Comparison of Oxygen Saturation by Pulse Oximetry and Co-oximetry During Exercise Testing in Patients With COPD*

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Introduction: Measurement of oxygen saturation by pulse oximetry (SpO₂) is frequently performed during exercise testing of patients with COPD to monitor for hypoxemia. The purpose of this study was to assess the accuracy and precision of pulse oximetry during exercise. We hypothesized that the SpO₂ would more closely reflect oxygen saturation as measured by co-oximetry (SaO₂) when it was corrected for carboxyhemoglobin (COHb). We also hypothesized that SpO₂ would more closely reflect SaO₂ when the pulse rate by oximeter was equivalent to the heart rate by ECG. Finally, we hypothesized that SpO₂ would be a better measure of SaO₂ at maximal workloads than at rest or submaximal workloads.

Methods: Eight white men with severe COPD (mean ± SD FEV₁, 0.91 ± 0.37) underwent progressive, symptom-limited exercise testing by cycle ergometry. SaO₂ was measured from arterial blood at each workload using a co-oximeter. SpO₂ and pulse rate were obtained by a pulse oximeter (Ohmeda 3700). Heart rate was continuously monitored by ECG.

Results: Reliable oximetric values as determined by a dicrotic notch in each waveform and adequate signal intensity were obtained in all eight patients. SpO₂ was a moderately accurate measure of SaO₂ (bias, 1.7%; precision, 2.9). The bias actually increased (4.1%) when SpO₂ was corrected for COHb. Accuracy of SpO₂ was not improved when pulse rate by oximeter and heart rate by ECG were equivalent, nor was the accuracy improved at maximal workloads relative to submaximal workloads during the exercise test.

Conclusion: Oxygen saturation as measured by pulse oximetry (SpO₂) in patients with COPD undergoing exercise testing is not sufficiently accurate to replace SaO₂ as the gold standard for oxygen saturation.

(CHEST 1996; 109:1151-55)

Key Words: chronic obstructive pulmonary disease; co-oximetry; exercise; pulse oximetry

Abbreviations: COHb=carboxyhemoglobin; SaO₂=oxygen saturation measured by co-oximetry; SpO₂=oxygen saturation measured by pulse oximetry; Sp*O₂=oxygen saturation measured by pulse oximetry and corrected for COHb

Pulse oximetry is used in many clinical and research settings as an indirect measurement of oxygen saturation.1-7 Oxygen saturation as measured by the pulse oximeter (SpO₂) is frequently performed during exercise testing of the patient with pulmonary disease to monitor for hypoxemia. As a noninvasive tool, it seems reasonable to use the pulse oximeter to spare the patient arterial punctures. Unfortunately, this noninvasive measure of oxygen saturation may not accurately reflect arterial saturation.2,3,5,8 We performed the present study to determine if there were certain conditions in which the SpO₂ improved accuracy and precision during exercise. We hypothesized that during exercise, the SpO₂, corrected for carboxyhemoglobin (COHb), would reflect more accurately the oxygen saturation as measured by co-oximetry (SaO₂) than would the uncorrected SpO₂. We also hypothesized that SpO₂ would reflect the SaO₂ more closely when the pulse rate by oximeter was equivalent to the heart rate by ECG. Finally, we hypothesized that SpO₂ would reflect SaO₂ best at maximal workloads relative to submaximal workloads.

The specific objectives of this study involving patients with COPD undergoing an exercise test were as follows: (1) to compare SpO₂ as measured by pulse oximetry with SaO₂ as measured by co-oximetry from arterial blood; (2) to compare SpO₂ corrected for COHb with SaO₂; (3) to compare SpO₂ uncorrected and corrected for COHb with SaO₂ when the pulse

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rate by oximetry was equivalent to the heart rate by ECG; and (4) to compare \( \text{SpO}_2 \) with \( \text{SaO}_2 \) at maximal workloads relative to submaximal workloads.

**MATERIALS AND METHODS**

**Patients**

Patients in stable condition with severe COPD, recruited from a concomitantly run study evaluating the effects of pentoxifylline on oxygenation and exercise tolerance,\(^9\) participated in the study. All of the participants signed an informed consent approved by our Institutional Review Board. The patients were all white men recruited from the Long Beach Veterans Administration Medical Center pulmonary outpatient population. Exercise tolerance was limited by dyspnea in all patients. In addition, a screening exercise test showed that all of the participants had less ventilatory reserve than cardiac reserve at their symptom-limited maximum.

Prior to the study, each patient underwent complete pulmonary function testing, including spirometry before and after bronchodilators, lung volumes, diffusing capacity, specific conductance, and airways resistance. Participants had stable COPD with \( \text{FEV}_1 \) less than 1.50 L and \( \text{FEV}_1/\text{FVC} \) less than 50%. They continued to receive their regular pulmonary medications.

**Exercise Testing**

Maximal exercise testing was performed on a cycle ergometer in an incremental fashion as previously described from this laboratory.\(^10\) Workload was increased by 5 to 15 W every minute, depending on the patient’s pulmonary function test results and the results of any previous exercise tests. A Borg score for dyspnea was obtained at the end of each minute interval. Metabolic variables were analyzed with a metabolic measurement cart (Horizon 4400 Metabolic Measurement Cart; Sensormedics; Yorba Linda, Calif).

**Assessment of Oxygen Saturation and Heart Rate**

An arterial catheter was placed in the radial artery following confirmation of collateral vessel flow by a modified Allen’s test. \( \text{SaO}_2 \) was measured from arterial blood collected at baseline and at each workload using a co-oximeter (IL 482 Co-oximeter; Instrumentation Laboratory; Lexington, Mass). Air bubbles, if present, were immediately expelled from the sample; the sample was sealed to maintain anaerobic conditions and packed in an iced container until evaluation immediately after the exercise test. \( \text{SpO}_2 \) and pulse rate were obtained using a pulse oximeter (Ohmeda 3700; Ohmeda; Louisville, Colo). Care was taken to monitor both the signal intensity and the presence of a dicrotic notch that reflect adequate pulse acquisition. This pulse oximeter unit is a self-calibrating unit.

The finger probe for the unit was placed on the index finger of the hand opposite the arterial line. Heart rate was continuously displayed by ECG (Diascope DS 521; Simonsen & Well; Denmark). Heart rate, pulse rate, \( \text{SpO}_2 \), and \( \text{SaO}_2 \) were recorded in the last 15 s of each 1-min exercise period.

**Correction of COHb**

We corrected all of the \( \text{SpO}_2 \) values according to the equation proposed by Barker and Tremper as discussed by Raemer et al\(^11\):

\[
\text{Sp}\text{*O}_2=\text{SpO}_2-0.9(\text{COHb})
\]

where \( \text{Sp}\text{*O}_2 \) is the \( \text{SpO}_2 \) corrected for COHb

**Statistical Analysis**

The pulse oximeter, as a reflection of the oxygen saturation, was assessed by calculating the bias and precision of the \( \text{SpO}_2 \) relative to the \( \text{SaO}_2 \). Bias is the mean difference between the \( \text{SaO}_2 \) and the \( \text{SpO}_2 \). Precision is the SD of the bias. Data are expressed as mean (±SD) unless otherwise indicated. In addition, the mean difference between \( \text{SpO}_2 \) and \( \text{SaO}_2 \) values was assessed by a paired student \( t \) test, and \( p \) values < 0.05 were considered to be statistically significant.

**RESULTS**

All of the eight participants completed the evaluation. Reliable oximetric values as determined by a dicrotic notch in each waveform and adequate signal intensity were obtained in all subjects. Table 1 shows pulmonary function data for all of the eight subjects. Three of the eight patients were current smokers (>1 pack per day).

The \( \text{SaO}_2 \) by co-oximetry did not closely reflect the \( \text{SpO}_2 \) when all the points were considered (Fig 1 and Table 2). The mean \( \text{SaO}_2 \) (91.5±3.5%) was significantly higher than the mean \( \text{SpO}_2 \) (89.8±3.7%) (\( p<0.001 \)). The bias of the pulse oximeter measurement relative to the \( \text{SaO}_2 \) was 1.7% while the precision was 2.9%.

The bias actually increased when the \( \text{SpO}_2 \) was corrected for the COHb levels (Fig 2 and Table 2). The \( \text{SaO}_2 \) (91.5±3.5) was significantly higher than the \( \text{Sp}\text{*O}_2 \) (87.4±3.5) (\( p<0.001 \)). From Figure 2, in which \( \text{SaO}_2 \) is plotted vs \( \text{Sp}\text{*O}_2 \), all but one of the points lie

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**Table 1—Mean (±SD) Values During Initial Screen**

<table>
<thead>
<tr>
<th>Patient Data</th>
<th>Mean±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>63.2±9.6</td>
</tr>
<tr>
<td>( \text{FEV}_1 ), L</td>
<td>0.91±0.37</td>
</tr>
<tr>
<td>( \text{FVC} ), L</td>
<td>2.75±0.29</td>
</tr>
<tr>
<td>( \text{Deo}^* ), mL/min/mm Hg</td>
<td>9.85±5.1</td>
</tr>
</tbody>
</table>

\*\( \text{Deo}^* \)=diffusing capacity of the lung for carbon monoxide.

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**Figure 1.** \( \text{SaO}_2 \) vs \( \text{SpO}_2 \) for all exercise data points (n=49). Solid line corresponds to line of identity and dotted line is determined by linear regression analysis.
above the line of identity. Despite the increase in bias, the precision improved from 2.9 to 2.2%.

To examine the saturation trends for each patient, we calculated the bias and precision for each patient individually, where bias is equal to the mean saturation difference (SaO₂ - SpO₂) and precision is the SD of the bias. The individual values for the bias were as follows: -2.80, -2.20, 0.87, 1.75, 2.61, 2.92, 3.60, and 7.37 (range, -2.80 to 7.37), while the individual values for the precision were as follows: 0.36, 0.58, 0.83, 0.89, 1.16, 1.46, 1.81, and 3.22 (range, 0.36 to 3.22). Correcting for the presence of COHb resulted in a general increase of values for mean bias (0.32 to 5.3). Despite the wide range of individual values, these values were equally distributed over the range.

Next, the relationship of the SpO₂ and the SaO₂ was assessed when pulse rate by oximetry was equivalent to heart rate by ECG. The accuracy of the SpO₂ measurement compared with the SaO₂ did not improve when the pulse and heart rates were equivalent, relative to when they were not equivalent. This conclusion held whether the SaO₂ was uncorrected (Fig 3) or corrected (Fig 4) for the COHb level.

Finally, the SpO₂ was compared with the SaO₂ for all exercise data points at the two highest workloads relative to all lower workloads. The bias and the precision of the SpO₂ relative to the SaO₂ were not significantly different, although both measures showed a trend toward improvement (Table 3).

**Discussion**

The present study demonstrates that the SpO₂ does not closely reflect the SaO₂ in patients with COPD undergoing exercise tests. In addition, the bias of the SpO₂ is not improved when the SpO₂ is corrected for COHb, when the pulse and heart rates are equivalent by oximetry and by ECG, respectively, or when exercising at higher workloads relative to lower workloads.

The results of the present study are similar to those of previous investigators who have shown that most pulse oximeters are accurate to ±4% in normal patients when SaO₂ is more than 70%. Other investigators have studied the reliability of pulse oximetry compared with co-oximetry on arterial blood by comparing SpO₂ with SaO₂ at end points of exercise, at random points in subjects at rest, and by adjusted, inhaled fraction of inspired oxygen concentrations.

One possible explanation for the relatively poor correlations in the present study is that our oximeter was particularly inaccurate. It has been reported that the unit (Ohmeda) that was used in this study is comparable in accuracy to other oximeters. The unit itself is self-calibrating. In addition, we obtained adequate signal intensity and a diacritic notch for all of the data points for the exercising patient, suggesting adequate pulse acquisition. To verify that our oximeter was comparable to other oximeters, we compared the SpO₂ obtained with the oximeter used in the present study to that obtained with two other oximeters (namely another Ohmeda 3700 unit and a Nellcor Model N-20 device). Using a sample of 12 patients with moderate to severe COPD from our outpatient clinic, agreement with the finger probe on either index finger in the resting and exercising state was within ±1%.

Since pulse oximeters using two wavelengths such as the one used in the present study cannot differentiate between COHb and oxyhemoglobin, we hypothesized that the SpO₂ would more closely reflect the SaO₂ if the SpO₂ were corrected for the COHb level. This would be particularly likely to happen in individuals who were current smokers. We used the equation proposed by Barker and Tremper as discussed by Raemer et al that assumes that the pulse oximeter “sees” COHb as if it were 90% oxyhemoglobin. Correcting the SpO₂ for COHb results in values lower than the measured SpO₂. Unfortunately, the accuracy of SpO₂ did not improve after this correction.

We also hypothesized that the SpO₂ measurements would more closely reflect the SaO₂ when the pulse

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**Table 2—Pulse Oximetry and Co-oximetry Data (Corrected and Uncorrected for COHb)**

<table>
<thead>
<tr>
<th></th>
<th>Bias (SaO₂ - SpO₂)</th>
<th>Precision (SD of Bias)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uncorrected for COHb</td>
<td>1.7</td>
<td>2.90</td>
</tr>
<tr>
<td>Corrected for COHb</td>
<td>4.1</td>
<td>2.23</td>
</tr>
</tbody>
</table>

*Mean COHb for all subjects (percent): 3.05±2.15.
rate from the oximeter was equivalent to the heart rate from the ECG. The equivalence of the pulses should indicate that the oximeter was detecting perfusion to the finger. Previous investigators have suggested that the pulse oximeter is more accurate when the rates were equivalent, but to our knowledge, no systematic analyses have been made. There is no evidence from the present study to suggest that the \( \text{SpO}_2 \) more accurately reflects the \( \text{SaO}_2 \) when the rates from the oximeter and ECG are equivalent (Fig 3 and Fig 4). The pulse oximeter (Ohmeda) displays heart rate data on a rolling average over a 6- to 12-s fixed time period. Since both the heart and pulse rates were measured in the last 15 s of each 1-min exercise period, there should not be a discrepancy between pulse rate by oximetry and heart rate by ECG due to time lag.

We also hypothesized that the \( \text{SpO}_2 \) would more accurately reflect the \( \text{SaO}_2 \) at higher workloads than at rest or lower workloads because the cardiac output would be higher, resulting in improved pulse acquisition by the finger oximeter. In a study analyzing the accuracy of pulse oximetry in healthy male subjects, the bias, or mean difference between the \( \text{SaO}_2 \) and \( \text{SpO}_2 \), was not significant at the highest workload, while differences at rest and at the lowest workload were significant, suggesting improved pulse acquisition at the higher workloads. The difference in findings between the latter study and the current one may lie in the differing patient populations. Perhaps the patients in the current study who were limited by ventilatory factors could not reach the higher cardiac outputs with consequent improved pulse acquisition attained by the younger, healthy subjects in the study. Yelderman and New, in their review on pulse oximetry, have suggested certain conditions that may significantly reduce finger pulse amplitude, namely hypotension, hypothermia, or the use of vasoconstrictor medications. In addition, cardiopulmonary bypass significantly reduces pulse amplitude and hypoperfusion. We found no significant difference in bias or precision when we compared the last two workloads (maximal) with the other workloads (submaximal) in this population of patients with COPD. It is not likely that tight grasping of the handlebars by the subject at peak workload would affect the data as pulse acquisition was deemed adequate as already noted.

Motion artifact did not appear to affect adversely the accuracy of the pulse oximeter relative to the co-oximeter. The probe for the pulse oximeter was relatively immobile and was located on the finger of a hand that was secured in position by the handlebars of the cycle. In addition, it was located on the finger of the hand opposite the arterial line. These measures of unit quality, as noted by the manufacturer (Ohmeda), suggest adequate pulse acquisition. The waveforms we

![Figure 3](image_url)

**Figure 3:** \( \text{SaO}_2 - \text{SpO}_2 \) (uncorrected for COHb) vs (ECG rate–pulse rate). No relationship exists between rate differences and saturation differences.

![Figure 4](image_url)

**Figure 4:** \( \text{SaO}_2 - \text{SpO}_2 \) (corrected for COHb) vs (ECG rate–pulse rate). There is still no relationship between the rate and saturation differences even when \( \text{SpO}_2 \) is corrected for the presence of COHb.

<table>
<thead>
<tr>
<th>( \text{SaO}_2 ) at maximal workloads relative to submaximal workloads</th>
<th>Bias ( (\text{SaO}_2 - \text{SpO}_2) )</th>
<th>Precision (SD of Bias)</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \text{SO}_2 ) at last 2 workloads (maximal)</td>
<td>2.1</td>
<td>3.04</td>
</tr>
<tr>
<td>( \text{SO}_2 ) at all other workloads (submaximal)</td>
<td>1.5</td>
<td>2.20</td>
</tr>
<tr>
<td>( p ) value</td>
<td>( &gt;0.05 )</td>
<td>( &gt;0.05 )</td>
</tr>
</tbody>
</table>

**Table 3—Oxygen Saturation (\( \text{SO}_2 \)) at Maximal Workloads Relative to Submaximal Workloads**
obtained from our patient population were not reflective of waveforms affected by motion artifact.

We recruited patients for this study from a concomitantly run study assessing the effects of pentoxifylline. Although some of the participants in this study were receiving the medication at the time of the exercise testing, it is not likely that the drug affected the relationship between the SaO2 and SpO2. There were no differences in exercise parameters in patients receiving either placebo or pentoxifylline.

In conclusion, SpO2 in patients with COPD undergoing exercise testing is not of sufficient accuracy to replace the gold standard SaO2 measured by co-oximetry on arterial blood. Accuracy and precision, as determined by bias and SD, respectively, are not improved when oxygen saturation is corrected for COHb, nor are they improved when heart and pulse rates are equivalent. Finally, measurement of SpO2 is not more accurate in exercising COPD patients relative to the SaO2 at maximal workloads compared with submaximal workloads.

REFERENCES