clinical investigations

Pulmonary Function Tests Cannot Predict Exercise-Induced Hypoxemia in Chronic Obstructive Pulmonary Disease*

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We studied 40 patients with chronic obstructive pulmonary disease (COPD) to determine whether measurements of pulmonary function could predict a fall in arterial oxygen pressure (PaO₂) with exercise. The PaO₂ fell more than 3 mm Hg in 21 patients (group 1), did not change (±3 mm Hg) in nine patients (group 2), and increased more than 3 mm Hg in ten patients (group 3). Group 3 had significantly less severe expiratory obstruction than groups 1 and 2. The most significant variables in predicting a change in PaO₂ with exercise were the ratio of the forced expiratory volume in one second over the forced vital capacity (FEV/FVC) and the single-breath carbon monoxide diffusing capacity (Dsb). Measurements of FEV/FVC of 0.50 or more and Dsb of 20 ml/min/mm Hg or more were 100 percent predictive in excluding a fall in PaO₂ with exercise. Measurements below these thresholds could not be used reliably to predict which patients would develop worsening hypoxemia with exercise. Because of wide variability in reference values from eight different published studies for diffusing capacity, recommended criteria based on the percent predicted Dsb should be used with caution. We conclude that pulmonary function measurements cannot be used to predict exercise-induced hypoxemia in patients with COPD; however, the measurements may be useful in identifying patients whose condition is less severe who are unlikely to develop worsening hypoxemia with exercise.

Hypoxemia is an important and common complication in patients with chronic obstructive pulmonary disease (COPD). For patients with COPD who have significant resting hypoxemia, continuous oxygen supplementation has been shown to improve survival. With physical activity, arterial oxygenation in COPD may change in a variable fashion (increase, decrease, or remain constant). For patients with significant exercise-induced hypoxemia, oxygen supplementation during exercise may improve exercise endurance or capacity, although most studies which addressed this issue have not controlled for a possible placebo effect. The effect on survival of oxygen supplementation during exercise is unknown.

Measurements of arterial oxygenation during exercise are made frequently in COPD in order to evaluate complaints of dyspnea on exertion and to assess the need for supplemental oxygen during physical activities. In order to minimize the number of unnecessary exercise tests, appropriate criteria for selecting patients with COPD who are likely to develop worsening arterial oxygenation during exercise testing would be useful. We conducted this study to investigate whether commonly used measurements of pulmonary function could be used in COPD to predict the development of exercise-induced hypoxemia and to select patients for exercise testing.

MATERIALS AND METHODS

Patients and Pulmonary Function Tests

Forty consecutive patients with stable COPD entering a pulmonary rehabilitation program were studied. All patients underwent pulmonary function testing. Spirometry was performed according to recommendations of the American Thoracic Society's Snowbird Conference. Plethysmographic measurements of functional residual capacity (FRC) and airway resistance (Raw) were made by the method of DuBois and colleagues, using a constant-volume plethysmograph (W. E. Collins, Inc). Total lung capacity (TLC) and residual volume (RV) were calculated from the measured FRC in combination with spirometric measurements of expiratory reserve volume (ERV) and vital capacity (VC). Single-breath diffusing capacity for carbon monoxide (Dsb) was performed following the method of Ogilvie and co-workers, but with the following modifications: inspired vital capacity was corrected from ambient temperature and pressure, dry (ATPD) (rather than saturated) to standard temperature and pressure, dry (STPD), calculations of alveolar volume incorporated correction factors for anatomic as well as...
Table 1—General Characteristics, Pulmonary Function, and Exercise Tests

<table>
<thead>
<tr>
<th>Data</th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
<th>p†</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>21</td>
<td>9</td>
<td>10</td>
<td>...</td>
</tr>
<tr>
<td>Age, yr</td>
<td>67±11</td>
<td>64±8</td>
<td>66±7</td>
<td>NS</td>
</tr>
<tr>
<td>Height, cm</td>
<td>166.1±7.8</td>
<td>164.1±5.5</td>
<td>169.9±4.8</td>
<td>NS</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>65.7±14.7</td>
<td>59.8±10.2</td>
<td>71.0±19.2</td>
<td>NS</td>
</tr>
<tr>
<td>VC, L</td>
<td>2.42±0.86 (71)</td>
<td>2.34±0.50 (73)</td>
<td>2.40±0.69 (67)</td>
<td>NS</td>
</tr>
<tr>
<td>FVC, L</td>
<td>1.98±0.75 (59)</td>
<td>1.73±0.60 (54)</td>
<td>2.07±0.63 (58)</td>
<td>NS</td>
</tr>
<tr>
<td>FEV1, L</td>
<td>0.80±0.32 (34)</td>
<td>0.81±0.28 (35)</td>
<td>1.28±0.50 (51)</td>
<td>&lt;0.01 (0.05)</td>
</tr>
<tr>
<td>FEV1/FVC%</td>
<td>40±6</td>
<td>47±7</td>
<td>61±11</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>FEF25-75%, L/sec</td>
<td>0.30±0.13 (13)</td>
<td>0.36±0.15 (15)</td>
<td>0.79±0.45 (31)</td>
<td>&lt;0.001 (0.001)</td>
</tr>
<tr>
<td>TLC, L</td>
<td>6.98±1.30 (131)</td>
<td>7.19±1.64 (140)</td>
<td>6.19±1.10 (110)</td>
<td>NS (&lt;0.01)</td>
</tr>
<tr>
<td>FRC, L</td>
<td>5.61±0.93 (179)</td>
<td>5.44±1.71 (178)</td>
<td>4.44±1.30 (138)</td>
<td>NS (&lt;0.01)</td>
</tr>
<tr>
<td>RV, L</td>
<td>4.55±0.89 (218)</td>
<td>4.84±1.71 (243)</td>
<td>3.79±1.06 (173)</td>
<td>NS (&lt;0.05)</td>
</tr>
<tr>
<td>RV/TLC, percent</td>
<td>66±8</td>
<td>66±10</td>
<td>61±11</td>
<td>NS</td>
</tr>
<tr>
<td>Dsb, ml/min/mm Hg</td>
<td>11.0±3.5</td>
<td>14.3±5.9</td>
<td>21.0±6.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Raw, cm H2O/L/sec</td>
<td>4.0±1.7</td>
<td>4.4±1.4</td>
<td>3.8±1.1</td>
<td>NS</td>
</tr>
<tr>
<td>MVV, L/min</td>
<td>28±13</td>
<td>29±10</td>
<td>43±20</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>VO2max, L/min</td>
<td>0.80±0.27</td>
<td>0.83±0.27</td>
<td>1.14±0.47</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>VO2max, ml/kg/min</td>
<td>12.1±3.0</td>
<td>14.1±4.7</td>
<td>15.8±3.6</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>VEmax, L/min</td>
<td>30±10</td>
<td>31±6</td>
<td>43±15</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>PaO2, mm Hg</td>
<td>67±5</td>
<td>75±13</td>
<td>70±11</td>
<td>NS</td>
</tr>
<tr>
<td>Rest</td>
<td>56±8</td>
<td>74±12</td>
<td>81±13</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SaO2, percent</td>
<td>93.3±1.3</td>
<td>94.5±2.0</td>
<td>93.4±2.2</td>
<td>NS</td>
</tr>
<tr>
<td>Rest</td>
<td>97.1±3.7</td>
<td>93.6±2.1</td>
<td>94.5±1.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PaCO2, mm Hg</td>
<td>41±5</td>
<td>38±13</td>
<td>39±11</td>
<td>NS</td>
</tr>
<tr>
<td>Rest</td>
<td>48±10</td>
<td>42±5</td>
<td>42±5</td>
<td>NS</td>
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</tbody>
</table>

*Results are expressed as mean ± SD; numbers within parentheses are percent of predicted.
†ANOVA; results in parentheses are for percent predicted values. NS, not significant.

Instrument dead space; washout volume was 500 ml; and sample volumes were between 750 and 1,000 ml. Measurements of Dsb were performed in duplicate, with additional measurements if the first two differed by more than 10 percent. The mean value of all technically acceptable measurements was reported. Reference normal values were those of Morris and co-workers who used spirometric data and those of Goldman and Becklake for lung volumes. For Dsb, in addition to the predictive values of Miller and co-workers, which are most appropriate for our laboratory, we also calculated predicted values using seven other widely used prediction equations.

Exercise Tests

An incremental, symptom-limited exercise test to the maximal tolerable level was performed on a cycle ergometer with a modified chair seat. Arterial blood was sampled through a radial arterial catheter placed percutaneously. The electrocardiogram was monitored continuously, and blood pressure was taken manually at periodic intervals. Ear oximetric arterial oxygen saturation (SaO2) was monitored in some patients. Patients breathed through a low-resistance breathing valve (Hans Rudolph 2700) connected to a mixing chamber for expired gas measurements of airflow with a pneumotachograph (Charles Meriam, Co) and expired oxygen (Applied Electrochemistry, Inc) and carbon dioxide (Cobalt Codart) concentrations by gas analyzers. Measurements of expired gas were analyzed on-line by a computer using standard techniques and were averaged over 20-second intervals to calculate minute ventilation (VE, body temperature and pressure, saturated [BTPS]), oxygen uptake (VO2, STPD), and carbon dioxide elimination (VCO2, STPD).

Each patient rested until expired gas measurements were stable and a resting arterial blood sample was drawn for measurement of arterial oxygen pressure (PaO2), arterial carbon dioxide tension (PaCO2), and pH. The patient then pedaled at zero W for three minutes; the workload was then increased each minute by 12.5 W until the patient reached a symptom-limited maximum or the test was terminated due to oximetric SaO2 less than 85 percent, to ST-T

![Figure 1](https://example.com/figure1.png)

**Figure 1.** FEV1/FVC% vs change in PaO2 with exercise in 40 patients with COPD. Patients are grouped according to change in PaO2 with exercise: group 1 (diamonds) had more than 3 mm Hg decrease in PaO2, group 2 (triangles) had no change (±3 mm Hg) in PaO2, and group 3 (squares) had more than 3 mm Hg increase in PaO2.
wave depression or serious arrhythmias noted on the ECG or to an excessively elevated blood pressure (greater than 250 mm Hg systolic or 120 mm Hg diastolic pressure). Arterial blood was sampled at rest, every three minutes during exercise, and at the maximal exercise level.

RESULTS

Response to Exercise

In 39 of the 40 patients, maximum exercise was limited by the following symptoms: dyspnea in 23; dyspnea and muscle fatigue in 12; dyspnea and muscle cramps in two; and muscle fatigue in two. In one patient, exercise was stopped due to premature ventricular contractions and ischemic changes noted on the ECG. No patient was stopped prior to a symptom-limit because of arterial oxygen desaturation from the ear oximeter.

For the purposes of this study, a change in PaO₂ at maximal exercise greater than 3 mm Hg compared to the resting value was considered to be significant. This threshold was selected as being greater than two standard deviations of repeat measurements of PaO₂ in our laboratory.14 In the 40 patients, PaO₂ at maximum exercise decreased more than 3 mm Hg in 21 patients (group 1), did not change (± 3 mm Hg) in nine patients (group 2), and increased more than 3 mm Hg in ten patients (group 3).

Pulmonary Function and Exercise Tests

The general characteristics, pulmonary function, results of exercise tests, and arterial blood gas levels are presented in Table 1 for each of the three groups of patients. Comparing the three groups by analysis of variance (ANOVA), there were significant differences in FEV₁, FEV₁/FVC, the mean forced expiratory flow during the middle half of the FVC (FEF25-75%), TLC (percent predicted), FRC (percent predicted), RV (percent predicted), Dsb, maximal voluntary ventilation (MVV), VO₂max, V̇emax, exercise PaO₂, and exercise SaO₂. There were no significant differences in age, height, weight, FVC, RV, FRC, TLC, resting PaO₂, resting SaO₂, resting PaCO₂, or exercise PaCO₂.

Further statistical analysis by Student’s t-tests demonstrated that the significant intergroup effects on pulmonary function were related to significantly less severe obstructive pulmonary disease in group 3. Except for the lower FEV₁/FVC noted in group 1, there were no significant differences between groups 1 and 2.

Stepwise multiple regression to predict change in PaO₂ with exercise was performed using all parameters significantly different between groups by ANOVA. This analysis indicated that FEV₁/FVC and Dsb contributed most significantly to the variance associated with change in PaO₂ with exercise (ΔPaO₂ [in millimeters of mercury] = 0.55 FEV₁/FVC + 0.45 Dsb − 35.23; r = 0.82; SEE = 6.23).

The relationship between change in PaO₂ with exercise and FEV₁/FVC is presented in Figure 1. A value for FEV₁/FVC of less than 0.50 was found in all 21 patients with exercise-induced hypoxemia (sensitivity, 100 percent) but also in seven out of 19 patients whose PaO₂ did not fall with exercise (specificity, 63 percent). Positive predictive value was 75 percent (21 out of 28 patients) for FEV₁/FVC of less than 0.50. Negative predictive value was 100 percent (12 out of 12 patients with FEV₁/FVC of 0.50 or more did not develop exercise-induced hypoxemia).

The relationship between change in PaO₂ with exercise and Dsb is presented in Figure 2 for the 37 patients with measurements of Dsb. (Two patients were unable to hold their breath long enough, and reproducible measurements could not be obtained in one patient). A Dsb of less than 20 ml/min/mm Hg was found in all 20 patients with exercise-induced hypoxemia (sensitivity, 100 percent) but also in 11 out of 17 patients whose PaO₂ did not fall with exercise (specificity, 35 percent). The positive predictive value of a Dsb of less than 20 ml/min/mm Hg was 65 percent (20 out of 31 patients). The negative predictive value was 100 percent (six out of six patients) for a Dsb of 20 ml/min/mm Hg or more.

Using the FEV₁/FVC and Dsb together did not provide any additional diagnostic accuracy compared to the FEV₁/FVC alone. Twenty out of 27 patients with an FEV₁/FVC ratio of less than 0.50 and a Dsb of less than 20 ml/min/mm Hg had exercise-induced hypoxemia (positive predictive value, 74 percent). Six out of six patients with an FEV₁/FVC of 0.50 or more and a Dsb of 20 ml/min/mm Hg or more did not have exercise-induced hypoxemia (negative predictive value, 100 percent).

![Figure 2. Single-breath diffusing capacity vs change in PaO₂ with exercise in 37 patients with COPD. Patients are grouped according to change in PaO₂ with exercise (as in Fig 1).](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21575/ on 06/05/2017)
Table 2—Variability in Eight Published Reference Sources for Predicting Dsb in 37 Patients with COPD

<table>
<thead>
<tr>
<th>Reference</th>
<th>Mean Dsb, percent of predicted (± SD)</th>
<th>Threshold Value, percent of predicted*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kanner and Morris14</td>
<td>79 ± 33</td>
<td>93</td>
</tr>
<tr>
<td>Miller et al19 (nonsmoker)</td>
<td>61 ± 27</td>
<td>70</td>
</tr>
<tr>
<td>Crapo and Morris14</td>
<td>53 ± 23</td>
<td>58</td>
</tr>
<tr>
<td>Cotes and Hall17</td>
<td>55 ± 22</td>
<td>70</td>
</tr>
<tr>
<td>Quanjer18</td>
<td>60 ± 25</td>
<td>67</td>
</tr>
<tr>
<td>Cotes19</td>
<td>62 ± 26</td>
<td>69</td>
</tr>
<tr>
<td>Viljanen et al20</td>
<td>66 ± 30</td>
<td>74</td>
</tr>
<tr>
<td>Burrows et al21</td>
<td>75 ± 27</td>
<td>90</td>
</tr>
</tbody>
</table>

*Value above which patients did not develop exercise-induced hypoxemia.

percent). Four patients with FEV1/FVC of 0.50 or more had a Dsb of less than 20 ml/min/mm Hg; none of these patients had exercise-induced hypoxemia.

Twelve of the 40 patients had PaO2 values of 55 mm Hg or less during maximum exercise, a level generally considered "clinically significant." All had measurements of Dsb less than 20 ml/min/mm Hg and FEV1/FVC less than 0.50 (sensitivity, 100 percent); however, 12 out of 28 patients with an exercise PaO2 of more than 55 mm Hg had an FEV1/FVC of 0.50 or more (specificity, 43 percent). For Dsb, six out of 26 patients with an exercise PaO2 of more than 55 mm Hg had a Dsb of 20 ml/min/mm Hg or more (specificity, 23 percent).

Variability in Predictions of Diffusing Capacity

Because of the widespread use of different reference studies for Dsb, predicted normal values were calculated from eight different published prediction equations.12,15-21 These eight different equations produced markedly different normal values for these patients. The results presented in Table 2 demonstrate the wide range of mean percent predicted Dsb for the 37 patients in whom these measurements were made. In addition, the threshold value above which patients did not develop exercise-induced hypoxemia in this study ranged from 58 to 93 percent of predicted in these eight published studies (Table 2). In examining the predicted values for individual patients, it was clear that some of the eight prediction equations produced variable results compared to other equations, rather than just consistently higher or lower values.

Discussion

Hypoxemia which worsens with exercise occurs variably in COPD.9,25 Previous studies have demonstrated that patients with COPD who have resting hypoxemia have increased morbidity and mortality1,25 but have not specifically examined the course in patients who have hypoxemia only with exercise. Other studies and the experience of pulmonary rehabilitation programs9,97 have shown that exercise training and increased physical activity can result in significant physiologic and psychologic benefits for patients with COPD; however, the occurrence of worsening hypoxemia with physical activity or exercise in such patients may contribute to exercise limitation and symptoms and poses a threat to safe exercise training and rehabilitation.

The availability of convenient portable systems for continuous oxygen delivery makes oxygen therapy during exercise practical. Because oxygen therapy is expensive and cumbersome to use, appropriate selection of patients is important; however, accurate assessment of oxygenation during exercise is a significant laboratory procedure. Obtaining samples of blood from single arterial punctures immediately after exercise may produce blood gas levels significantly different from those obtained during exercise.96 Therefore, indwelling arterial catheters are used commonly to obtain samples of arterial blood during exercise. Newer noninvasive techniques, such as ear oximetry, may not be sufficiently accurate or sensitive to changes in arterial oxygenation during exercise to provide reliable assessment for oxygen therapy (95 percent confidence limits are ± 4 to 5 percent of true arterial oxygen saturation and ± 3 percent for change in saturation).9,99 Therefore, it would be useful to identify criteria which would select for exercise testing, with invasive blood sampling only those patients more likely to develop worsening arterial oxygenation during exercise.

The results of this study indicate that resting measurements of pulmonary function or arterial blood gas levels could not reliably predict exercise-induced hypoxemia in these patients with COPD but could be used to identify patients unlikely to develop worsening hypoxemia during exercise and so, therefore, would not need to undergo exercise testing. Patients with measurements of FEV1/FVC of 0.50 or more and Dsb of 20 ml/min/mm Hg or more were unlikely to develop exercise-induced hypoxemia (negative predictive values, 100 percent); however, measurements below these threshold values could not reliably predict worsening hypoxemia with exercise (positive predictive values, 75 percent and 65 percent, respectively).

These results might appear to contradict those of Owens and co-workers,9 who concluded that diffusing capacity and FEV1 could be useful in identifying patients with COPD who are likely to develop desaturation during exercise; however, careful examination of their data reveals little discrepancy. Owens and co-workers9 found that thresholds of 55 percent of predicted for both diffusing capacity and FEV1 could be used to select patients with an increased likelihood of desaturation with exercise. Above these thresholds, desaturation was unlikely to occur (as in our study);
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however, below these thresholds, 68 percent (Dsb) and 46 percent (FEV1) of the patients did develop desaturation. Also, Owens and co-workers divided patients into only two groups, those who developed desaturation and those who did not. The results of our study would suggest that the differences in pulmonary function between these two groups are due to those patients who develop an increase in saturation with exercise. Pulmonary function tests in those patients whose oxygen saturation did not change with exercise were not different from those who developed desaturation. In analyzing our data in a fashion similar to Owens and co-workers, combining groups 2 (no change in PaO2) and 3 (increase in PaO2) vs group 1 (decrease in PaO2) produced statistically significant differences for most of the pulmonary function parameters found to be significantly different only because of less severe abnormalities in group 3 alone.

One significant difference between our study and that by Owens and co-workers is in the use of criteria for diffusing capacity based on percentage of normal predicted values. As demonstrated in Table 2, predicted reference values for Dsb differ significantly between various studies. This could lead to confusion and misinterpretation of results depending on the method and reference values used. For instance, the Intermountain Thoracic Society's reference values for diffusing capacity cited by Owens and co-workers produced the lowest predicted values in 21 of 40 patients in our study. Using these values in our patients, we would have identified a threshold value for Dsb of 93 percent of predicted to select patients who did not need to undergo exercise testing to detect exercise-induced hypoxemia (vs the 55 percent of predicted suggested by Owens and co-workers from their measurements and patients). On the other hand, using the reference values of Miller and co-workers which are most appropriate for normal subjects tested in our laboratory, we would have selected 70 percent of predicted as the appropriate threshold.

Therefore, given the marked variability of existing published predicted values for Dsb, we believe that recommendations of threshold values for clinical decision-making based on the percent predicted Dsb derived in one laboratory should be used with caution in other laboratories. Better standardization of testing methods and attention to selecting appropriate reference values are necessary before specific threshold values of Dsb expressed as percent predicted can be used universally.

The development of exercise-induced hypoxemia in certain patients with COPD which is not directly related to the severity of the disease as assessed by standard pulmonary function tests raises interesting questions about the possible mechanisms of such changes. Normal individuals do not develop exercise-induced hypoxemia, except possibly at very high exercise levels. Patients with interstitial pulmonary disease may develop exercise-induced hypoxemia early in the course of their disease, and this test has been suggested as an early marker for these diseases; however, patients with moderate to severe COPD may develop hypoxic exercise-induced hypoxemia in an unpredictable fashion. On the other hand, patients with less severe COPD may develop improved arterial oxygenation with exercise.

These observations suggest that the determinants of changes in arterial oxygenation during exercise in patients with COPD are multiple. Using the multiple inert gas elimination technique in a group of patients with COPD, Wagner and co-workers found that the change in PaO2 with exercise could be explained by the effect of a decrease in mixed-venous oxygen pressure (PvO2) through areas of low ventilation/perfusion (VA/Q) ratios and shunt. In a group of patients with severe COPD, Dantzker and D'Alonzo found that exercise-induced hypoxemia was due to both hypoventilation and the influence of a decrease in PvO2 on areas of low VA/Q and shunt. Thus, the variability of changes in arterial oxygenation with exercise in COPD may be due to the complex interaction of several factors including alveolar hypoventilation and the effect of a decrease in PvO2 through areas of low VA/Q and shunt. The latter effect may be significantly influenced by the degree of right heart failure.

It should be emphasized that exercise-induced hypoxemia in patients with moderate to severe COPD may occur with modest exercise which corresponds to levels of activity reached during routine daily activities and may, therefore, contribute significantly to the development of exercise limitation, pulmonary hypertension, and right heart failure in these patients. The availability of convenient portable systems for delivery of ambulatory oxygen therapy has stimulated interest in detecting significant exercise-induced hypoxemia and determining the appropriate amount of oxygen necessary to provide an acceptable level of arterial oxygenation. This study was not designed to detect only "clinically significant" hypoxemia with exercise, but rather to identify patients whose arterial oxygenation was likely to change from resting levels with exercise. Only 12 of the 21 patients in this study with exercise-induced hypoxemia had an exercise PaO2 of 55 mm Hg or less, a level generally used to justify supplemental oxygen therapy. Although the pulmonary function criteria used in this study were sensitive in detecting this level of exercise hypoxemia, they were not specific.

On the basis of this study, we conclude that measurements of pulmonary function cannot be used to predict reliably exercise-induced hypoxemia in COPD; however, they may be useful in identifying patients un-
likely to develop worsening hypoxemia with exercise and, therefore, avoiding unnecessary exercise tests. In this study, all patients with COPD whose FEV/FVC was 0.50 or more or whose Dsb was 20 ml/min/mm Hg or more did not demonstrate a significant fall in PaO₂ with exercise. Until better standardization of testing methods and appropriate selection of normal reference values are achieved, caution should be exercised in using diagnostic criteria for diffusing capacity expressed as percent predicted for clinical decision-making in different laboratories.

REFERENCES


6 Fulmer JD, Snider GL. ACCP-NHLBI national conference on oxygen therapy. Chest 1984; 86:234-47


26 Hughes RL, Davison R. Limitations of exercise reconditioning in COLD. Chest 1983; 83:241-49


28 Ries AL, Fedullo PF, Clausen JL. Rapid changes in arterial blood gas levels after exercise in pulmonary patients. Chest 1983; 83: 454-56


30 Jones NL, McHary GJR, Naimark A, Campbell EJM. Physiological dead space and alveolararterial gas pressure differences during exercise. Clin Sci 1966; 31:19-29


