Predictability of Oxygen Desaturation During Sleep in Patients With Cystic Fibrosis*

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Background: The purpose of this study was to determine how common sleep-related desaturation with preserved awake resting pulse oximetric saturation (SpO2) was in a large cohort of adult cystic fibrosis (CF) patients with variable degrees of pulmonary disease. We then determined whether nocturnal desaturation could reliably be predicted from standard clinical and exercise parameters.

Methods: Seventy CF patients participated in the study (mean [SD] age, 27.3 [8.7] years; women, 54%; percent predicted FEV1 [%predFEV1], 55.7% [23.9%]). Nocturnal, resting, and exercise Spo2 were measured. Nocturnal oximetry was measured in the patient’s home. Maximal oxygen capacity (Vo2max) was determined from a graded exercise test on a stationary bicycle ergometer. The Shwachman-Kulczycki (S-K) illness severity score was calculated incorporating categories of functional capacity, physical examination, nutrition, and chest radiograph.

Results: Multivariate analysis reported significant differences (p < 0.0001) between pulmonary disease severity and overall distribution of nocturnal SpO2, with the main difference being for patients with severe pulmonary disease (%predFEV1 of < 50%) compared to patients with mild or moderate disease in the SpO2 intervals of 100 to 96% (p < 0.0001) and 90 to 86% (p = 0.0001). Pulmonary function, S-K clinical scores, Vo2max, and resting and maximal SpO2 correlated significantly (p < 0.05) with nocturnal SpO2 levels. Stepwise discriminant analysis identified %predFEV1 (or S-K scores) and resting SpO2 as the parameters that could best discriminate patients not likely to experience nocturnal desaturation. Specifically, our equation could predict 91% of cases less likely to nocturnally desaturate, but could only modestly predict those more likely to desaturate (ie, 26% of cases).

Conclusions: Spirometric parameters and measurements of awake resting oxygenation are of limited utility in predicting nocturnal desaturation. Nocturnal oximetry should be considered in patients with moderate to severe lung disease even with preserved awake resting SpO2.

Key words: cystic fibrosis; exercise desaturation; nocturnal hypoxemia

Abbreviations: CF = cystic fibrosis; HR = heart rate; kp = kilopond; %predFEV1 = percent predicted FEV1; REM = rapid eye movement; Spo2 = pulse oximetric saturation; Spo2start-max = Spo2 at start of exercise minus Spo2 at maximal effort; S-K = Shwachman-Kulczycki; Vo2 = oxygen uptake; Vo2max = maximum oxygen capacity; V/Q = ventilation/perfusion

Cystic fibrosis (CF) is an autosomal recessive disorder with mortality in most instances accounted for by lung disease. The primary pathophysiologic process that is responsible for the premature death and disability in patients who have CF is chronic airway inflammation and infection. This begins in early childhood and leads to progressive airflow obstruction and restriction. These processes lead to bronchiectasis, pulmonary hypertension, and often ultimately to respiratory failure.

Hypoxemia is a feature of more advanced lung disease, with deleterious effects that have been well established in patients with COPD.1,2 Supplemental oxygen used appropriately in this setting reduces morbidity and mortality.1,2 In CF, as in other lung diseases, desaturation is accentuated during sleep. Desaturation is caused by changes in mechanics of
breathing, derecruitment of ventilatory muscles (particularly in rapid eye movement [REM] sleep), ventilation/perfusion (V/Q) mismatch, and possible reduced mixed venous oxygenation.

A goal of this study was to determine how common sleep-related desaturation occurred with preserved awake resting pulse oximetric saturation (SpO2). We also sought to determine whether nocturnal desaturation could be reliably predicted from standard clinical parameters evaluating pulmonary disease. These issues are of relevance in identifying those requiring oxygen supplementation. Hemoglobin desaturation during sleep has been documented to be more prevalent in CF patients with more severe pulmonary disease. These patients have been characterized as generally exhibiting percent predicted FEV1 (%predFEV1) and resting (oxygen saturation) SpO2 values < 65% and 94 to 93%, respectively. Limited information is available describing exercise-related changes in SpO2 and measures of aerobic capacity to nocturnal oxygen. We sought to extend these relationships to a larger group of patients with more variable pulmonary disease and to monitor nocturnal SpO2 in the home environment, reasoning that this was a better estimation than in-hospital, with the confounding effects of pulmonary infection and intervention.

**Materials and Methods**

**Sample**

Seventy adult patients with CF (age range, 17 to 53 years), equally distributed for gender and severity of lung disease (as characterized with %predFEV1) participated in the study (Table 1). Details of the experimental procedures and an explanation of the risks involved were provided for each subject before obtaining written consent. The study was approved by the Ethics Committee of the University of British Columbia and St. Paul’s Hospital. Patients underwent a battery of tests that included pulmonary function, resting and maximal exercise cardiopulmonary, overnight oximetry, and chest radiography. Tests were performed on the patients during stable clinical status. Stable clinical status was defined as the absence of the following: (1) pulmonary exacerbation requiring IV antibiotics over the past 2 months or the need for current oral antibiotics, and (2) any of the following clinical symptoms: increased cough, sputum volume and purulence, increased dyspnea, reduced weight, and a fall in FEV1 of > 10% from stable baseline.

**Pulmonary Function**

The values for spirometry were expressed as the ratio of percentage of normal values based on age, gender, and height. Spirometry was carried out according to American Thoracic Society criteria. The best value of a minimum of three adequate postbronchodilator measurements was taken with the FEV1, and the FVC of the best two of these efforts not varying by > 5% or 100 mL, whichever was greater. The values for spirometry were expressed as the ratio of percentage of normal values based on age, gender, and height.

**Resting and Exercise Tests**

The maximum oxygen capacity (V\(\text{O}_2\)max) test was performed on a Monark stationary bicycle (Monark Ergometric 818E; Monark; Stockholm, Sweden) and followed a continuous progressive incremental regimen. The workload was increased each minute by 0.25 kiloponds (kp) and patients asked to maintain pedal frequency within the range of 60 to 80 revolutions/min throughout the test. Initial workload was individually set and was dependent on physique, fitness level, and severity of disease for each patient and ranged from 0 to 1.5 kp and increased thereafter by 0.25 kp. Metabolic measurements were obtained at 30-s intervals utilizing a Beckman metabolic measurement cart (Beckman Instruments; Anaheim, CA). Heart rate (HR) and SpO2 were monitored throughout the test with a finger pulse oximeter (Ohmeda Biox 3700; BOC Healthcare; Louisville, CO) (an HR monitor [Accurex 900730; POLAR Electro; Oy, Finland] was also used to monitor HR), and minute values were recorded. The 10-point Borg scale was used to report leg and chest scores for perceived exertion and dyspnea, respectively, every 2 min during the test. The test was terminated by the subject due to volitional fatigue or by the researchers if there was a plateau in oxygen uptake (V\(\text{O}_2\)) and HR for > 1 min, or SpO2 levels fell to < 82%. V\(\text{O}_2\)max was substantiated with subjects meeting at least two of the following three criteria (except in cases where the test was terminated due to SpO2 of < 82%): a plateau for > 1 min of V\(\text{O}_2\) (± 2 mL/kg/min) and HR (± 3 beats/min) with increasing workload and a respiratory exchange ratio of ≥ 1.10. Subjects were asked to refrain from eating 2 to 3 h prior to testing, and from heavy exercise 2 days before V\(\text{O}_2\)max testing.

**Nocturnal Oximetry**

Nocturnal, awake resting, and exercise SpO2 were measured by pulse oximetry (Ohmeda Biox 3700; BOC Healthcare) using a finger probe. The patient completed the nocturnal test at their home. Patients were trained in the use of the oximeter and the proper fit of the finger probe in order to ensure that their resting SpO2 recording sitting upright was similar to the values obtained in the laboratory. The acoustic signals for pulse volumes and alarms were silenced. The Satmaster (EMG Scientific; Los Angeles, CA) software package was used for data presentation of nocturnal results.

**Table 1—Clinical Characteristics of the Study Population**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean (SD)</th>
<th>Range</th>
</tr>
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<tbody>
<tr>
<td>Age, yr</td>
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<td>17.0–53.0</td>
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<tr>
<td>Sex, M/F</td>
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<tr>
<td>BMI, kg/m²</td>
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<tr>
<td>%predFEV₁</td>
<td>55.7 (23.9)</td>
<td>21.0–113.0</td>
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<tr>
<td>Nocturnal SpO₂ (% &lt; 90%), %</td>
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<td>0–99.0</td>
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<tr>
<td>Mean nocturnal SpO₂</td>
<td>92.3 (4.1)</td>
<td>70.0–98.0</td>
</tr>
<tr>
<td>Resting SpO₂, %</td>
<td>96.3 (1.9)</td>
<td>89.5–100.0</td>
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<tr>
<td>Maximum SpO₂, %</td>
<td>92.3 (4.9)</td>
<td>81.0–100.0</td>
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<tr>
<td>S-K scores</td>
<td>64.2 (16.1)</td>
<td>25–100</td>
</tr>
<tr>
<td>Brasfield scores</td>
<td>14.2 (4.3)</td>
<td>4–23</td>
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</table>
Clinical Scores

Shwachman-Kulczycki (S-K) and Brasfield scores were calculated. S-K scores were calculated from a composite of four categories: general activity, physical examination, nutrition, and chest radiographic findings when patients were clinically stable (nonpulmonary exacerbation). Chest radiographs were scored utilizing the Brasfield clinical scoring system.

Statistical Analysis

The Statistical Package for the Social Sciences (SPSS; Chicago, IL) statistical software package was utilized. Pearson product moment correlations were used to investigate the relationship between resting and exercise metabolic parameters, spirometry, clinical parameters, and nocturnal oximetry. Multivariate analysis was performed to compare differences between groups (according to disease severity) and overall distribution of their nocturnal SpO₂. Univariate analysis was used to determine where there were differences in nocturnal SpO₂, and concluded with post hoc analysis to ascertain group differences. Stepwise discriminant analysis was performed to assess prediction of nocturnal desaturation (whether CF patients desaturate or not during sleep) from standard clinical, resting, and exercise parameters. Level of significance was set at 0.05.

RESULTS

The study sample consisted of 70 CF patients with heterogeneous disease severity. Table 1 presents some overall clinical status parameters and the age and gender distribution of the study sample. SpO₂ is presented during rest, sleep and maximal exercise, as well as VO₂max in Table 2 with patients grouped by disease severity according to %predFEV₁ into mild (%predFEV₁ > 70%), moderate (%predFEV₁ from 50 to 70%) and severe (%predFEV₁ < 50%) pulmonary disease. According to pulmonary function grouping, there were 18, 21, and 31 patients classified as having mild, moderate, and severe pulmonary disease, respectively. Figure 1 presents the percentage of time patients within each severity grouping spent sleeping within each SpO₂ interval during sleep. Figures 2, 3 present nocturnal oximetry values (mean nocturnal SpO₂ and percent time spent at < 90% SpO₂, respectively) plotted against resting SpO₂ and %predFEV₁. We found all patients in our sample with awake resting SpO₂ of < 93% (n = 3) to markedly desaturate during sleep. A heterogeneous relationship was observed with awake SpO₂ of > 93%, with only a threshold of > 98% reliably excluding nocturnal desaturation. Nocturnal desaturation was uncommon in patients with %predFEV₁ of > 65% predicted (2 of 21 patients), but was common in patients with %predFEV₁ of < 65% predicted (25 of 49 patients). However, nocturnal saturations varied considerably between individuals below this value. For example, the percent of sleep time < 90% saturation for individuals with %predFEV₁ of 30% predicted ranged from 0 to 100%.

Multivariate analysis reported significant differences (p < 0.0001) between groups in the overall distribution of their nocturnal SpO₂. Univariate results identified the groups differing specifically in the SpO₂ intervals of 100 to 96% (p < 0.0001) and 90 to 86% (p = 0.0001). Post hoc analysis found significant differences only in these intervals for the severe group compared to the other two groups (p < 0.05), but no differences were exhibited between the mild and moderate disease groups. Table 3 displays Pearson product moment correlations for clinical and exercise parameters with nocturnal oximetry. Stepwise discriminant analysis was performed to assess prediction of nocturnal desaturation (whether CF patients desaturate or not during sleep) from standard clinical measures (ie, %predFEV₁, Brasfield and S-K clinical scores, resting SpO₂), and common exercise parameters (ie, SpO₂ at start of exercise minus SpO₂ at maximal effort [SpO₂start-max], exercise capacity [VO₂max percent of predicted]). We defined nocturnal desaturation as SpO₂ levels of

| Table 2—Comparison by Severity of Disease of the Parameters of Oxygen Saturation During Rest, Maximal Exercise, and Sleep and Exercise Capacity |
|------------------|------------------|------------------|------------------|
| Variables        | Mild             | Moderate         | Severe           |
|                  | (SD) Range       | (SD) Range       | (SD) Range       |
| Mean nocturnal SpO₂, % | 95.3 (1.9) 90.0–98.0 | 92.2 (5.5) 70.0–97.0 | 90.6 ± 2.8 84.0–97.0 |
| Nocturnal SpO₂ (% time < 90%), % | 3.3 (10.4) 0–41.0 | 7.3 (17.3) 0–74.0 | 32.0 (34.1) 0–89.0 |
| Resting SpO₂, % | 97.6 (1.3) 96.0–99.0 | 96.3 (1.4) 93.0–98.0 | 95.6 (2.1) 90.5–100.0 |
| Maximum SpO₂, % | 96.1 (2.0) 93.0–100.0 | 94.3 (3.6) 84.0–98.0 | 88.5 (4.5) 81.0–97.0 |
| SpO₂start-max, % | 1.3 (1.6) –2.0–3.0 | 2.1 (2.5) 0–8.0 | 6.1 (3.4) –0.5–13.0 |
| VO₂max, mL/kg/min | 33.3 (7.8) 19.1–49.5 | 33.5 (10.0) 19.9–51.6 | 28.0 (8.8) 11.9–44.6 |
| VO₂max, L/min | 2.18 (0.64) 1.11–3.19 | 1.98 (0.72) 0.88–3.36 | 1.61 (0.62) 0.68–3.10 |
| VO₂max % of predicted* | 86.0 (16.0) 54.0–119.0 | 83.0 (16.0) 59.0–123.0 | 64.0 (19.0) 32.0–109.0 |

*Based on VO₂max prediction equation: (3.20 × height in centimeters) − (0.024 × age) + (0.019 × weight in kilograms) − (0.49 × sex) − 3.17.10
< 90% for > 5% of sleep time, based on previous investigations on nocturnal desaturation in CF and chronic obstructive lung disease. Patients were grouped for this analysis into two groups based on whether they desaturated during sleep or not based on the above definition. All 70 patients were used for the analysis; according to our grouping criteria, there were 43 patients who did not desaturate to < 90% for > 5% of sleep time and 27 patients who did desaturate. Initially, the analysis was run including all the variables described above (ie, %predFEV1, Brasfield and S-K clinical scores, resting SpO2, SpO2start-max, V̇O2max percent of predicted), and then without S-K. We took this approach, as S-K was highly correlated with %predFEV1, and S-K is not commonly measured clinically. There was statistically significant separation among the two groups from the seven predictors (χ² = 42.32, p < 0.01) and with the S-K predictor excluded (χ² = 30.25, p < 0.01). One discriminant function accounted for 100% of the between-group variability. The loading matrix of correlations between predictors and discriminant functions (Table 4) suggests that the best predictors for distinguishing between patients who desaturate and those patients who do not are resting SpO2 and S-K scores, or alternatively SpO2 and %predFEV1. S-K scores and %predFEV1 were highly correlated (r = 0.78, p = 0.001), as were chest radiographic scores (ie, Brasfield scores) with both S-K scores (r = 0.73, p = 0.001) and %predFEV1 (r = 0.68, p = 0.001). We showed moderate relationships between resting and exercise oxygen saturation and exercise capacity with the above S-K and Brasfield clinical scores and %predFEV1. Based on the former discriminant analysis, 70.0% of grouped cases were correctly classified. Specifically, 90.7% of patients (39 of 43) who did not desaturate were correctly predicted, and 37.0% of patients (10 of 27) who did desaturate were correctly grouped. Based on the latter discriminant analysis, 65.7% of grouped cases were correctly classified. Specifically, 90.7% of patients (39 of 43) who did not desaturate were correctly predicted; however, only 25.9% of patients (7 of 27) who did desaturate were correctly grouped. Patients were not excluded from the study based on their pharmacologic treatment. Nevertheless, there were no patients receiving oral corticosteroids, theophylline, or antidepressants that may have altered sleep-related breathing.

**Discussion**

Nocturnal oxygen desaturation was commonly observed in our population of adult CF patients. Approximately 40% of a representative group of our clinic had SpO2 values of < 90% for > 5% of the night. Although the threshold of nocturnal desaturation that is physiologically significant in CF patients remains unclear, adverse outcomes in right ventricular hemodynamics and survival in patients who have COPD have been reported at comparable levels. The degree of nocturnal desaturation correlated with various measures of lung disease severity, including clinical, spirometric, and radiographic parameters. Resting awake SpO2 was also predictive of nocturnal oxygenation. We were unable, however, to determine thresholds in any of these measurements that could reliably exclude those who did not desaturate. Nocturnal SpO2 measurements can therefore be...
justified as an independent parameter in the assessment of patients with more advanced CF.

Mean nocturnal SpO₂ was lower with increased pulmonary disease severity, and patients with severe disease spent more of their sleep time at SpO₂ levels of < 90% (Table 2). We also found a greater decline in SpO₂ levels with maximal exercise in those CF patients who had more severe pulmonary disease, with negligible differences seen in those patients who had mild and moderate levels of pulmonary disease. This relationship was best illustrated when the nocturnal SpO₂ values were plotted as percentage of time patients spent at each SpO₂ interval (Fig 1). The mild and moderate groups (based on severity of pulmonary disease) spent the majority of their sleep time in the SpO₂ interval of 100 to 96% and minimal time in the SpO₂ interval of 90 to 86%. The opposite was true for the group with severe lung disease.

Figure 2. Relationship between resting SpO₂ and nocturnal SpO₂. Nocturnal SpO₂ is expressed as mean nocturnal SpO₂ values (top, A) and as percent of sleep time with SpO₂ values < 90% (bottom, B).
We used discriminate analysis to determine which clinical and exercise parameters predicted patients more prone to desaturate during sleep. Resting SpO₂ and %predFEV₁ or S-K and resting SpO₂ were the parameters that were best able to distinguish the patients who did vs those patients who did not desaturate during sleep. However, while the above parameters could predict correctly > 90% of the patients who did not desaturate, only 37% of the patients who did desaturate during sleep could be predicted from these parameters.

A relationship between the severity of lung disease and decrements in nocturnal SpO₂ as found in this study and others⁴,¹⁵,¹⁶ seems intuitive. Alterations in gas exchange and the mechanics of breathing compromise ventilatory function in CF patients. Abnormal ventilation/perfusion (V/Q) relationships and increases in anatomic and physiologic dead space are
ubiquitous with more advanced lung disease. Expira-
tory flow limitation, compensatory hyperinflation with
inspiratory threshold loading, and an increased elastic
load can account for augmented work of breathing.
Ventilatory muscles are less able to meet these de-
mands because of mechanical disadvantage and often
metabolic abnormalities (eg, nutritional compromise).
With these baseline limitations, there is less ability to
adapt to perturbations in ventilatory function with
sleep. This state is characterized by worsening V/Q
defects with a reduction in functional residual capacity,
hypoventilation, alterations in airway tone and caliber,
and possible increased retention of secretions.

Although we did find a correlation between sever-
ity of airflow obstruction and nocturnal desaturation,
this relationship was limited. As previously reported,
CF patients with significant nocturnal desaturation
tend to have advanced pulmonary disease.11,12 How-

ever, appreciable numbers with this severity of air-
flow obstruction have well maintained saturations
during sleep. Versteegh and coworkers4 identified a
%predFEV1 of ≤ 65% to be associated with noctur-
nal desaturation. This is indicative of the relatively
poor correlation of parameters quantitating airflow
mechanics and gas exchange. While we found noc-
turnal desaturation to be very uncommon in those
with milder lung disease (%predFEV1 of > 65% pred-
icted), measurement is required to detect the
presence of nocturnal desaturation in those with
more severe lung disease.

The level of resting awake oxygenation is an
important determinant of subsequent nocturnal
SpO2. We observed, as has previously been report-
ed,4,5,11,12 more nocturnal desaturation in patients
with lower baseline SpO2. Montgomery and col-
leagues11 have shown patients with resting SpO2 of
> 91% spent < 20% of their sleep time with SpO2 of
< 90%, while patients with resting SpO2 of < 92% spent
> 80% of their sleep time with SpO2 of
< 90%. Versteegh and coworkers,4 Braggion and
colleagues,12 and Smith and coworkers5 identified a
resting Sp02 value of ≤ 93% to be associated with
nocturnal desaturation. Such a finding has also been
consistent in patients with other lung diseases, such
as COPD. This can be accounted for by the vul-
erability of patients with values close to the linear
portion of the oxyhemoglobin dissociation curve to
sleep-related changes in gas exchange and the me-
chanics of breathing as outlined above. One expla-
nation for the somewhat lower correlation coeffi-
cients in our study is the underrepresentation of patients
with more reduced awake SpO2 (only three
patients had resting SpO2 levels < 93%; Fig 2).
Nonetheless, we found limited utility of specific
thresholds in Sp02 in determining those who desatu-
rate at night. While all patients with awake resting
SpO2 of < 93% desaturated, there was a very heter-
ogeneous response in individuals with values of Sp02
of > 93%. This study was not designed to determine
why individuals with comparable resting SpO2 had
variable degrees of nocturnal desaturation. Potential
explanations include interpatient variability in drives
to breathe, ventilatory muscle recruitment, V/Q
catching, and venous admixture.

We examined measures of oxygenation with exercise
and levels of maximal exercise capacity to determine
whether these improved identification of patients with
nocturnal desaturation. While there was a general
association, these parameters did not add to the pre-
dictability conferred by spirometry and resting SpO2.
This has generally been consistent with other reports,
although Versteegh and coworkers4 noted higher inter-
correlations for VO2max and exercise SpO2. Their
findings may be accounted for by smaller numbers of

Table 3—Correlation Values Between Lung Function, Indexes of Exercise Capacity, Clinical Scores, Resting SpO2, and Nocturnal SpO2*

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<th>Parameters</th>
<th>Mean Nocturnal SpO2</th>
<th>Nocturnal SpO2 (% Time &lt; 90%), %</th>
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<tr>
<td>%predFEV1</td>
<td>0.49</td>
<td>− 0.48</td>
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<td>S-K clinical scores</td>
<td>0.45</td>
<td>− 0.54</td>
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<tr>
<td>Brasfield scores</td>
<td>0.35</td>
<td>− 0.41</td>
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<tr>
<td>VO2max % of predicted</td>
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<tr>
<td>SpO2max</td>
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<tr>
<td>SpO2start-max</td>
<td>− 0.36</td>
<td>0.41</td>
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<tr>
<td>Resting SpO2</td>
<td>0.54</td>
<td>− 0.56</td>
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*All intercorrelations had significant p values of at least 0.05. SpO2max = maximum SpO2.

Table 4—Results of Discriminant Function Analysis

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<th>Variables with discriminant function</th>
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<th>Univariate F (1,67)</th>
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<td>S-K</td>
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<td>%predFEV1</td>
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<td>SpO2start-max</td>
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<td>VO2max % of predicted</td>
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<td>Canonical R</td>
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<td>Eigen value</td>
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<th>Predictor variables excluding S-K with discriminant function</th>
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<td>SpO2start-max</td>
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<tr>
<td>Canonical R</td>
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<td>Eigen value</td>
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patients with very homogeneous disease severity. We compared the magnitude of fall in SpO2 levels with sleep and exercise. While we found similar absolute values for mean nocturnal and maximal SpO2 levels and similar percent fall in SpO2 levels from resting SpO2 levels (Table 2), the utility of these findings is questionable. Limited correlations between SpO2 changes with sleep and exercise are not surprising. Sleep and exercise have discrepant consequences on the V/Q relationship abnormalities and levels of ventilation. Ventilation increases with exercise and improvements in V/Q mismatch can occur with recruitment of underventilated but perfused areas of lung. Alternatively hypventilation can occur during sleep, greatest in magnitude during REM sleep. Muller and coworkers17 have showed a 7.4% drop in SpO2 to occur during REM sleep, and this drop was associated with a fall in functional residual capacity. Stokes and associates18 showed drops in PO2 (1 to 23 mm Hg) with change in position from upright sitting to supine in their group of CF patients characterized as having mild lung disease (mean %predFEV1 of 72%) as contributing to the nocturnal hypoxemia.

The deleterious effects of hypoxemia for patients with CF can reasonably be extrapolated from patients with COPD. In this patient group, two landmark studies1,2 have shown improvements in survival with supplemental oxygen, better for continuous as opposed to nocturnal use. Other outcome measures improved with oxygen include attenuation in increases in pulmonary hypertension, and improved quality of life with better cognitive function and exercise capacity. These studies established efficacy in individuals with marked hypoxemia (PO2 of < 55 mm Hg) equating roughly to SpO2 values of 88 to 90%. Limited information is available for those with less severe abnormalities in oxygenation. Indeed, Gorecka and colleagues19 found no survival advantage in COPD patients with PO2 of 56 to 65 mm Hg. Hypoxemia isolated to sleep has been shown to confer an adverse prognosis in patients who have COPD with a favorable outcome in survival for this subgroup receiving domiciliary oxygen.13,14 While our definition of nocturnal desaturation was arbitrary, in all cases it exceeded that of ≥ 5 min of SpO2 of < 90%, with a nadir of SpO2 of ≤ 85% employed in these studies. In CF patients, it remains to be established whether nocturnal oxygen supplementation will alter quality of life or disease outcome. Until such information is available, it seems appropriate to correct oxygenation, although the exact threshold of intervention is also speculative.

We conclude that nocturnal oxygen desaturation is very unusual in CF patients with FEV1 of > 65% and is to be expected with stable awake resting SpO2 of < 93%. For patients where these thresholds do not apply, SpO2 changes were heterogeneous, such that measurement would be required to detect and optimally treat this clinically relevant complication of lung disease.

REFERENCES
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