Research

Do changes in pulse oximeter oxygen saturation predict equivalent changes in arterial oxygen saturation?

Gavin D Perkins¹, Daniel F McAuley², Simon Giles³, Helen Routledge⁴ and Fang Gao⁵

¹Specialist Registrar, Intensive Care Unit, Birmingham Heartlands and Solihull NHS Trust (Teaching), Birmingham Heartlands Hospital, Birmingham, UK
²Specialist Registrar, Intensive Care Unit, Birmingham Heartlands and Solihull NHS Trust (Teaching), Birmingham Heartlands Hospital, Birmingham, UK
³Nurse Consultant, Intensive Care Unit, Birmingham Heartlands and Solihull NHS Trust (Teaching), Birmingham Heartlands Hospital, Birmingham, UK
⁴Specialist Registrar, Intensive Care Unit, Birmingham Heartlands and Solihull NHS Trust (Teaching), Birmingham Heartlands Hospital, Birmingham, UK
⁵Consultant in Anaesthesia and Intensive Care Medicine, Intensive Care Unit, Birmingham Heartlands and Solihull NHS Trust (Teaching), Birmingham Heartlands Hospital, Birmingham, UK

Correspondence: F Gao, f.g.smith@bham.ac.uk

Introduction

Pulse oximetry is used almost universally in the management of critically ill patients in the intensive care unit (ICU) and operating theatre [1]. Its uses include the detection of hypoxia [1], avoidance of hyperoxia [2], reduction in the frequency of blood gas analysis [3], titration of fractional inspired oxygen [4] and for weaning from mechanical ventilation [5].

An arterial oxygen saturation (SaO₂) of 90% has been proposed as a target for adequate oxygenation during mechanical ventilation [5]. Previous studies investigating the use of pulse oximeter oxygen saturation (SpO₂) in intensive care patients have reported that the minimum SpO₂ levels to maintain SaO₂ at 90% range between 92% and 96% [4,6,7]. However, these studies have not answered the question of whether, after achieving a target SaO₂, a subsequent change in SpO₂ predicts a corresponding change in SaO₂ in the critically ill.

Some studies have reported that anaemia reduces the precision of pulse oximetry [8] by increasing the signal to noise ratio with low haemoglobin concentrations, whereas others failed to demonstrate this phenomenon [9,10]. Acidosis may

Abstract

Introduction This study investigates the relation between changes in pulse oximeter oxygen saturation (SpO₂) and changes in arterial oxygen saturation (SaO₂) in the critically ill, and the effects of acidosis and anaemia on precision of using pulse oximetry to predict SaO₂.

Patients and methods Forty-one consecutive patients were recruited from a nine-bed general intensive care unit into a 2-month study. Patients with significant jaundice (bilirubin >40 µmol/l) or inadequate pulse oximetry tracing were excluded.

Results A total of 1085 paired readings demonstrated only moderate correlation (r=0.606; P<0.01) between changes in SpO₂ and those in SaO₂, and the pulse oximeter tended to overestimate actual changes in SaO₂. Anaemia increased the degree of positive bias whereas acidosis reduced it. However, the magnitude of these changes was small.

Conclusion Changes in SpO₂ do not reliably predict equivalent changes in SaO₂ in the critically ill. Neither anaemia nor acidosis alters the relation between SpO₂ and SaO₂ to any clinically important extent.

Keywords acidosis, anaemia, arterial oxygen saturation, critical care, pulse oximetry


This article is online at http://ccforum.com/content/7/4/R67

© 2003 Perkins et al., licensee BioMed Central Ltd
(Print ISSN 1364-8535; Online ISSN 1466-609X). This is an Open Access article: verbatim copying and redistribution of this article are permitted in all media for any purpose, provided this notice is preserved along with the article’s original URL.

Received: 23 September 2002
Revisions requested: 2 December 2002
Revisions received: 25 February 2003
Revisions requested: 10 December 2002
Revisions received: 29 March 2003
Accepted: 12 May 2003
Published: 11 June 2003

ICU = intensive care unit; SaO₂ = arterial oxygen saturation; SD = standard deviation; SpO₂ = pulse oximeter oxygen saturation.
also influence the relation between \( \text{SpO}_2 \) and \( \text{SaO}_2 \). The *in vitro* method employed by the carbon monoxide (CO)–
oximeter requires red blood cell lysis, whereas the pulse
oximeter analyzes haemoglobin in whole blood [11]. The dif-
ference between intracellular and extracellular hydrogen ion
concentrations under normal physiological conditions has
been incorporated into the pulse oximeter algorithms. However, the robustness of this adjustment has not been
evaluated in the critically ill and acidic patient.

We therefore conducted a prospective observational study to
test the hypothesis that a change in \( \text{SpO}_2 \) would predict an
equivalent change in \( \text{SaO}_2 \). Such a relation, if it exists, would be
invaluable in deciding when to titrate fractional inspired oxygen
and/or repeat arterial blood gases in the individual patient. Fur-
thermore, we examined the effects of anaemia and acidosis on
the precision of using the pulse oximeter to predict the \( \text{SaO}_2 \) in
a heterogeneous group of critically ill patients.

**Patients and methods**

This study was considered by the local research and ethics
committee and the need for informed consent was waived in
view of the observational nature of the study.

During a 2-month period all patients admitted to our ICU who
had an arterial line for the measurement of blood gases and
who were being monitored by continuous pulse oximetry
were recruited. The following patients were excluded: those
with significant jaundice (bilirubin >40 \( \mu \text{mol/l} \)) or a history of
smoke inhalation; those with an inadequate \( \text{SpO}_2 \) trace (as
determined by visual analysis of a flat, absent, or irregular
signal waveform); and those in whom fewer than two arterial
blood gas readings were taken.

Serial arterial blood gas samples were taken after 5 ml blood
had been discarded when indicated as part of routine clinical
care. No samples were taken solely for the study nor was any
attempt made to vary inspired oxygen concentration or
mechanical ventilation for the purposes of the study. The
samples were analyzed in a standardized manner within 2 min
of sampling. Arterial blood gas samples were analyzed using a
CO-oximeter (ABL725, Radiometer, Copenhagen, Denmark)
that was calibrated daily by laboratory staff and has a 2-hourly
automatic internal calibration sequence. Haemoglobin concen-
tration (g/dl), hydrogen ion concentration (nmol/l), and per-
centage \( \text{SaO}_2 \) were recorded for each sample. Precision and
accuracy of a whole blood sample for \( \text{SaO}_2 \), hydrogen ion concen-
tration and haemoglobin concentration are 0.3 and 0%,
0.034 and 0.008 nmol/l, and 0.12 and 0.4 g/dl, respectively,
within a haemoglobin range of 5–20 g/dl.

Pulse oximetry readings were recorded simultaneously with
blood gas sampling using a Nellcor (Puritan Bennett,
Pleasanton, NJ, USA) finger probe attached to a Hewlett
Packard (Palo Alto, CA, USA) Merlin monitor. The pulse
oximeter displays an average \( \text{SpO}_2 \) from the preceding 5-s
beat by beat analysis. The measurements between healthy
individuals \((n=12)\) had a coefficient of variation of 0.4% at a
\( \text{SpO}_2 \) of 97%. Probes were attached to a finger, choosing
the digit that gave the best trace and but not necessarily on
the arm from which the arterial blood gas sample was drawn.
However, the same probe was used for all measurements
from the same patient.

**Statistical analysis**

Data were stored using Microsoft Excel 97 and analyzed
using SigmaStat for Windows 95 (SPSS Inc. Chicago, IL,
USA) and GLIM (Generalized Linear Interactive Modeling)
version 4, update 8 (Royal Statistical Society, London, UK),
running on a DEC Alpha AXP mainframe computer under the
Ultix operating system (OSF/1). The changes in residuals
were tested for normality and found to be normally distrib-
uted. The linear relations between differences in two succes-
sive measurements of \( \text{SpO}_2 \) and \( \text{SaO}_2 \) in all patients were
analyzed using Pearson correlation coefficient \((r)\), linear
regression and goodness-of-fit (adjusted \( R^2 \)). The variations
between and within the patients were examined using com-
parisons of the residual standard deviations (SDs) between
a single line from a common slope through all the changes for
all 41 patients and a separate line to each patient.

The effects of anaemia and acidosis on the agreement
between the two measurement techniques were examined
using a Bland–Altman plot [12] in which the difference
between \( \text{SpO}_2 \) and \( \text{SaO}_2 \) was plotted against their average
[13]. Bias and the limits of agreement were calculated. Bias
was calculated as the mean of the differences between the
CO-oximeter and pulse oximeter readings \((\text{SaO}_2–\text{SpO}_2)\) [11].
Positive bias indicated that the pulse oximeter underesti-
mated the \( \text{SaO}_2 \), whereas negative bias indicated that the
pulse oximeter was overestimating the \( \text{SaO}_2 \). The limits of
agreement were taken as the bias ± \((1.96 \times \text{SD})\) [6,13].

Approximately 95% of data fell within the haemoglobin concen-
tration range 8–11.9 g/dl and the hydrogen ion concentration
range 25–62.9 nmol/l (\( \text{pH}7.2–7.6 \)). Therefore, haemoglobin
concentrations \( \leq 7.9 \) or \( \geq 12 \) g/dl or hydrogen ion concen-
trations \( \geq 63 \) nmol/l were regarded in the study as extremes. The
differences of biases between these three groups were ana-
yzed using one-way, repeated measure analysis of variance.
\( P \leq 0.05 \) was considered statistically significant.

**Results**

Forty-one (22 male) patients (age \([\text{mean} \pm \text{SD}]\) 70 ± 14 years)
were recruited into the study. A total of 1132 simultaneous
arterial blood gas and pulse oximeter readings were taken
(mean \([\text{range}]\) 27 [3–91] readings per patient). Sequential
readings in each patient were grouped together into pairs,
which gave 1085 paired readings (47 readings were
excluded because they were either not paired or unidentifi-
able to a particular patient, or the patient had fewer than two
readings taken). These data were analyzed to determine the
The relation between changes in \( \Delta \text{SpO}_2 \) and changes in \( \Delta \text{SaO}_2 \).

The mean ± SD for \( \text{SpO}_2 \) was 94.6 ± 2.7% and the mean for \( \text{SaO}_2 \) was 95.9 ± 2.4%. In terms of predicting \( \Delta \text{SaO}_2 \) from \( \Delta \text{SpO}_2 \), fitting a single line from all the 41 patients, gives a residual SD of 1.303 and fitting a separate line to each patient gives a residual SD of 1.288 \((P=0.999)\). Therefore, there was no significant difference in residual SD within patients overall. Although we found moderate correlation between \( \Delta \text{SpO}_2 \) and \( \Delta \text{SaO}_2 \) \((r=0.606; P<0.01; \text{Fig. 1})\), only 36.7% of the variation in this relation was due to the association of changes in \( \text{SpO}_2 \) with changes in \( \text{SaO}_2 \) (adjusted \( R^2 = 0.367 \)). The prediction of \( \Delta \text{SaO}_2 \) from \( \Delta \text{SpO}_2 \) \((\Delta \text{SaO}_2 = 0.003 + 0.477 \Delta \text{SpO}_2)\) demonstrates that the pulse oximeter overestimates actual changes in \( \text{SaO}_2 \).

The 1085 simultaneous arterial blood gas and pulse oximeter readings from the 41 patients were analyzed to determine the effects of anaemia and acidosis on bias and limits of agreement. For the data altogether, the bias was 1.34 and the limits of agreement were \(-2.29\) and \(+4.97\) (Fig. 2). There were only small changes in bias with anaemia \(+2.09\) and acidosis \(+0.38\), as shown in Table 1. The difference in bias between hydrogen ion concentrations of 25–63 nmol/l and \(\geq 63\) nmol/l \((P<0.01)\), and between haemoglobin concentrations of \(<8\) g/dl, 8–12 g/dl and \(>12\) g/dl \((P<0.01)\) all achieved statistical significance. The bias was not significantly different between haemoglobin concentrations of \(<7.9\) g/dl and 8–11.9 g/dl \((P=0.24)\). There were insufficient numbers in the group with hydrogen ion concentration \(<24.9\) nmol/l \((n=10)\) for analysis to be done.

**Discussion**

The present study shows that changes in \( \text{SpO}_2 \) do not reliably predict equivalent changes in \( \text{SaO}_2 \), with the pulse oximeter tending to overestimate actual changes in \( \text{SaO}_2 \). We also showed that \( \text{SpO}_2 \) underestimates \( \text{SaO}_2 \) to a greater extent with progressive anaemia, whereas acidosis increases the \( \text{SpO}_2 \) estimate of \( \text{SaO}_2 \). However, the clinical significance of these changes is small.

**Table 1**

<table>
<thead>
<tr>
<th>The effects of anaemia and acidosis on bias and limits of agreement</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Haemoglobin concentration (g/dl)</strong></td>
</tr>
<tr>
<td>-------------------------------------</td>
</tr>
<tr>
<td>Total</td>
</tr>
<tr>
<td>(n) (measurements)</td>
</tr>
<tr>
<td>Bias</td>
</tr>
<tr>
<td>Upper limit</td>
</tr>
<tr>
<td>Lower limit</td>
</tr>
</tbody>
</table>

*P<0.01, versus *haemoglobin >12 g/dl, †haemoglobin <8 g/dl, ‡haemoglobin 8–12 g/dl and §hydrogen 25–63 nmol/l.
The titration of fractional inspired oxygen during weaning from mechanical ventilation is frequently adjusted with the goal of maintaining a target \(\text{SpO}_2\) value. Jubran and Tobin [4], in a study involving 54 ICU patients, reported that levels of \(\text{SpO}_2\) of 92% in white patients and 95% in black patients maintained arterial oxygen tension at 8 kPa or greater in 92% and 85% of patients, respectively. Seguin and coworkers [6] defined a minimum \(\text{SpO}_2\) of 96% to ensure that no patients had a \(\text{SaO}_2\) below 90%. This approach avoided hypoxia, but 15% of patients had a \(\text{SaO}_2\) of 98% or greater.

Although target values can be helpful, it would be valuable to know whether a change in \(\text{SpO}_2\) would predict a similar change in \(\text{SaO}_2\) in critically ill patients over time. Hypothetically, the relatively static patient factors that interfere with pulse oximetry (skin colour, finger size, carboxyhaemoglobin, methaemoglobin) do not change, and so the correlation between changes in \(\text{SaO}_2\) and \(\text{SpO}_2\) might be expected to be closer than that between absolute values from a mixed patient population. This could allow individualized target \(\text{SpO}_2\) to be set, based on a single, one-off \(\text{SaO}_2\) reading. Only one small study has attempted to address this question in the intensive care setting. In a series of 45 patients (135 measurements), Van de Louw and coworkers [14] recently reported that changes in \(\text{SpO}_2\) could not accurately predict changes in \(\text{SaO}_2\). Our larger study supports and extends this early finding. The prediction of \(\Delta\text{SaO}_2\) from \(\Delta\text{SpO}_2\) (\(\Delta\text{SaO}_2=0.003+0.477\Delta\text{SpO}_2\)) demonstrates that, on average, the pulse oximeter overestimates actual changes in \(\text{SaO}_2\). This suggests that a similar degree of caution is required in interpreting changes in pulse oximetry in the critically ill as in one-off readings.

Progressive reductions in haemoglobin concentration may reduce the precision of the pulse oximeter as the signal:noise ratio from surrounding tissue increases [15]. Early studies examining the effects of anaemia on the precision of the pulse oximeter found reduced precision in association with anaemia. Lee and coworkers [8] demonstrated a deterioration in bias and precision in dogs with a haematocrit below 10%, and Severinghaus and coworkers [16] reported increased error in anaemic humans when the \(\text{SaO}_2\) was less than 75%. In contrast, case reports have described cases in which the pulse oximeter remained precise at haemoglobin concentrations of 2.7 g/dl [17] and 3.0 g/dl [10]. A subsequent case series of 17 patients with acute anaemia due to haemorrhage (haemoglobin concentration 2.3–8.7 g/dl) did not detect any deterioration in the accuracy of measurements using the pulse oximeter in the absence of hypoxia [9]. Our study did not include sufficient numbers with hypoxia (\(\text{SpO}_2 <90\%\)) for the influence of anaemia on bias and precision to be studied in this patient group. However, under normal physiological conditions (\(\text{SpO}_2 >90\%\)) our results support and extend previous findings in demonstrating that anaemia has only a minor impact on the precision of measurements using the pulse oximeter.

Our data show that, in the presence of acidosis, the degree to which \(\text{SpO}_2\) underestimates \(\text{SaO}_2\) was reduced. One possible explanation for this finding may relate to the differences in the techniques used for measuring oxygen saturation. The pulse oximeter analyzes haemoglobin saturation in whole blood \textit{in vivo} [18], whereas \(\text{SaO}_2\) measured by CO-oximetry requires red blood cell lysis prior to analysis. Under normal physiological conditions, algorithms incorporated in the pulse oximeter account for this [11], although the validity of this adjustment has not been tested outside normal physiological ranges. Alternatively, the effects of the complex interactions between cardiac output [19], systemic vascular resistance [20], temperature [19] and vasoactive drugs [14,21] on precision of measurements using the pulse oximeter might have contributed to this finding. A further study looking at the precise contribution of each of these factors would be required to elucidate the aetiology of these findings definitively.

There are several potential confounding variables that were not controlled for in the study design. First, like in other studies [4,6], we did not analyze the influence of carboxyhaemoglobin and methaemoglobin concentrations on bias and precision. The pulse oximeter is unable to distinguish between these two forms of haemoglobin and oxyhaemoglobin, leading it to overestimate the actual \(\text{SaO}_2\) if significant concentrations of either are present [22,23]. We excluded patients with a history of smoke inhalation, in whom carboxyhaemoglobin levels may be high. In nonsmokers carboxyhaemoglobin levels are normally less than 2% and methaemoglobin levels are less than 1% [15]—levels that are already accounted for by the built-in algorithms of pulse oximeters. In cigarette smokers carboxyhaemoglobin is initially elevated (average 4.78%) but falls over time (half life 5–6 hours) [24]. The clearance of carboxyhaemoglobin is also accelerated by ventilation [25]. Because most patients had been ventilated for several hours before entry into the study, this is unlikely to have significantly confounded the results. We excluded patients with significant jaundice—a group known to have high carboxyhaemoglobin levels [26]—in order to minimize this potential error, and no patients were admitted following smoke inhalation during the study period. Anaemia and acidosis have not been found to influence carboxyhaemoglobin or methaemoglobin concentrations. Although we believe that the influence of carboxyhaemoglobin levels in the study was minimal, we are unable to rule it out as a potential confounding variable.

Second, we did not classify patients according to skin colour or race, which may impact on accuracy of the pulse oximeter [4]. Because skin colour is constant, comparisons of changes in \(\text{SpO}_2\) are unlikely to have been affected. Data for the assessments for bias and precision caused at the extremes of anaemia and acidosis were collected from 19 and 14 patients, respectively, and there did not appear to be any systematic difference in the groups’ racial composition from that in the overall study population. No patients to our knowl-
edge had sickle cell anaemia/trait [17], although this was not specifically tested for.

Third, the mean SpO₂ reading for the total data was 94.6%, with a corresponding SaO₂ value of 95.9%. This is consistent with previous investigators’ recommendations for minimal target values for SpO₂ during mechanical ventilation. However, less than 5% of data fell in the range of SpO₂ levels below 90%. Fig. 2 shows increasing positive bias and greater variation as saturations fall. This is consistent with a worsening of bias and precision with pulse oximetry when the SaO₂ is less than 90% [14]. At lower saturations the effects of anaemia and acidosis may become more prominent, and our results should therefore be applied with caution in this situation.

Finally, the pulse oximeter presents SpO₂ data as integers whereas the CO-oximeter presents SaO₂ data to 1 decimal place. With over 1000 data points, it is likely that the oximeter rounded up and rounded down a similar number of times, and so these differences will most likely cancel each other out. At most, the maximum differences due to the measurement of SpO₂ in integers will account for less than 1% of the observed bias as compared with SaO₂.

Conclusion
In conclusion, in a heterogeneous group of ICU patients, we showed that changes in pulse oximetry do not reliably predict equivalent changes in SaO₂. We also demonstrated that neither anaemia nor acidosis alters the precision of measurements between the Nellcor pulse oximeter and CO-oximeter to any clinically important extent. The pulse oximeter remains a valuable tool in the care of intensive care patients, but an awareness of its limitations is an important component of enhancing the quality of care.

Competing interests
None declared.

Acknowledgement
We thank Professor WW Mapleson for advice on statistics.

References