Can Spirometry, Pulse Oximetry and Dyspnea Scoring Reflect Respiratory Failure in Patients with Chronic Obstructive Pulmonary Disease Exacerbation?

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Key Words

Chronic obstructive pulmonary disease • Respiratory failure • Pulse oximetry • Dyspnea scoring • Spirometry

Abstract

Objective: To evaluate the extent to which oximetry, spirometry and dyspnea scoring can reflect hypoxemia and hypercapnia among patients admitted to the emergency department (ED) with acute exacerbations of chronic obstructive pulmonary disease. **Subjects and Methods:** Spirometry, oxygen saturation by pulse oximetry (SpO₂), arterial blood gas analysis and dyspnea scoring assessments were made in the ED. Correlations of these parameters were evaluated by means of Pearson's test. Pulse oximetry cutoff values to express hypoxemia were demonstrated by receiver operating characteristic (ROC) curves. **Results:** 76 patients with a mean age of 68.0 years were included in the study. Mean spirometric values, expressed as percentages of predicted values, were forced expiratory volume in 1 s (FEV₁) = 23.1 ± 9%; forced vital capacity (FVC) = 32.8 ± 11%, and mean FEV₁/FVC = 72.4 ± 21.6%. While there was a positive correlation between the SpO₂, SaO₂ and PaO₂ values (r = 0.91 and 0.80, respectively), a negative correlation (r = -0.74) was observed between PaCO₂ and SpO₂. In determining hypoxemia, both SpO₂ and FEV₁ were sensitive (83.9 and 90.3%,

respectively) while dyspnea scoring was the most sensitive (93.5%). In the evaluation by means of an ROC curve, a saturation of 88.5% for the pulse oximeter was the best cutoff value to reflect hypoxemia (sensitivity 95.6%, specificity 80.6%). **Conclusion:** SpO₂ alone appears to be as highly specific as a combination of other tests in the evaluation of hypoxemia. A cutoff value for SpO₂ of ≤88.5% is proposed as a criterion in screening for hypoxemia.

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Introduction

According to a report of the World Health Organization, chronic obstructive pulmonary disease (COPD) was the sixth leading cause of death in the world, and it is estimated that by the year 2020, COPD will be the third leading cause of death and the fifth leading cause of disability worldwide [1, 2]. Moreover, approximately 10% of all hospitalizations are attributable to COPD [3]. The mortality risk for acute exacerbations of COPD (AECOPD) has been shown to be about 8 and 23%, respectively, in hospital and 1 year after discharge in a prospective study [4].

Patients with COPD exacerbations are frequently referred to the emergency department (ED), but during the

treatment period there, it is not always easy for the clinician to make a decision about discharge of the patient from the ED or whether to keep the patient hospitalized for monitoring. Direct measurement of the pulmonary function with a bedside spirometer and arterial blood gas (ABG) analysis have been suggested as valuable measurements when determining the severity of an AECOPD and in making a decision about the state of the patient [5–7]. Although physical examination is important in the evaluation of the patients in ED, the signs of airway obstruction are not always obvious before a significant deterioration in pulmonary function [8]. To what extent is it justified to make a decision about the severity of the disease on the basis of forced expiratory volume in 1 s (FEV₁) during the period of acute attack? Lung function tests may be difficult for sick patients to undergo. Also in Turkish hospitals, a costly method like ABG analysis is not always available.

According to current clinical practice, patients suffering from acute hypoxemia or hypercapnia are hospitalized [9]. However, COPD patients with chronic respiratory insufficiency are usually both hypoxemic and hypercapnic.

The present study was undertaken to identify tests using simpler technical equipments to determine COPD severity and evaluate patients more effectively. The question posed was whether or not spirometry, pulse oximetry and dyspnea scoring would reflect respiratory failure in patients with COPD exacerbations. These measurements were assessed regarding their merit for identifying hypoxemia and hypercapnia in comparison with results from the ABG analysis of samples.

Methods

The study was carried out during January to September, 2001, in the Department of Emergency Medicine, Faculty of Medicine, Dokuz Eylül University, Izmir, Turkey, after obtaining approval of the local Ethics Committee. Seventy-six patients, mean age 68 ± 9.3 years (range 48–90), 47 male and 29 female, were included in the study. All were followed up in a Chest Clinic for 9.0 ± 7.6 years (range 1–35). Each patient signed an informed consent form and completed a questionnaire giving demographic and health particulars.

Seventy-four of the 76 patients had been using various bronchodilator drugs and 25 of them were administered continuous oxygen therapy due to respiratory failure. In 28, there were additional diseases accompanying COPD, such as congestive heart failure and coronary artery disease. Fifty-eight patients were smokers and the mean amount of smoking was 29 ± 13 packs per year (range 10–60).

Diagnosis and severity of COPD were established by a respiratory physician on the basis of Global Initiative for Chronic Obstructive Lung Disease criteria [10]. Existence of at least one of the following criteria was considered an acute exacerbation of the COPD patient: worsening of dyspnea, increase in sputum production and/or purulence. Exclusion criteria were being under the age of 40 years, lack of cooperation, early intubation due to respiratory failure, inability to perform spirometry, having asthma, or another acute condition, i.e., pneumonia, pneumothorax, pulmonary emboli or acute congestive heart failure warranted hospital admission.

Pulse rate, blood pressure, temperature values and the respiration rate were measured; chest radiograph was taken through the use of a portable X-ray machine to rule out other acute comorbid conditions. Bradypnea was accepted if the respiration rate was below 12/min and tachypnea if above 20/min [11, 12]. Pulmonary function tests, ABG analysis, oxygen saturations obtained with pulse oximetry (SpO₂) and dyspnea scoring were determined. The condition of the patients after treatment (being discharged from the ED or being hospitalized) and monitoring were all reported in the questionnaire.

Pulmonary function tests were assessed with forced expiratory maneuvers through the use of the portable Micro Medical Micro Plus Spirometer which was calibrated and checked weekly with a 6-liter injector with an outer connection of 30 mm length. The physician demonstrated the procedure to all patients. Prior to beginning any treatment, the patient was asked to perform the maneuver while seated and wearing nose clips. Each patient performed at least two or three acceptable forced expiratory maneuvers. The highest values of measured FEV₁, forced vital capacity (FVC) and peak expiratory flow (PEF) were obtained and shown as a percentage of predicted normal values based on age, height and sex according to the European Community for Coal and Steel report [13]. Based on the GOLD criteria [10], COPD was considered very severe if FEV₁ was <30% of the predicted value; severe if $30 \leq \text{FEV}_1 < 50\%$; moderate if $50 \leq \text{FEV}_1 < 80\%$, and mild if the FEV₁/FVC ratio was <70 and FEV₁ $\geq 80\%$ of the predicted value.

A blood gas sample while breathing room air was obtained from the radial artery before the initiation of any therapy. The arterial blood was collected in a heparinized glass syringe, placed on ice and immediately transported to our laboratory for analysis on the model Nova Stat 9. The pH, PaO₂, PaCO₂, HCO₃ and SaO₂ values were measured. PaO₂ <60 mm Hg in ABG as hypoxemia, and PaCO₂ >50 mm Hg as hypercapnia were considered acceptable as reference values [14].

SpO₂ values were obtained by a pulse oximeter (Athena) before treatment while breathing air, and were recorded as a percentage of saturation. Throughout monitoring, patients were followed with pulse oximetry. Those with SpO₂ values <90% were considered to have a respiratory failure as well as hypoxemia [10, 15]. For an objective evaluation of dyspnea, a five-stage scoring system suggested by the American Thoracic Society was used. Dyspnea scores 3 and 4 were regarded as serious dyspnea [15].

Statistical Analysis

SPSS 10.0 statistics program was used to conduct the statistical analysis. The correlations between pulmonary function test parameters, SpO₂ values, the results of ABG analyses and dyspnea scoring were evaluated by means of Pearson's test. Sensitivity,

specificity, negative and positive predictive values of the use of dyspnea scoring and pulse oximetry were evaluated. Additionally, an index was developed which was defined as: A = $FEV_1 < 30\% + SpO_2 < 90\%$; B = $FEV_1 < 30\% + SpO_2 < 90\% +$ severe dyspnea scores; C = $SpO_2 < 90\% +$ severe dyspnea scores. The sensitivity, specificity, negative and positive predictive values were calculated using this index. The pulse oximetry cutoff values to express hypoxemia were demonstrated by drawing receiver operating characteristic (ROC) curves.

Results

At the first evaluation in the ED, a mean pulse rate of 106 ± 20 beats/min, a mean systolic blood pressure of 151 ± 28 mm Hg and a mean diastolic blood pressure of 95 ± 19 mm Hg were detected. The mean respiration rate was 33 ± 6 breaths/min (range 24–48), and the mean dyspnea score was 2.9 ± 0.9 (range 0–4); clinically serious dyspnea (levels 3 and 4) was present in 55 patients (72%). Mean spirometric percentages of predicted values were: $FEV_1 = 23 \pm 9.3\%$, $FVC = 33 \pm 11.5\%$, $PEF = 20 \pm 10.9\%$, and mean $FEV_1/FVC = 72.4 \pm 21.6\%$. In the spirometric evaluation of cases during the postexacerbation period, mean FEV_1 of patients was $40.6 \pm 15.2\%$, mean FVC was $56.1 \pm 17.4\%$, and mean FEV_1/FVC was $55.8 \pm 12.5\%$. When referred to the ED, FEV_1 was $< 30\%$ of the predicted value in 61 (80%) of the patients, and in 2 it was $> 50\%$ of the predicted value. Twenty-eight (36.8%) of the patients were hospitalized. Those hospitalized had lower FEV_1 , SpO_2 and PaO_2 values than the nonhospitalized or those discharged from ED. $PaCO_2$ and dyspnea scores were higher in hospitalized patients. While SpO_2 of hospitalized patients was 79.6% on average, that of discharged patients was 91.9%, and while PaO_2 of hospitalized patients was 50.1 mm Hg, that of those discharged was 66.2 mm Hg.

Mean ABG values were as follows: $pH = 7.40 \pm 0.04$ (range 7.30–7.51); $PaO_2 = 60 \pm 24$ and $PaCO_2 = 46 \pm 14$ mm Hg; $SpO_2 = 87 \pm 10\%$. Thirty-one patients (41%) were determined to be hypoxemic and the average pH was 7.38 ± 0.05 . In the remaining 45 cases, the PaO_2 value was > 60 mm Hg and the mean pH was 7.41 ± 0.03 . There was a very strong correlation ($r = 0.91$, $r = 0.80$) of the SpO_2 and PaO_2 with SpO_2 values ($p = 0.000$ and 0.000 , respectively), whereas there was again a strong but a negative correlation ($r = -0.74$; $p = 0.000$) between $PaCO_2$ and SpO_2 values. Additionally, reasonable correlations were observed between FEV_1 and PaO_2 and between dyspnea score and PaO_2 (table 1). These values indicate that a deterioration in the ABG values that accompanies wors-

Table 1. The correlation coefficients between the ABG values and pulse oximetry results

	$PaCO_2$	PaO_2	SpO_2
FEV_1	-0.44*	0.40*	0.16*
FVC	-0.36*	0.35*	0.35*
PEF	-0.30*	0.13	0.23*
SpO_2	-0.74*	0.80*	0.91*
Dyspnea scores	0.37*	-0.45*	-0.45*

* $p < 0.05$.

ening of respiratory function leads to changes in $PaCO_2$ and PaO_2 in particular. Compared to $PaCO_2$, a more apparent correlation (table 1) was seen between PaO_2 , SpO_2 and SpO_2 values with an increase in dyspnea scoring. As the dyspnea scoring increased, so did the PaO_2 and SpO_2 values (table 1).

In determining hypoxemia, compared with ABG values, oxygen saturations obtained with both pulse oximetry (SpO_2) and FEV_1 are highly sensitive, but dyspnea scoring is the parameter that gives the highest sensitivity. Only SpO_2 was found to be highly specific in determining hypoxemia (table 2). For the detection of hypercapnia, dyspnea scoring was the most sensitive, whereas the test having the highest specificity was SpO_2 (table 3).

The combinations of FEV_1 , SpO_2 and dyspnea severity scoring resulted in decreased sensitivity (80.7%), but increased specificity (88.9%) for detecting hypoxemia. However, SpO_2 alone seemed to be as highly specific as the combination of other tests in the evaluation of hypoxemia. For hypercapnia, the application of these tests together indicated lower sensitivity but higher specificity (73.1 and 78%, respectively) than when used individually.

When the sensitivity and specificity of pulse oximetry analysis were assessed with the ROC curve in the diagnosis of hypoxemia, the area below the curve was determined to be 0.95 ± 0.02 (fig. 1). The highest and most acceptable value for sensitivity was 95.6% and for specificity 80.6% corresponding to 88.5, which would be considered the cutoff value of SpO_2 (table 4).

Discussion

For outpatients who are referred to the ED with AECOPD, the morbidity and mortality risk increases if there is a severe functional deterioration. However, sever-

Table 2. Sensitivity, specificity and predictive values for FEV₁, SpO₂ and dyspnea scoring for the detection of hypoxemia using ABG values as the reference

	ABG analysis (PaO ₂ <60 mm Hg)			
	sensitivity, %	specificity, %	PPV, %	NPV, %
FEV ₁ <30%	90.3 (28/31)	26.7 (12/45)	45.9 (28/61)	80.0 (12/15)
SpO ₂ <90%	83.9 (26/31)	88.9 (40/45)	83.9 (26/31)	88.9 (40/45)
Dyspnea scores ¹	93.5 (29/31)	42.2 (19/45)	52.7 (29/55)	90.5 (19/21)

PPV = Positive predictive value; NPV = negative predictive value.

¹ Dyspnea scores of 3 and 4 were regarded as serious dyspnea.

Table 3. Sensitivity, specificity and predictive values for FEV₁, SpO₂ and dyspnea scoring for the detection of hypercapnia using ABG values as the reference

	ABG analysis (PaCO ₂ >50 mm Hg)			
	sensitivity, %	specificity, %	PPV, %	NPV, %
FEV ₁ <30%	88.5 (23/26)	24.0 (12/50)	37.7 (23/61)	80.0 (12/15)
SpO ₂ <90%	73.1 (19/26)	76.0 (38/50)	61.3 (19/31)	84.4 (38/45)
Dyspnea scores ¹	92.3 (24/26)	38.0 (19/50)	43.6 (24/55)	90.5 (19/21)

PPV = Positive predictive value; NPV = negative predictive value.

¹ Dyspnea scores of 3 and 4 were regarded as serious dyspnea.

Table 4. The various levels of screening cutoff for the detection of hypoxemia by pulse oximetry

SpO ₂ saturation evaluations, %	Sensitivity %	Specificity %
84.5	97.8	54.8
86.0	97.8	64.5
87.5	97.8	74.2
88.5	95.6	80.6
89.5	88.9	83.9
90.5	71.1	96.8
91.5	66.7	96.8

ity of symptoms and functional parameters may not reflect the real condition of the patient [16–18]. The active use of sternocleidomastoid muscles, pulsus paradoxus, and serious airway obstructions have been detected in only 48% of the patients that have FEV₁ below 1 liter [19]. Poor sensitivity of symptoms alone makes it necessary to assess objective respiratory function of COPD patients in the ED as shown in our study, where 72% of the patients had clinically serious dyspnea (levels 3 and 4).

Conversely, for the evaluation of respiratory failure in patients with COPD acute attacks, most diagnostic and

Fig. 1. Demonstration of the sensitivity and specificity of pulse oximetry with the ROC curve in the diagnosis of hypoxemia.

treatment guidelines recommend ABG analysis. Raffin [20], however, does not recommend this as a specific test for the evaluation of COPD patients. The American Thoracic Society [21] suggests ABG analysis for COPD pa-

tients whose FEV₁ is below 1.5 liters and who have to be hospitalized due to respiratory failure. In our study, when FEV₁ was <30%, the sensitivity was high and the specificity low for the detection of hypoxemia and hypercapnia. Because the specificity of the test was low, only a few of those who were not really sick during the period of acute attack could be identified, thus giving very high false-positive results. Emerman et al. [16] have noted a weak negative correlation between PaCO₂ and FEV₁ in patients with AECOPD; however, they have not detected such a correlation with PaO₂ [6]. We observed a moderate but significant correlation between FEV₁ and both PaO₂ and PaCO₂.

Does SpO₂ predict the results of ABG analysis? Currently, in many clinical cases, pulse oximetry is used instead of ABG analysis. For the evaluation of room air ABG results, Witting and Lueck [22] detected the value 96% for SpO₂ when they used the cutoff value for hypoxemia (PaO₂ <70 mm Hg) and hypercapnia (PaCO₂ >50 mm Hg). This level of SpO₂ gave 100% sensitivity and 54% specificity for hypoxemia. While the sensitivity for hypercapnia remains the same, specificity decreased to 31% [22]. Also, Neihoff et al. [23] have determined that the value ≤94% of SpO₂ detected patients having PaO₂ <70 mm Hg, with 100% sensitivity. Considering the cutoff value for SpO₂ as <88.5%, we observed that according to hypoxemia detected by ABG analysis, the sensitivity and specificity of SpO₂ was significantly high. Additionally, both the positive predictive value and negative predictive value were also higher for SpO₂ than other non-invasive parameters in revealing hypoxemia.

To our knowledge, there is only one study evaluating the role of pulse oximetry in AECOPD [24]. Using a cutoff value of SpO₂ ≤92% for those admitted to the ED, these authors reported 100% sensitivity and 86% specificity as a screening test for hypoxemia. In the present study, the use of ≤88.5% of SpO₂ as the cutoff value gave 96% sensitivity and 81% specificity in revealing hypoxemia.

CO₂ retention in COPD is a late finding. When the correlation of PaCO₂ and SpO₂ is evaluated with the patient breathing room air, hypercapnia may be estimated by considering the SpO₂ value. Witting and Lueck [22] reported a SpO₂ value of <94% in patients with a PaCO₂ value of >60 mm Hg and <96% in those with PaCO₂ of >50 mm Hg. For PaCO₂ values >50 mm Hg, the sensitivity and specificity were 100 and 31%, respectively. In our study, the sensitivity of SpO₂ in detecting hypercapnia during exacerbation was 73.1%, and the specificity was 76%.

A mild correlation was observed between hypoxemia and hypercapnia with dyspnea scoring. It was also observed that the mean PaO₂ value in the cases with dyspnea of 3rd to 4th degree was in accordance with hypoxemia, and hypercapnia was also seen in patients whose dyspnea score was 4. There was a higher sensitivity but lower specificity than with other tests (FEV₁, SpO₂) when only dyspnea scores were used in determining hypoxemia and hypercapnia. When dyspnea scoring was added to the other tests, a minimal decrease in the sensitivity of the tests was observed while there was an apparent increase in the specificity. In the hospital EDs where ABG analysis and spirometry are not available, hypoxemic patients can be detected with 81% sensitivity and 89% specificity using pulse oximetry and dyspnea scoring. For hypercapnia, the application of these tests together gave lower sensitivity and specificity (73.1 and 78%, respectively).

One third of the patients required continuous O₂, so their ABG and SpO₂ values are likely to meet 'exacerbation' cutoff values all the time. Thirty-one patients (41%) were determined to be hypoxemic and in the remaining 45 cases, the PaO₂ value was >60 mm Hg. The average pH of 7.38 in the hypoxemic group and the lowest pH of 7.30 suggest that very few were experiencing a significant acute rise in PaCO₂ that causes respiratory acidosis. Therefore, it was not possible to evaluate the usefulness of the mentioned noninvasive parameters in acute hypercapnic situations (or respiratory failure), which can be considered an important limitation of the present study.

Nearly 40% of the patients were hospitalized. The hospitalized patients had lower FEV₁, SpO₂ and PaO₂ values than nonhospitalized patients. PaCO₂ and dyspnea scores were also higher in hospitalized patients. The ongoing symptoms of the patients despite the medical therapy in the ED, and hypoxemia detected in pulse oximetry monitoring guided the decision on hospitalizing patients.

Conclusion

Although there are objective clinical and noninvasive assessments that reveal the clinical severity during an AECOPD, in this study, the highest sensitivity and specificity was achieved by application of SpO₂ alone. This study shows that for patients who are admitted to the ED due to AECOPD, functional deterioration can be easily managed if SpO₂ is over 88%; however, if SpO₂ is less than 88%, the patients are more likely to receive better care if referred to a clinical center with full monitoring capabilities.

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