considered a sedentary activity that occurs with the activation of chewing, swallowing and arm muscles, it can be counted as a physical activity that may require effort, and cause shortness and irregular patterns of breathing in severe COPD patients. In addition, studies have suggested that changes in tidal volume or respiratory frequency during eating alters ventilation/ perfusion rate that may be important in COPD (9,10).

Variations in body mass index (BMI) among COPD patients may influence underlying pathophysiology of the disease, consequently impacting patient outcomes. In COPD patients, there is direct correlation between elevated BMI values and increasing dyspnea (11). Furthermore, BMI exerts a discernible effect on pulmonary volumes. There is a notable reduction in functional residual capacity (FRC) and expiratory reserve volume (ERV) with increasing BMI, attributable to the modification of chest wall mechanics (12).

Clinically, it is possible to feed the patient in different ways. These are enteral, oral and parenteral nutrition methods (13). If there is no physical obstacle to oral feeding, the patient can eat actively and without assistance. There are limited numbers of studies investigating the relationship between active eating and pulmonary functional capacity in COPD patients. Determining whether or not active eating has an effect on pulmonary capacity is important for correct and effective treatment of COPD patients, especially for the timing of rehabilitation practices. In this research, we examine the hypothesis that pulmonary capacity, oxygen saturation (SpO₂) and dyspnea level decrease after eating.

Materials and Methods

This study was carried out in the Süreyyapaşa Chest Diseases and Thoracic Surgery Education and Research Hospital between January 2016 and July 2016. Forty hospitalized COPD patients classified as class D group according to GOLD were included. Among the participants, 11 were utilizing oxygen supplement, while the remaining 29 were not. All participants were between 50 and

70 years old, clinically stable and were able to eat without any assistance. Subjects were included in the study on a voluntary basis, informed about the purpose of the study, and an informed consent form was signed. Exclusion criteria were pneumonia, emphysema, pacemaker, history of thoracic or cardiac surgery and unstable cardiopulmonary conditions. Patients having additional pneumonia and emphysema were not included. Those cases had been identified mainly through chest X-ray and CT (Computerized Tomography) or HRCT (High Resolution CT) examination when necessary.

All patients included in this study were under optimal medication. Participants were fed with a balanced, standardized solid hospital meal menu consisting of carbohydrates, protein, fat, and vitamins prepared by the nutrition and dietetics department. All participants fasted for 4 hours before eating. Eating time was 12:30 pm and same for all patients. Eating position for the participants was half-sitting on the patient bed with their trunks adequately supported. Participants actively ate their meals orally and without any assistance. Duration of eating was approximately 25-30 minutes. Spirometric measurement was performed with a MIR Spirobank II 910513 device. Forced vital capacity (FVC), forced expired volume in one second (FEV₁), FEV₁/FVC values were recorded. At the same time, the patients' SpO₂ values were taken using a pulse oximeter. Degrees of dyspnea were recorded by using BORG scale between 0-10. On this scale, 0 point indicates no dyspnea, and 10 points indicates severe dyspnea (Figure 1). All evaluations were made one hour before and after active eating for all participants and for non-obese (<30 kg/m²) and obese (≥30 kg/m²) subgroups.

Study Protocol: This study was conducted during April 2016 - May 2016, and it was approved by the Clinical Research Ethics Committee of Marmara University Faculty of Medicine with the protocol number of 09.2016.224.

Sample Size Estimation: The sample size of the study was based on the percentage measurement values related to the methods to be studied in the literature review. It was calculated as 42 participants using the G Power (version 3.1.9.4) program, with an effect size of 0.50, a power of 80%, and a margin of error of 0.05.

Statistical Analysis: Data analysis of the study was performed using "Statistical Package for Social Sciences" SPSS 23.0 (SPSS Inc., Chicago, IL, USA) program. Descriptive statistics, mean ±,

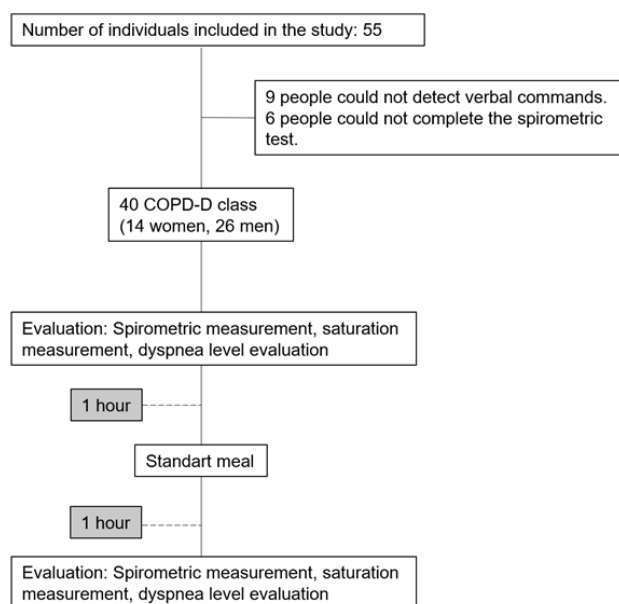


Fig. 1. Study Design

standard deviation ($X \pm SD$) or percentages (%), were gathered. The level of significance was accepted as $p \leq 0,05$. Paired Samples T test was used to compare the values between before and after active eating.

Results

Age, gender, BMI, smoking and O_2 supplement status of the participants are shown in Table 1. Comorbidities of the participants are shown in Table 2.

Comparison of FEV_1 , FVC, FEV_1/FVC , BORG and SpO_2 values before and after active eating are shown in Table 3. While no statistically significant difference was observed in the FEV_1 , FVC, FEV_1/FVC and SpO_2 values ($p > 0.05$), a statistically significant difference was observed in the BORG values ($p < 0.05$).

Comparison of FEV_1 , FVC, FEV_1/FVC , BORG and SpO_2 values before and after active eating according to BMI Subgroups are shown in Table 4. While there was no statistically significant difference in FEV_1/FVC and SpO_2 results ($p < 0.05$), statistically significant difference was found in FEV_1 , FVC and BORG values before and after active eating in the non-obese group ($p < 0.05$). In obese group, we found no significant difference in any of the evaluated parameters ($p < 0.05$).

Discussion

In this study, we found no significant difference in the FEV_1 , FVC, FEV_1/FVC and SpO_2 values but we found decreased dyspnea level after active eating in general population. In view of subgroups, there was no difference in FEV_1/FVC and SpO_2 results, but FEV_1 , FVC and dypnea level were different in non-obese group. In obese group, we found no difference in any of evaluated parameters.

Studies have shown that energy consumption during ventilation, which is 36-76 kcal/day in healthy people may increase to tenfold in patients with COPD (430-720 kcal/day). This is associated with increased respiratory workload, and decreased efficiency of respiratory muscles (13). In addition, daily living activities including eating may worsen the general situation of COPD patients. Eating is particularly an effortful activity for these restricted patients. Also, it can lead to increased dyspnea and inadequate pulmonary capacity due to the weakening of respiratory muscles, increased work of breathing and uncoordinated breathing. Likewise, patients with COPD have difficulty in chewing and swallowing due to respiratory distress. The aforementioned changes have also been noted in previous research; Eating in severe COPD patients reduces SpO_2 , decreases lung function, and increases dyspnea (14–16). There are many physiological events that have the potential to affect a patient's respiratory capacity after a meal (4). Moreover, studies have suggested that the effects of eating on the cardiovascular, metabolic and gastrointestinal systems also affect lung function (17).

Pulmonary functions change according to activity level in patients with COPD; a decrease in respiratory capacity and an increase in dyspnea level (7). Wolkove et al. (15) showed that eating, parallel to our study, increases the level of dyspnea in COPD patients. This finding suggests that eating activity is an effort-requiring activity for severe COPD patients. Swallowing, chewing and arm muscle activities during eating may increase dyspnea level. A study conducted by Cassiani et al. (17) demonstrated an increase in the level of dyspnea after eating. They stated that this condition is associated with severe COPD. and eating activity increases the level of dyspnea. Similar to these findings, our study shows that active eating increases dyspnea level in patients with COPD. Interestingly this change is only

Table 1. Demographic Information

Gender (Female/Male)	14/26
Age (year) (Mean±SD)	65,3±10,9
BMI (kg/m2) (Mean±SD)	27,9±8,2
Non-Obese (n= 25)	22,75±3,36
Obese (n= 15)	36,67±6,40
Smoking (Smoker/Non-smoker)	22/18
O ₂ Supplement (User/Non-user)	11/29

BMI: Body Mass Index

Table 2. Comorbidities

	Frequency (%)	
	W (n=14)	M (n=26)
Hypertension	7 (50)	10 (38,5)
Diabetes Mellitus	5 (35,7)	5 (19,2)
Heart Failure	1 (7,1)	5 (19,2)
Osteoarthritis	2 (14,3)	0 (0)
Gout	1 (7,1)	0 (0)
Biomass Exposure	4 (28,6)	2 (7,7)
Anemia	0 (0)	1 (3,8)
Colon Cancer	0 (0)	1 (3,8)

W: Women, M: Men

Table 3. Comparison of FEV₁, FVC, FEV₁/FVC, BORG and SpO₂ Values Before and After Active Eating

	Before (n=40) Mean±SD	After (n=40) Mean±SD	p (n=40) (Before-After)
FEV₁ (L)	0,88±0,28	0,92±0,30	0,955*
FVC (L)	1,75±0,58	1,84±0,68	0,351*
FEV₁/FVC (%)	51,8±11,5	52,4±14	0,439*
SpO₂ (%)	95,2±3,85	95±3,94	0,903*
BORG	5,78±2,13	5,98±2,22	0,003*

FEV₁: Forced expired volume in one second, FVC: Forced vital capacity, SpO₂: Oxygen saturation, *Paired Samples T Test

prominent in non-obese group, not in obese group.

Many findings confirm that lung volumes, particularly FRC and ERV, decrease with increasing body weight (12). On the other hand, a study has shown that low BMI is a risk factor for lung function declining, especially in FEV₁, compared to normal BMI and high BMI values have a protective effect with a slower decrease in lung function paradoxically (18). Differences in BMI in patients with COPD may play a role in the pathophysiology of the disease and affect patient outcomes. There is a positive relationship between higher BMI values in COPD patients and higher

rates of dyspnea, increased use of inhaled medications, lower quality of life (11). In our study, FEV₁, FVC and dyspnea level in non-obese group showed significant changes after active eating compared to before, while there was no change in other parameters. In the obese group, there was no change in any evaluated parameters. This suggested that the BMI of the participants being non-obese or obese would not have an effect on the immediate results of active eating. The reason why there was no change in any of the parameters evaluated in the obese group may be due to the fact that chest compliance is already limited due to the decrease

Table 4. Comparison of FEV₁, FVC, FEV₁/FVC, BORG and SpO₂ Values Before and After Active Eating According to Non-Obese and Obese Subgroups

	Non-Obese (n= 25)			Obese (n= 15)		
	Mean±SD			Mean±SD		
	Before	After	p	Before	After	p
FEV₁ (L)	0,81±0,28	0,88±0,33	0,003	0,98±0,26	1,01±0,25	0,510
FVC (L)	1,67±0,61	1,84±0,77	0,021	1,89±0,51	1,87±0,51	0,779
FEV₁/FVC (%)	51,21±12,26	50,59±14,03	0,743	52,77±10,57	55,47±13,77	0,204
SpO₂ (%)	94,83±4,61	94,79±4,76	0,900	95,20±2,43	95,33±2,16	0,164
BORG	5,68±2,30	5,92±2,45	0,011	5,93±1,87	6,07±1,87	0,499

FEV₁: Forced expired volume in one second, FVC: Forced vital capacity, SpO₂: Oxygen saturation, *Paired Samples T Test

in chest wall mechanics and that they have difficulty in realizing the effect of active eating effort on the existing dyspnea.

Spirometry is a safe, practical, and standardized measurement method used to determine the functional capacity of the lung in COPD (19). Although ATS recommends patients not to eat large portions of meals within two hours before the test, there is no standardized procedure for whether spirometry should be done before or after a meal, or at least how many hours after a meal (20). In clinical practice, not eating at least 2 hours before the test can be quite difficult due to other accompanying problems (4).

Some researchers have emphasized that there is no relationship between eating and spirometric evaluations (4,21). Moreover, some other studies show that eating does not have a negative effect on pulmonary function (22). Findings of our study support the literature and show that Active eating does not have a negative effect on pulmonary function in general, but for non-obese group there is a difference after eating.

In many studies, daily activities in COPD patients were found to be associated with a decrease in SpO₂ values (9,10). In their study, Wolkowe et al. (15) found a significant decrease in the SpO₂ values of COPD patients after eating. They emphasized that this decrease was associated with an increase in dyspnea and increased upper extremity activity. Cassiani et al. (17) highlighted that eating activity led to a significant decrease in SpO₂ values in moderate and severe COPD cases. Unlike those results, we found no difference in SpO₂ values both in general and subgroups. This discrepancy may be due to applied different measurement methods such as arterial blood gas analysis.

This study has some limitations. Because of its being non-invasive, easy, and cheap application,

pulse oximetry can be considered an advantageous method, but more comprehensive evaluation could be made by arterial blood gas analysis. This difficulty arises from hospital over-load which did not allow collecting blood samples of patients before and after active eating. In the study, FEV₁, FVC, FEV₁/FVC SpO₂ and BORG values were compared in obese and non-obese subgroups on the basis of BMI. Since the number of participants was not sufficient to seek statistical significance in five different subgroups, participants could not be classified as underweight, normal, overweight, obese and extremely obese.

Considering the results of our study, in group D COPD patients, active eating is an effortful activity and causes a significant increase in dyspnea level for all participants and for non-obese subgroup. We appreciate that this result may be important in terms of planning any activity that could give rise to more energy consumption and should not be recommended to patients just after a meal.

Conflict of interest: Authors state no conflict of interest.

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