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

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Abstract

Introduction This study investigates the relation between changes in pulse oximeter oxygen saturation (Spo_2) and changes in arterial oxygen saturation (Sao_2) in the critically ill, and the effects of acidosis and anaemia on precision of using pulse oximetry to predict Sao_2.
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 Spo_2 and those in Sao_2, and the pulse oximeter tended to overestimate actual changes in Sao_2. Anaemia increased the degree of positive bias whereas acidosis reduced it. However, the magnitude of these changes was small.
Conclusion Changes in Spo_2 do not reliably predict equivalent changes in Sao_2 in the critically ill. Neither anaemia nor acidosis alters the relation between Spo_2 and Sao_2 to any clinically important extent.

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

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 (Sao_2) 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 (Spo_2) in intensive care patients have reported that the minimum Spo_2 levels to maintain Sao_2 at 90% range between 92% and 96% [4,6,7]. However, these studies have not answered the question of whether, after achieving a target Sao_2, a subsequent change in Spo_2 predicts a corresponding change in Sao_2 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

ICU = intensive care unit; Sao_2 = arterial oxygen saturation; SD = standard deviation; Spo_2 = pulse oximeter oxygen saturation.

also influence the relation between Spo_2 and 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 difference 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 acidotic patient.

We therefore conducted a prospective observational study to test the hypothesis that a change in Spo_2 would predict an equivalent change in 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. Furthermore, we examined the effects of anaemia and acidosis on the precision of using the pulse oximeter to predict the 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 µmol/l) or a history of smoke inhalation; those with an inadequate 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 (ABL 725, Radiometer, Copenhagen, Denmark) that was calibrated daily by laboratory staff and has a 2-hourly automatic internal calibration sequence. Haemoglobin concentration (g/dl), hydrogen ion concentration (nmol/l), and percentage Sao_2 were recorded for each sample. Precision and accuracy of a whole blood sample for Sao_2, hydrogen ion concentration 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 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 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 Ultirx operating system (OSF/1). The changes in residuals were tested for normality and found to be normally distributed. The linear relations between differences in two successive measurements of Spo_2 and 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 comparisons 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.

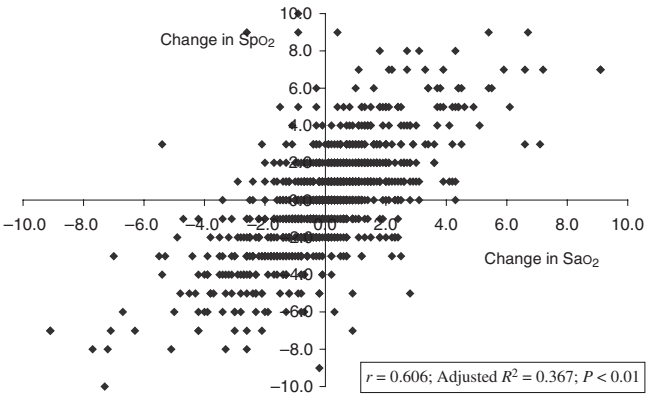
The effects of anaemia and acidosis on the agreement between the two measurement techniques were examined using a Bland–Altman plot [12] in which the difference between Spo_2 and Sao_2 was plotted against their average [13]. Bias and the limits of agreement were calculated. Bias was calculated as the mean of the differences between the CO-oximeter and pulse oximeter readings (Sao_2–Spo_2) [11]. Positive bias indicated that the pulse oximeter underestimated the Sao_2, whereas negative bias indicated that the pulse oximeter was overestimating the Sao_2. The limits of agreement were taken as the bias ± (1.96 × SD) [6,13].

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

Results

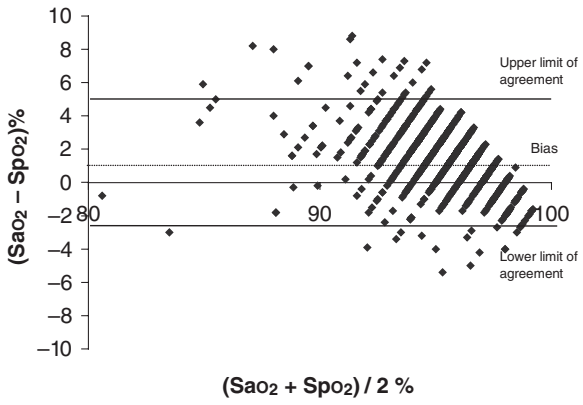
Forty-one (22 male) patients (age [mean ± SD] 70 ± 14 years) were recruited into the study. A total of 1132 simultaneous arterial blood gas and pulse oximeter readings were taken (mean [range] 27 [3–91] readings per patient). Sequential readings in each patient were grouped together into pairs, which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

Figure 1



Linear relations between changes in pulse oximeter oxygen saturation (Spo_2) and arterial oxygen saturation (Sao_2).

Figure 2



Bland and Altman plot for bias and limits of agreement for total data. Sao_2, arterial oxygen saturation; Spo_2, pulse oximeter oxygen saturation.

relation between changes in Spo_2 (ΔSpo_2) and changes in Sao_2 (ΔSao_2).

The mean ± SD for Spo_2 was 94.6 ± 2.7% and the mean for Sao_2 was 95.9 ± 2.4%. In terms of predicting ΔSao_2 from ΔSpo_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 ΔSpo_2 and ΔSao_2 ($r = 0.606$; $P < 0.01$; Fig. 1), only 36.7% of the variation in this relation was due to the association of changes in Spo_2 with changes in Sao_2 (adjusted $R^2 = 0.367$). The prediction of ΔSao_2 from ΔSpo_2 ($\Delta Sao_2 = 0.003 + 0.477 \Delta Spo_2$) demonstrates that the pulse oximeter overestimates actual changes in 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 ≥63 nmol/l ($P < 0.01$), and between haemoglobin concentrations of <8 g/dl, 8–11.9 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 Spo_2 do not reliably predict equivalent changes in Sao_2, with the pulse oximeter tending to overestimate actual changes in Sao_2. We also showed that Spo_2 underestimates Sao_2 to a greater extent with progressive anaemia, whereas acidosis increases the Spo_2 estimate of Sao_2. However, the clinical significance of these changes is small.

Table 1

The effects of anaemia and acidosis on bias and limits of agreement

		Haemoglobin concentration (g/dl)			Hydrogen ion concentration (nmol/l)	
	Total	<8	8–12	>12	25–63	>63
n (measurements)	1132	49	963	120	1064	58
Bias	1.34	2.09*	1.37*	0.72†‡	1.39	0.38§
Upper limit	4.97	7.04	4.87	4.37	4.95	4.61
Lower limit	−2.29	−2.79	−2.14	−2.93	−2.18	−3.85

$P < 0.01$, 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 SpO_2 value. Jubran and Tobin [4], in a study involving 54 ICU patients, reported that levels of 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 SpO_2 of 96% to ensure that no patients had a SaO_2 below 90%. This approach avoided hypoxia, but 15% of patients had a SaO_2 of 98% or greater.

Although target values can be helpful, it would be valuable to know whether a change in SpO_2 would predict a similar change in 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 SaO_2 and SpO_2 might be expected to be closer than that between absolute values from a mixed patient population. This could allow individualized target SpO_2 to be set, based on a single, one-off 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 SpO_2 could not accurately predict changes in SaO_2. Our larger study supports and extends this early finding. The prediction of ΔSaO_2 from ΔSpO_2 ($\Delta SaO_2 = 0.003 + 0.477\Delta SpO_2$) demonstrates that, on average, the pulse oximeter overestimates actual changes in 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 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 (SpO_2 <90%) for the influence of anaemia on bias and precision to be studied in this patient group. However, under normal physiological conditions (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 SpO_2 underestimates 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 *in vivo* [18], whereas 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 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 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-

Key messages

- Changes in SpO_2 do not reliably predict equivalent changes in SaO_2 in the critically ill
- Anaemia and acidosis have only a minor influence on the precision of measurements of SpO_2 and SaO_2

edge had sickle cell anaemia/trait [17], although this was not specifically tested for.

Third, the mean SpO_2 reading for the total data was 94.6%, with a corresponding SaO_2 value of 95.9%. This is consistent with previous investigators' recommendations for minimal target values for SpO_2 during mechanical ventilation. However, less than 5% of data fell in the range of SpO_2 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 SaO_2 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 SpO_2 data as integers whereas the CO-oximeter presents SaO_2 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 SpO_2 in integers will account for less than 1% of the observed bias as compared with SaO_2.

Conclusion

In conclusion, in a heterogeneous group of ICU patients, we showed that changes in pulse oximetry do not reliably predict equivalent changes in SaO_2. 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.

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