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The accuracy of the Biox 3700 pulse oximeter in patients receiving vasoactive therapy

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Abstract. The accuracy of pulse oximetry for the prediction of oxygen saturation of arterial blood in patients with shock has been hardly studied. This study was undertaken to determine if O_2 arterial saturation estimated by the Biox 3700 pulse oximeter (SpO_2) with an ear probe could reliably substitute for the measurements of O_2 saturation (HbO_2) with an IL-282 Co-Oximeter in samples of arterial blood obtained from 24 caucasian patients. All patients were mechanically ventilated and needed vasoactive drugs (dopamine and/or dobutamine). Of 24 patients 13 had shock: cardiogenic ($n = 6$) and septic ($n = 7$). The mean difference between HbO_2 and SpO_2 was 2.49% ± 4.24, with a 95% confidence interval of 0.7% to 4.3% ($p = 0.009$). There were clinically important differences between both methods since in 9 of 24 patients (37%) SpO_2 values were at least 4% lower or greater than HbO_2. This disagreement was also apparent in patients with (6/13, 46%) or without shock (3/11, 27%). To conclude, pulse oximetry is not always a sufficiently reliable method to predict HbO_2 in patients with or without shock treated with vasoactive drugs.

Key words: Pulse oximetry – Shock – Vasoactive drugs

Pulse oximetry is a noninvasive technique, which enables the continuous monitoring of arterial oxygen saturation (SaO_2) of hemoglobin. This technique was introduced in areas such as anaesthesia, intensive care, evaluation of sleep apnea syndrome and exercise testing [1–4]. Nevertheless, it requires a careful evaluation of its accuracy before admitting à priori that its performance is correct [5]. It is known that the output of the pulse oximeter is good in the steady state, but not all commercial equipments can detect the transient changes of arterial oxygenation with the same rapidity and accuracy [6].

It has been demonstrated that the use of pulse oximetry in critically ill patients receiving mechanical ventilation, in whom changes of the oxygen inspiratory fraction (FiO_2) and the positive and expiratory pressure (PEEP) take place, gives a correct estimation of SaO_2, provided that it is done in situations of hemodynamic stability [7, 8]. On the contrary, validation of pulse oximetry in shock patients is quite scarce [9]. A priori, it could be deduced that any decrease in blood flow to the tissues where the probe has been placed, as happens in the clinical setting of shock, could interfere with the correct reading of SaO_2, as pulse oximetry is based on the changes of light absorption secondary to the arterial pulsation.

This study was done to check the accuracy of pulse oximetry in patients with and without shock treated with vasoactive drugs.

Materials and methods

We undertook a prospective study of 24 patients of white race treated with mechanical ventilation (Table 1). Eight patients had cardiogenic shock due to acute myocardial infarction ($n = 7$) or traumatic contusion of the myocardium ($n = 1$). Two patients with cardiogenic shock had complete AV block treated with pacemaker. Sixteen patients presented with septic shock due to: bronchopneumonia ($n = 11$), peritonitis ($n = 4$) and pyelonephritis ($n = 1$). During this study, all the patients needed vasoactive drugs (dopamine and/or dobutamine) to maintain their arterial blood pressure, but only 13 of 24 patients had a clinical state of shock: cardiogenic ($n = 6$) and septic ($n = 7$).

Shock was defined when all of the following criteria were present: hypotension manifested by systolic blood pressure ≤ 90 mmHg, tachycardia (>100 beats per minute), oligo-anuria (urinary output of less than 0.5 ml/min), mental confusion, metabolic acidosis ($pH \leq 7.3$) and arterial hypoxemia (the ratio of PaO_2 to $FIO_2$was <250). Sepsis was diagnosed if at least 4 of the following clinical signs of sepsis were present: fever or hypothermia (temperature >38 °C or <35 °C); tachycardia (>100 beats per minute); tachypnea (respiratory rate >28 breaths per minute) or the need of mechanical ventilation; abnormal WBC count (<3.5 or $>15\times10^3/\mu l$); thrombocytopenia (<100,000/ml) and the presence of an obvious septic site.

Radial arterial catheters were used to monitor the systolic arterial blood pressure and to take samples of the arterial blood. The direct determination of haemoglobin (Hb), oxyhemoglobin ($O_2Hb\%$) and carboxyhemoglobin (CoHb%) were done immediately with an IL-282 Co-oximeter which was calibrated with a reference solution (IL Cal Dye). Its accuracy and precision were checked through a comparison of the Hb value obtained with the IL-282 Co-oximeter and the cyanmethaemo-

Table 1. Summary of clinical data

Sex F/M	1/23
Age (years)	62±11.6
Dopamine (μg/kg/min) $n=20$	15.4±7.7
Dobutamine (μg/kg/min) $n=8$	14±3.1
Systolic BP (mmHg)	90±14.8
HR (beats/min)	108±22
Temperature (°C)	37.4±0.89
Diuresis (ml/min)	0.73±0.85

BP, blood pressured; HR, heart rate

globin method. The arterial blood gases were measured with an IL-1312 blood gas analyser calibrated with tonometered blood.

The non-invasive SaO_2 was measured with a Ohmeda Biox 3700 pulse oximeter using a reusable ear probe. SaO_2 value was provided by pulse oximetry (SpO_2) only when a normal perfusion signal on the pulse oximeter was indicated by a signal-intensity bar graph. The equipment was used in the "fastest" mode (3 s of averaged SaO_2 time). Motion of the probe and high ambient lighting were carefully avoided.

To evaluate the accuracy of the pulse oximeter in predicting HbO_2%, we compared in each patient the values of HbO_2% with SpO_2 values. Both values were obtained after a period of 20 min of stable monitoring with the pulse oximeter (which readings did not oscillate more than 1%).

Statistical analysis

The results are published as mean ±SD. The mean values of SaO_2 obtained with both methods were compared with the Students's *t*-test for paired samples. The linear regression analysis was used to determine the accuracy of the Biox 3700 to predict the HbO_2% measured with the IL-282-Co-oximeter. The lack of agreement between both techniques was analysed according to the methodology proposed by Bland and Altman [10], calculating the mean difference (HbO_2% − SpO_2), the standard deviation of the differences, the 95% confidence interval and the limits of agreement.

Results

The results obtained are summarized in Table 2. The mean difference between the HbO_2% and SpO_2 was 2.49%±4.24, ($p=0.009$) with a 95% confidence interval of 0.7% to 4.3%. The differences within the mean difference ±2 SD (referred as the "limits of agreement") were clinically important since HbO_2% could be 6% lower or 11% greater than SpO_2. In Fig. 1 it is shown that 7 patients had SpO_2 values that were at least 4% lower than HbO_2% and 2 patients had SpO_2 values that were at least 4% higher than their HbO_2% values. Of these 9 patients 6 had shock: septic ($n=3$) and cardiogenic ($n=3$). The regression equation to predict HbO_2% from pulse oximeter saturation was $HbO_2\% = 29.8 + 0.7 \times SpO_2$, standard error of the estimate (SEE) = 3.69 and the correlation coefficient was $r=0.82$ ($p<0.0001$; Fig. 2).

Table 3 summarizes the main results obtained in each group. In 13 patients with shock the mean difference and SD between HbO_2% and SpO_2 was of 1.7%±5.2

Table 2. Arterial blood gases results in 24 patients

FiO_2	0.56±0.31
PO_2 (mmHg)	80±39.9
PCO_2 (mmHg)	39±7.6
pH	7.4±0.07
Hb (g/dl)	12±1.83
CoHb (%)	1.07±0.67
HbO_2 (%)	93.1±6.3
SpO_2 (%)	90.6±7.4

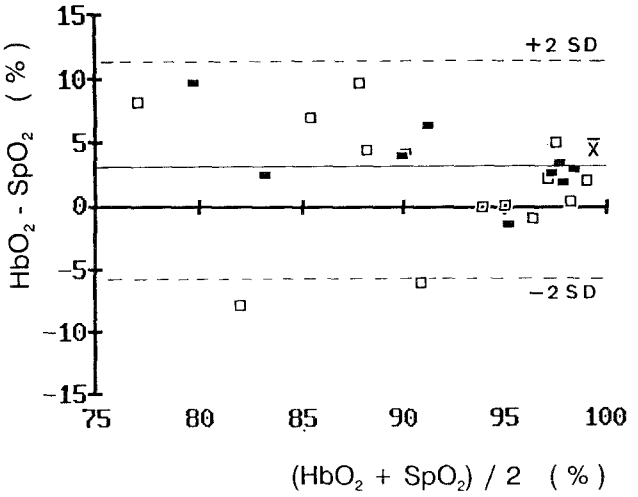

Fig. 1. Differences between HbO_2 (%) and SpO_2 (%), related with their mean values in 24 patients. The mean difference and ±2SD are represented. *Open boxes* represent patients with shock and *closed boxes* patients without shock

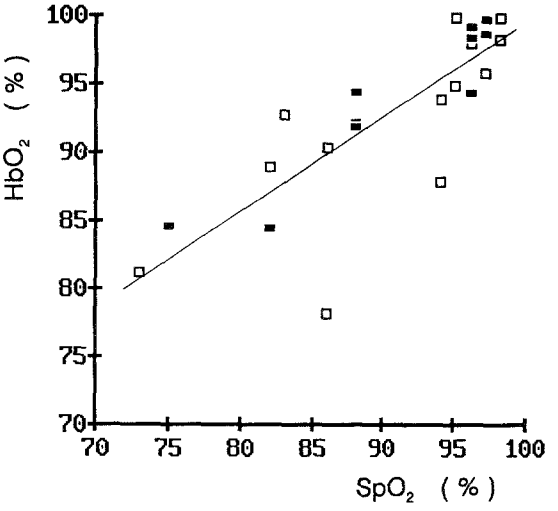

Fig. 2. Correlation between HbO_2 and SpO_2 readings in patients with and without shock. *Open* and *closed boxes* represent patients with and without shock

Table 3. Results of patients with and without shock

	Shock	*n*	Without shock	*n*
Dopamine (μg/kg/min)	17±6.2	11	13±8.9	9
Dobutamine (μg/kg/min)	14±2.9	6	13±4.9	2
Systolic BP	79±12	13	101±6	11*
HR	104±21	13	112±22	11
Diuresis	0.3±0.35	13	1.3±0.94	11**
FiO_2	0.6±0.33	13	0.5±0.3	13
HbO_2%	92.2±6.9	13	94.2±5.6	13
SpO_2	90.5±7.8	13	90.8±7.3	13

*$p=0.008$, **$p=0.0001$

($p = 0.25$) and 3.4% ± 2.8 ($p = 0.002$) in the 11 patients without shock. Of the 13 patients with shock 6 (46%) and of 11 patients without shock 3(27%) had SpO_2 readings that were at least ±4% different than HbO_2%. No relationship was found among systolic blood pressure, drug dosage with the mean difference of $HbO_2\% - SpO_2$.

Discussion

The results obtained in this study show that there can be a considerable discrepancy between the measurement of arterial O_2 saturation by an IL-282 Co-oximetry and its estimation with the Biox 3700 pulse oximeter. Pulse oximetry gave a significantly lower mean value than Co-oximetry. In 9 of 24 (37%) patients there was considerable disagreement between both techniques, which does not allow acceptance of pulse oximetry as a technique able to confidently predict SaO_2 in patients treated with vasoactive drugs, whether or not in clinical shock.

None of our patients had dark skin pigmentation changes [11] or high levels of carboxyhaemoglobin [12] which could have interfered in the correct reading of the oximeter. Jubran and Tobin [13] have found inaccurate pulse oximetry readings (with a difference >4% between SpO_2 and HbO_2) in 6 of 55 (11%) critically ill normotensive patients of white race and in 12 of 45 (27%) black patients. In that study, the accuracy of pulse oximetry deteriorated when $HbO_2$was ≤90%, which is in conflict with studies achieved with healthy volunteers, where oximetry keeps its accuracy until SaO_2 is lower than 75% [14].

Very recently, Severinghaus and Spellman [15] tested the limitations of pulse oximetry when systolic blood pressue is lower than 60 mmHg. Using non-disposable finger probes of 3 oximeters in 9 normal volunteers, they detected a decrease in the Ohmeda 3740 oximeter readings during normoxia (from 97.5% to 87%) and a delay of 6 min in the detection of an induced hypoxic transient. These authors using a finger plethysmograph also found that when blood flow decreased to zero, the pulse oximeter continued to display a good perfusion signal. Mihm et al. [9] recorded pulse oximetry with a finger probe in 9 patients treated with vasoactive drugs who had a mean arterial pressure lower than 60 mmHg, and found an adequate correlation ($r = 0.97$) to the oximetry of an arterial blood sample. Nevertheless, this study does not specify the regression equation, and neither the bias nor the precision.

Though our patients did not demonstrate hypothermia, this is another factor that can change the efficiency of the pulse oximeter increasing the detection time for induced hypoxemia even if there is a normal pulse signal [16]. The reliability of the pulse oximeter responses to a rapid changing situation of arterial saturation in patients with shock is still to be investigated. Severinghaus [6] has demonstrated that ear probes are more accurate than finger probes in normal volunteers but we did not detect an accurate response of the Biox 3700 pulse oximeter to accept it as clinically useful when patients are treated with vasoactive drugs.

The conclusion of this study is the following: pulse oximetry with an ear probe is not always capable of reliably detecting the arterial oxygen saturation in patients with or without clinical shock treated with vasoactive drugs.

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