Validation of Pulse Oximetry During Progressive Normobaric Hypoxia Utilizing a Portable Chamber

Jon C. Kolb1, Peter Farran2, Stephen R. Norris1,3, David Smith1, and Joachim Mester4

Catalogue Data

Key words: oxygen saturation, co-oximetry, altitude
Mots-clés: saturation d’oxygène, co-oxymétrie, altitude

Abstract/Résumé
Validation of pulse oximetry in commercially available normobaric hypoxic chambers (NHC) has not been previously reported. The present study examined the validity of pulse oximetry (SpO2) against direct measurements of arterial oxygen saturation (SaO2) via co-oximetry (AVOXimeter 4000) in 13 young adults age 21.3 ± 0.6 years. Over a period of 2.5 hrs, the inspired fraction of oxygen inside a NHC (Hypoxico, Inc.) was progressively reduced from 20.9% to 11.5%. Measurements of SaO2 at baseline and at 15, 30, 60, 90, 120, and 150 min during the hypoxic exposures were compared with SpO2 estimates of oxygen saturation (Nellcor 295) using reflectance (RS-10, temporal) and transmission (D-25, finger) sensors. Regression analysis and methods for assessing agreement (bias, b; precision, p) of SaO2 with SpO2 were similar (R2 = 0.92, 0.89; b = 0.016, –0.47; p = 2.47, 3.03; RS-10 and D-25, respectively). When SaO2 < 85%, RS-10 had greater validity than D-25 (R2 = 0.73, 0.56; b = 1.38, 1.13; p = 2.72, 4.34; RS-10 and D-25, respectively). In light of these findings, caution should be exercised when monitoring individuals with pulse oximetry during desaturation episodes below 85%. When employing frequent NHC exposures, a priori validation of SpO2 utilized to assess blood oxygen status appears warranted.

1Faculty of Kinesiology, Univ. of Calgary, 2500 University Dr. NW, Calgary, AB, T2T 5C7; 2Alberta Children’s Hospital, 1820 Richmond Rd SW, Calgary, AB, T2N 1N4; 3Canadian Sport Centre–Calgary, Univ. of Calgary, Calgary, AB; 4Institute of Training & Movement Science, German Sport Univ., Carl-Diem-Weg 6, Cologne, Germany, D-50927.
Il n’y a pas d’études publiées concernant la validation de l’oxymétrie pulsée dans les chambres hypoxiques et normobariques (NHC) disponibles commercialement. L’étude actuelle analyse la validité de l’oxymétrie pulsée (SpO₂) par rapport aux mesures directes de la saturation artérielle d’oxygène obtenues par co-oxymétrie (AVOXimeter 4000) chez 13 jeunes adultes 21,3 ± 0,6 ans. La fraction d’oxygène inspiré en 2,5 h dans la NHC (Hypoxico inc.) est graduellement réduite de 20,9% à 11,5%. Les mesures de SaO₂ contrôle et 15ᵉ, 30ᵉ, 60ᵉ, 90ᵉ 120ᵉ et 150ᵉ minute pendant l’exposition hypoxique sont comparées aux estimations de la saturation d’oxygène (Nellcor 295) obtenues par des capteurs de réflectance (RS-10, temporal) et de transmission (D-25, doigt). L’analyse de régression et les méthodes de vérification de la concordance (biais, b; précision, p) entre les valeurs de SaO₂ et de SpO₂ donnent des résultats semblables (R² = 0,92 et 0,89; b = 0,016 et – 0,47; p = 2,47 et 3,03; RS-10 et D-25, respectivement). Quand SaO₂ est < 85%, RS-10 a plus de validité que D-25 (R² = 0,73 et 0,56; b = 1,38 et 1,13; p = 2,72 et 4,34; RS-10 et D-25, respectivement). Compte tenu de ces résultats, il faut faire preuve de circonspection lors du monitrage des individus sous oxymétrie pulsée au cours de séances de désaturation en dessous de 85%. Dans le cas de nombreuses expositions à la NHC, il est opportun de valider au préalable les mesures de SpO₂ pour établir la concentration sanguine d’oxygène.

Introduction

The state of oxygenation is crucial in understanding patient health status, the hypoxia associated with high altitude, or the impact of high intensity exercise. Pulse oximetry provides a simple noninvasive method of estimating the percentage of hemoglobin that is saturated with oxygen (SpO₂), and thus is a valuable diagnostic tool in monitoring subjects or patients during desaturation episodes. Technological advances over the past 25 years, and the relative ease of operation combined with continuous data output, have made pulse oximetry the minimal standard of care for patient monitoring during anesthesia as well as an essential tool in the practice of emergency medicine (Sinex, 1999; Tremper and Barker, 1989). Furthermore, there is a growing interest in using pulse oximeters to detect levels of hypoxemia during intensive exercise (Benoit et al., 1997; Martin et al., 1992; Yamaya et al., 2002) and to monitor the hypoxia associated with high altitude (Hussain et al., 2001; Roach et al., 1998).

Recently endurance athletes and high altitude climbers have gained access to commercially available portable normobaric hypoxic chambers (NHC), which are used to stimulate physiological changes similar to those observed during acclimatization to altitude or experimental acclimation to hypobaric hypoxia via decompression chambers (Powell and Garcia, 2000). In most cases the intended use of the portable normobaric hypoxic equipment is to enhance athletic performance or pre-acclimatize mountaineers to the rigor of high altitude expeditions. Pulse oximetry has been used to monitor oxygen saturation during NHC exposures (Kolb et al., 2001; Townsend et al., 2002). While pulse oximetry has become a generally acceptable method for detecting and quantifying hypoxemia, numerous reports in the past decade have shown that pulse oximeters become inaccurate during desaturation events both at rest (Grace, 1994; Thrush and Hodges, 1994; Trivedi et al., 1997) and during exercise (Wood et al., 1997; Yamaya et al., 2002). The mag-
The magnitude of the error depends on the specific monitor, location of the sensor, and degree of hypoxia (Sinex, 1999).

Given that NHC can elicit a wide range of hypoxia (F\textsubscript{I\textsubscript{O}}\textsubscript{2} 20.9% to 9.5%), and that at present there are no guidelines as to the appropriate level or time frame of hypoxic exposures, studies are warranted to address the basic efficacy of altitude simulation chambers and the validation of pulse oximeters employed to monitor humans. However, to our knowledge no scientific studies have tested the reliability of pulse oximetry measurements against recognized standards in response to the hypoxia generated by commercially available NHC. The general aim of the present study was to compare noninvasive pulse oximetry with direct arterial blood measurements via co-oximetry throughout the condition of progressive normobaric hypoxia. The specific aims of this study were to address three main questions: First, is Sp\textsubscript{O}\textsubscript{2} a valid estimate of Sa\textsubscript{O}\textsubscript{2} at the levels of normobaric hypoxia typically used in research and recreational settings? Second, does the location of pulse oximetry sensors have any effect on Sp\textsubscript{O}\textsubscript{2} estimation? Finally, are changes in Sp\textsubscript{O}\textsubscript{2} and Sa\textsubscript{O}2 similar relative to the severity of hypoxemia?

Methods

SUBJECTS AND EXPERIMENTAL DESIGN

Thirteen young adults (6 F, 7 M) age 21.3 ± 0.6 years volunteered to participate in the study. All were healthy nonsmokers and were not taking any medication. None reported any history of cardiovascular, cerebrovascular, or respiratory disease. Each volunteer gave informed consent according to ethical guidelines approved for this study by the Conjoint Health Research Ethics Board, University of Calgary.

The project utilized a modified one-group time series design (Campbell and Stanley, 1966) that incorporated sequential single-subject analyses during the treatment protocol, in order to compare two methods which measure the same factor (Altman and Bland, 1983). The method of co-oximetry provided direct arterial blood measurements of Sa\textsubscript{O}2 for comparison against noninvasive pulse oximetry estimates of oxyhemoglobin saturation. Method comparisons were made from a total of seven arterial blood samples, collected from each subject at designated intervals during the progressive normobaric hypoxic exposure, according to the procedure described below.

PROGRESSIVE NORMOBARIC HYPOXIA

An NHC equipped with air separation generators (Hypoxico, Inc., New York), was used to progressively reduce the normoxic F\textsubscript{I\textsubscript{O}}\textsubscript{2} of 20.9% over a 150-min period to an endpoint F\textsubscript{I\textsubscript{O}}\textsubscript{2} of approximately 11.5%. The choice of hypoxic exposure is well supported in the literature for both normobaric and hypobaric studies (Benoit et al., 1992; Rodriguez et al., 1999), while the time frame provided ideal logistical conditions for blood sampling and analysis. The F\textsubscript{I\textsubscript{O}}\textsubscript{2} inside the NHC was monitored on a continuous basis throughout the progressive normobaric hypoxic exposure using a TrueMax 2400 metabolic measurement system (Parvo-Medics, Salt Lake City, UT).
PULSE OXIMETRY

HR and SpO$_2$ were continuously monitored via pulse oximetry (Nellcor N-295, Nellcor Inc., Hayward, CA). The noninvasive computerized monitor, with memory storage and capable of real-time download, was attached to the subject’s index finger using a comfortable near-infrared light-emitting diode (LED) probe (D-25) secured in place with opaque tape to prevent signal artifacts from fluorescent lighting. Additionally, a second set of pulse oximetry data was obtained from each subject via a separate monitor with an LED probe (RS-10) placed over the eyebrow relative to the pulsatile events of the anterior branch of the temporal artery. The dual sets of probes were established to retrieve information regarding possible variability of SpO$_2$ measurements between sites (site dependency).

BLOOD SAMPLES AND ANALYSES

Radial artery catheterization was established and monitored by a physician. Prior to entering the hypoxic room, the subjects underwent an initial 1-ml blood draw into a sterile, heparinized syringe under ambient atmospheric conditions (inspired fraction of oxygen [F$_{I\ O_2}$] = 20.9%). The second sample was obtained following 15 min of hypoxic exposure. The remaining five blood collections occurred at 30-min intervals while the F$_{I\ O_2}$ was progressively reduced to approximately 11.5%. Blood gas analysis and co-oximetry measurements were performed immediately following sample collection. Arterial blood samples were assessed for pH, PaCO$_2$, PaO$_2$, HCO$_3$, base excess, and total hemoglobin concentration, with a CIBA-Corning 280 blood gas analyser (East Walpole, MA). The blood gas analyzer is equipped with a preheater which warmed each blood sample to 37°C prior to entering the electrode sample path.

Nonhemolyzing co-oximetry assays of oxyhemoglobin fraction (FO$_2$Hb), carboxyhemoglobin fraction (FCOHb), methemoglobin fraction (FM$_{ET}$Hb), and total hemoglobin were acquired with an AVOXimeter 4000 (San Antonio, TX). Strong precision and accuracy (oxyhemoglobin linear relationship, $R^2 = 0.999$, precision = 0.85%, bias = 0.04%) of the AVOXimeter 4000 compared with a conventional IL 482 co-oximeter has previously been reported (Bailey et al., 1997). To obtain SaO$_2$, or functional saturation, for direct comparison with pulse oximetry SpO$_2$ data, we employed the following equation (AVOX Systems, 2001):

$$\text{SaO}_2 = 100 \times \text{FO}_2\text{Hb} / 100 - (\text{FCOHb} + \text{FM}_{ET}\text{Hb})$$

Both the CIBA-Corning blood gas analyzer and the AVOXimeter 4000 were calibrated daily using standard procedures according to the manufacturer’s specifications.

SYNCHRONIZATION OF BLOOD MEASUREMENTS WITH PULSE OXIMETRY

The internal digital timing mechanisms for each pulse oximeter, one with a temporal probe and the other with a finger probe, were synchronized exactly to within 1 sec. The Nellcor NPB-295 stored HR and SpO$_2$ data at 2-sec intervals throughout the experimental treatment. An event marker was recorded to identify the precise moment at which each blood sample was drawn. The F$_{I\ O_2}$ inside the chamber was
also recorded during each blood sampling procedure. Subsequently, a 40-sec interval of pulse oximetry data, 20 sec on either side of the blood collection event, was used for comparison with the direct blood gas analysis.

**STATISTICAL ANALYSIS**

The correlation between direct blood measurements of SaO$_2$ and noninvasive indirect measurements from the two pulse oximetry devices (SpO$_2$) were initially evaluated using parametric statistics. Pearson product-moment correlation coefficients, $R^2$, and regression analyses were examined between the dependent variables. To examine differences between regression lines, we used the Chow Test (Dillon and Goldstein, 1984) to assess whether the coefficients estimated over one group of the data were equal to those estimated over another group of data. Significance was accepted at $p < 0.05$. To further address the accuracy of difference between the instruments, we used method comparison analysis (Altman and Bland, 1983; Bland and Altman, 1986). First, the differences between methods of measurement for each collection period were determined ($N = 84$). Then the bias ($b$), or the mean value between the differences of SaO$_2$ and SpO$_2$, in conjunction with precision ($p$), or the standard deviation of the differences, were calculated to graphically illustrate the limits of agreement between the methods. These lines of identity then represent the 95% confidence interval, or ±2 standard deviations of the differences:

$$
b = \text{SaO}_2 - \text{SpO}_2 \div n - 1$$

$$p = SD \text{ of } (\text{SaO}_2 - \text{SpO}_2) = SD_{\text{diff}}$$

Limits of Agreement = $b ± 2SD_{\text{diff}}$

**Results**

Summary data obtained from co-oximetry, blood gas analysis, and pulse oximetry for all subjects during the progressive normobaric hypoxia are shown in Figure 1. The summary graph, constructed from the means of all data points ($N = 84$) during each synchronized blood collection event, illustrates decreasing SaO$_2$ and SpO$_2$ throughout the progressive hypoxic protocol (lower panel). The upper panel of Figure 1 shows an initial rapid decline in PaO$_2$ (partial pressure of arterial oxygen), which then descended more slowly from approximately 65 mmHg to 40 mmHg as the normobaric hypoxia progressed from an F$_{O_2}$ of approximately 20% to 11.5%. In contrast, the PaCO$_2$ (partial pressure of arterial carbon dioxide) demonstrated a small decrease over the period of hypoxia.

**LINEAR REGRESSIONS AND THE CHOW TEST**

Regression analysis between SaO$_2$ measured from direct blood samples, and SpO$_2$ estimated with pulse oximetry, are presented in Figure 2 for all observations and Figure 3 for observations <85%. Both SpO$_2$ temporal (LED probe RS-10) and SpO$_2$ finger (LED probe D-25) were highly correlated with co-oximetry SaO$_2$ ($R^2 = 0.92$ and 0.89, respectively). However, the strength of the correlation between co-oximetry and both pulse oximetry devices (temporal and finger) was substantially reduced
when blood saturation levels below 85% were compared (n = 26). When co-oximetry SaO2 < 85% was compared with SpO2, the temporal probe had a much higher \( R^2 \) relative to the finger probe (\( R^2 = 0.71 \) and 0.56, respectively). The Chow Test identified significant differences between the methods when all the data from SpO2temporal and SpO2finger were compared with SpO2finger < 85% (\( p < 0.001 \)).

** AGREEMENT BETWEEN METHODS OF MEASUREMENT **

The results for \( b \) and \( p \) between SaO2 and SpO2temporal and SpO2finger are presented in Figure 2 for all observations, and Figure 3 for observations < 85%, adjacent to regression lines. Relative to site specificity of the two LED probes when averag-
Validation of Pulse Oximetry

Figure 2. Regression lines (left), and limits of agreement (right) for all data points ($N = 84$) comparing SpO$_2$ temporal (RS-10, Panels A and C) and SpO$_2$ finger (D-25, Panels B and D) with SaO$_2$. Long and thick dash lines represent ±95% confidence interval; short and thin dash line identifies the bias. Values for limits of agreement and regression analysis are listed in Table 1.

From all the data, the RS-10 temporal probe had the lowest $b$ and greatest $p$ compared to the D-25 finger probe ($b, p = 0.016, 2.47$ vs. $0.47, 3.03$, respectively). The $b$ for SpO$_2 < 85\%$ was similar for both LED probes, while $p$ was much greater for the temporal probe compared to the finger probe ($1.37, 2.72$ vs. $1.12, 4.34$, respectively). The limits of agreement and regression analysis are summarized in Table 1.
Figure 3. Regression lines (left), and limits of agreement (right) for all SaO2 < 85% observations (n = 26) comparing SpO2 temporal (RS-10, Panels A and C) and SpO2 finger (D-25, Panels B and D) with SaO2. Long and thick dash lines represent ± 95% confidence interval; short and thin dash line identifies the bias. Values for limits of agreement and regression analysis are listed in Table 1.

Discussion

Three new findings emerge from this study. First, pulse oximeters provide reasonable accuracy for estimating arterial oxygen saturation in an NHC when SaO2 > 85%. However, the accuracy of the oximetry devices used in this experiment deteriorated as hypoxemia increased. Second, in response to progressive normobaric hypoxia, the RS-10 (temporal probe) provided greater validity than did the D-25 (finger probe) when compared to in vitro arterial blood samples analyzed by co-oximetry. The enhanced performance of the temporal probe is reflected by a higher
Table 1  Method Comparison Data Showing Regression Correlation and Limits of Agreement

<table>
<thead>
<tr>
<th>Methods</th>
<th>$R^2$</th>
<th>Slope</th>
<th>Intercept</th>
<th>Bias</th>
<th>Precision</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\text{SaO}_2$ vs. $\text{SpO}_2^{\text{temporal}}$</td>
<td>0.92</td>
<td>1.08</td>
<td>−7.33</td>
<td>0.02</td>
<td>2.47</td>
<td>−5.41 to 4.47</td>
</tr>
<tr>
<td>$\text{SaO}_2$ vs. $\text{SpO}_2^{\text{finger}}$</td>
<td>0.89</td>
<td>1.10</td>
<td>−8.56</td>
<td>−0.47</td>
<td>3.03</td>
<td>−6.52 to 5.58</td>
</tr>
<tr>
<td>$\text{SaO}_2$ vs. $\text{SpO}_2^{\text{temporal}}$</td>
<td>&lt; 85%</td>
<td>0.73</td>
<td>1.02</td>
<td>−2.80</td>
<td>1.38</td>
<td>2.72 to −4.06</td>
</tr>
<tr>
<td>$\text{SaO}_2$ vs. $\text{SpO}_2^{\text{finger}}$</td>
<td>&lt; 85%</td>
<td>0.56</td>
<td>1.19</td>
<td>−15.90</td>
<td>1.13</td>
<td>4.34 to −7.55</td>
</tr>
</tbody>
</table>

Note: $\text{SpO}_2^{\text{temporal}}$ = arterial oxygen saturation via pulse oximeter with probe placed over the anterior branch of temporal artery (RS-10); $\text{SpO}_2^{\text{finger}}$ = arterial oxygen saturation via pulse oximeter with probe placed on index finger (D-25); < 85% = incorporates hypoxic data points below 85%. CI = confidence interval.

correlation, smaller $b$, greater $p$, and a narrower ±95% confidence interval in the sample from this study. Third, when the data were limited to low saturation levels (< 85%), the finger probe performed significantly worse than the temporal probe ($p < 0.001$).

SITE DEPENDENCY

Given that the pulse oximetry monitors used in this study operated with identical algorithms for estimating $\text{SpO}_2$, the discrepancy between the temporal probe (RS-10) and finger probe (D-25) may have been due to specific site location and/or design differences between the probes. The temporal probe is a reflectance instrument with the LED and detector on the same side of the tissue, while the finger probe is a transmission instrument that emits light through the fingernail, tissue, and blood with a detecting sensor located on the opposite side of the finger. In the present work, the finger probe was placed on the right index finger as per the manufacturer’s directions, while the temporal probe site was identified by palpation of the anterior branch of the temporal artery above the eyebrow, followed by a brief search for the area that generated the highest arterial saturation and strongest plethysmography event.

There is disagreement in the literature relative to the effect of pulsating arteries on reflectance pulse oximetry probes. Some researchers have reported enhanced performance (Dassel et al., 1995; Trivedi et al., 1997) while others suggest a deterioration in accuracy (Hamber et al., 1999; Jorgensen et al., 1995; Kelleher and Ruff, 1989). Reduced accuracy of reflectance pulse oximetry has been reported in endotracheally intubated and ventilated adults undergoing general anesthesia and surgery, as well as in adult volunteers placed in the Trendelenberg (head-
down tilt) position (Jorgensen et al., 1995). Of note is a contrary report which states that a small amount of pressure (7.3 to 11.9 kPa) applied to reflectance probes located on the forehead improves $\text{SpO}_2$ accuracy (Dassel et al., 1995). Interestingly, the RS-10 temporal probe used in the present study was held in place with an elastic headband, which exerted some pressure on the sensor against the skin of the forehead and may have augmented performance.

Seven pulse oximeters (reflectance and transmission) were examined for accuracy and response time (sensitivity) to a single brief 30- to 90-sec hypoxic event ($\text{F}_1\text{O}_2 = 10\%$) engendered via reduced oxygen gas mixtures (Trivedi et al., 1997). While all devices exhibited varying levels of inaccuracy at low saturations ($b$ range = 1.2 to 8.5, $p$ range = ±3.1 to 7.5), the Nellcor 200 reflectance forehead probe and the Ohmeda ear probe had the best performance. Trivedi and colleagues (1997) suggested that the reflectance forehead probe and ear probe may have monitored desaturation events more accurately and faster due to their placement being central, as opposed to the peripheral finger probe location. It is possible that the issue of central vs. peripheral site dependency may have contributed to the improved performance of the centrally fixed Nellcor RS-10 temporal probe in the present study. In support of this, centrally located ear probes have recently been identified to produce significantly ($p < 0.05$) enhanced performance during desaturations of $\text{SaO}_2 < 90\%$ when compared with probes placed peripherally on the finger and toe (Hamber et al., 1999).

Pulse oximetry devices similar to those used during our NHC protocol at rest were evaluated by Yamaya et al. (2002) in response to exercise while breathing hypoxic gas ($\text{F}_1\text{O}_2 = 12\%$). Our results for the RS-10 temporal probe ($b = 0.02$, $p = 2.47$) compare favorably with those of Yamaya and colleagues ($b = 0.3$, $p = 2.5$). However, our findings suggest that the D-25 finger probe is more accurate during rest ($b = -0.47$, $p = 3.03$) than during hypoxic exercise ($b = -1.4$, $b = 7.9$). This discrepancy between rest and exercise may be due to exercise-induced peripheral vasoconstriction causing reduced perfusion at the site of measurement, or to motion artifact induced by exercise (Severinghaus and Kelleher, 1992). Collectively, our results and those reported by Yamaya et al. (2002) suggest that during hypoxic episodes, both at rest and during exercise, reflectance pulse oximetry probes are more reliable than transmission probes.

**LOW LEVELS OF OXYGEN SATURATION**

Yelderman and William (1983) compared direct arterial blood oxygen saturation with results from a pulse oximeter fitted with a finger probe. They obtained 79 data points from five subjects exposed to stepwise hypoxia to $\text{SaO}_2$ of approximately 70% and reported a correlation coefficient of 0.98. Examination of their scatterplot, however, suggests that variability may have increased at lower saturations. Furthermore, they did not present data regarding $b$ (mean differences) or $p$ (standard deviation of the differences). Despite strong correlations between pulse oximetry and $\text{SaO}_2$, care should be exercised in interpreting the data presented by Yelderman and William, since strong bivariate relationships do not necessarily imply agreement between the two methods (Atkinson and Nevill, 2000).
Alternatively, in a study by Severinghaus et al. (1989), a rapid desaturation technique utilizing compressed low oxygen delivered by a mouthpiece assessed the accuracy of 14 pulse oximeters while addressing the agreement between methods. Seven probes were of the finger type and seven were reflectance forehead probes. When compared to co-oximetry SaO₂, the correlation coefficients for these devices ranged from $R^2 = 0.81$ to 0.96. At very low saturations of approximately 55%, the average $b$ varied greatly (–21.60 to 1.86) while the average $p$ also demonstrated considerable variability (2.46 to 9.26).

More recently, the accuracy of four pulse oximeters were examined in healthy adults at rest in response to hypoxic stages of SaO₂ < 80%, 85–90%, 90–95%, and 95–100%. Thrush and Hodges (1994) reported a statistically significant ($p < 0.05$) deterioration in accuracy from all four devices as oxygen saturation decreased. They expressed concern about the reliability of pulse oximetry and revealed numerous falsely elevated SpO₂ values above 90% when SaO₂ measurements (co-oximetry) for the same subjects were below 90%, suggesting that greater degrees of hypoxemia were elicited than was indicated by the pulse oximeters. When saturations below 80% were analyzed using method comparisons, $b$ varied from 0 to 2 while $p$ ranged from ±3 to 5. Our own findings for both temporal RS-10 and finger D-25 probes for oxygen saturation levels < 85% are consistent with the data of Thrush and Hodges (1994).

**CONCLUSION**

In summary, the pulse oximetry devices used in the present study operated with higher accuracy and precision, under the condition of progressive normobaric hypoxia established with air separation generators, when the arterial oxygen saturation was > 85%. Therefore SpO₂ is a valid estimate of SaO₂ when SaO₂ > 85%. In the sample population from this study, reflectance sensor probes obtained significantly better pulse oximetry recordings than did transmission probes when SaO₂ was < 85%, suggesting that design differences or central vs. peripheral location may impact performance during low levels of oxygen saturation.

From a practical perspective, our results suggest that pulse oximetry provides reliable monitoring of hypoxemia at rest when utilizing normobaric hypoxic chambers within the range of 20.9% to 15% FIO₂, equivalent to an altitude range of sea level to approximately 3,500 m (Ward et al., 2000). Practical relevance for exercise is beyond the scope of this paper. However, some useful information can be derived from studies at rest. It is important to recognize that all pulse oximeters estimate in vivo arterial oxygen saturation by measuring the light absorbance of hemoglobin at two different wavelengths, and also that there are many differences in the algorithms and filtering methods used by individual manufacturers to determine SpO₂. In this regard, caution should be taken when monitoring individuals with pulse oximetry, especially those exposed to low saturation levels generated by normobaric hypoxia. It would seem prudent that, when anticipating regular use of commercially available normobaric hypoxic chambers, the pulse oximetry devices for assessing SpO₂ should be validated against arterial blood measurements to ensure the health status of the user.
Acknowledgments

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References


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