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Accuracy of pulse oximetry in the intensive care unit

Received: 16 November 2000 Final revision received: 5 July 2001 Accepted: 19 July 2001 Published online: 28 August 2001 © Springer-Verlag 2001

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A. Harf Physiology Department, Henri Mondor Hospital, Assistance Publique-Hôpitaux de Paris, University Paris XII, Créteil, France **Abstract** *Objective*: Pulse oximetry (SpO₂) is a standard monitoring device in intensive care units (ICUs), currently used to guide therapeutic interventions. Few studies have evaluated the accuracy of SpO₂ in critically ill patients. Our objective was to compare pulse oximetry with arterial oxygen saturation (SaO₂) in such patients, and to examine the effect of several factors on this relationship.

Design: Observational prospective study.

Setting: A 26-bed medical ICU in a university hospital.

Patients: One hundred two consecutive patients admitted to the ICU in whom one or serial arterial blood gas analyses (ABGs) were performed and a reliable pulse oximeter signal was present.

Interventions: For each ABG, we collected SaO2, SpO₂, the type of pulse oximeter, the mode of ventilation and requirement for vasoactive drugs.

Measurements and results: Three hundred twenty-three data points were collected. The mean difference between SpO₂ and SaO₂ was -0.02 % and standard deviation of the differences was 2.1 %. From one sample to another, the fluctuations in SpO₂ to arterial saturation difference indicated that SaO₂ could not be reliably predicted from SpO₂ after a single ABG. Subgroup analysis showed that the accuracy of SpO₂ appeared to be influenced by the type of oximeter, the presence of hypoxemia and the requirement for vasoactive drugs. Finally, high SpO₂ thresholds were necessary to detect significant hypoxemia with good sensitivity. Conclusion: Large SpO₂ to SaO₂ differences may occur in critically ill patients with poor reproducibility of SpO₂. A SpO₂ above 94 % appears necessary to ensure a SaO₂ of 90 %.

Keywords Pulse oximetry · Monitoring · Oxygen saturation · Intensive care

Introduction

The human eye is poor at recognizing hypoxemia [1], and the detrimental effects of this condition have long been recognized [2, 3, 4]. Efforts have thus been made to develop a method for monitoring arterial oxygen saturation (SaO₂) [5]. With the widespread use of pulse oximeters in operating rooms, perioperative hypoxemia was found to be much more common than previously suspected [6] and, despite the lack of definite evidence,

the generalized use of pulse oximetry (SpO₂) has probably reduced perioperative morbidity and mortality.

In intensive care units (ICUs), pulse oximetry has become a standard monitoring device and therapeutic interventions are frequently based on the SpO₂ values [7, 8]. Nevertheless, few large scale studies are available on the accuracy of SpO₂ in critical care patients. A good agreement between SpO₂ and the reference method of arterial blood gas analysis (ABG) has been reported by some authors [9], but not always found by others

[10, 11]. Some studies have pointed out that SpO₂ may not always be a reliable method to predict SaO₂ [7, 12] and that target SpO₂ values used by the physicians could result in significant hypoxemia. Moreover, decreased accuracy of SpO₂ has been described in hypoxemic [10] or hemodynamically compromised patients [11, 13], in whom an accurate and reliable monitoring is of major importance.

We therefore undertook a prospective observational study to determine the accuracy of SpO₂ in an ICU population. We also focused on subgroups of patients to determine whether the type of pulse oximeter, the presence of mechanical ventilation, the presence of hypoxemia or the need for vasoactive drugs could influence the accuracy of SpO₂. Finally, we tested the reproducibility of this technique to determine whether an initial SpO₂ to SaO₂ difference could be extrapolated to subsequent measurements.

Materials and methods

This observational study was undertaken in the medical ICU at the university hospital Henri Mondor (Paris, France). This unit is a general, multidisciplinary medical ICU with patients admitted for a wide spectrum of diagnoses.

Patients

All patients admitted to the ICU between June and October 1999 and for whom an ABG was prescribed by the attending physician, were selected for inclusion in this study. Patients were not included if the plethysmographic waveform or the signal of the moving light bar of the SpO₂ were of poor quality (by visual analysis of a flat or irregular signal waveform or when intermittent display was present). No ABG was performed specifically for the study if not needed by the patient's physical status. No patient was suspected of having a significant level of methemoglobin or carboxyhemoglobin

Pulse oximeters

Three pulse oximeters were used in the ICU, all with finger probes: Hewlett-Packard Viridia 24C (Hewlett-Packard, Boeblingen, Germany), Nellcor N-200 (Nellcor, Hayward, Calif., USA) and Ohmeda 3700 (Ohmeda, Boulder, Colo., USA).

Data collected

For each patient, an arterial sample was withdrawn from the radial artery and the finger probe of the pulse oximeter was placed on the same side. After waiting for a stable plethysmographic waveform or moving light bar signal, the SpO₂ value was recorded by the nurse and the arterial blood sample was obtained simultaneously. Arterial oxygen saturation was measured using a hematoxymeter (ABL 520, Radiometer, Copenhagen, Denmark), and the SaO₂ value was recorded subsequently. For patients requiring multiple ABGs during their ICU stay, each data point was collected. Mode

of ventilation was recorded as spontaneous or assisted (non-invasive ventilation, assisted controlled ventilation, pressure support ventilation). The type of pulse oximeter and the requirement for vasoactives drugs (dopamine higher than $5\mu g/kg$ per min, epinephrine or norepinephrine) were also noted.

Statistical analysis

To assess agreement between SpO_2 and the ABG, we used the method described by Bland and Altman [14], calculating the mean difference (bias, d) and the standard deviation of the differences (precision, s) between SpO_2 and hematoxymeter, and the limits of agreement $(d \pm 2s)$.

The reproducibility of the difference between SpO_2 and SaO_2 was analyzed graphically using a linear regression model. For all patients having more than two ABGs, the three data points $[SpO_2; SaO_2]$ were considered. Three SpO_2 to SaO_2 differences were obtained $(SpO_2\text{-}SaO_2=\Delta)$, represented as $\Delta 1$, $\Delta 2$ and $\Delta 3$ (in chronological order). Two graphs were constructed, the first representing $\Delta 2$ (y-axis) versus $\Delta 1$ (x-axis), the second representing $\Delta 3$ (y-axis) versus $\Delta 2$ (x-axis).

A Student's *t*-test was used for comparison of SpO₂ to SaO₂ values between selected subgroups of patients. To assess the sensitivity and specificity of the SpO₂ to detect hypoxemia, we chose three thresholds of SaO₂ (90%, 92% and 95%), based on published studies and recommended objectives [15, 16, 17]. For each threshold of SaO₂, which we represented with the letter "A" for the calculations below, we tested all SpO₂ values obtained in the study and calculated for each SpO₂ value, represented by the letter "B" in the calculations, the sensitivity and specificity of this cut-off point B for SpO₂ to detect a SaO₂ of A or less, as follows:

Sensitivity 0 (data points with $SaO_2 \le A$ and $SpO_2 \le B$)/(data points with $SaO_2 \le A$)

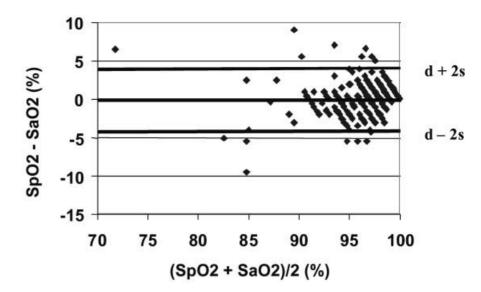
Specificity = (data points with $SaO_2 > A$ and $SpO_2 > B$)/(data points with $SaO_2 > A$)

Then, we determined an "optimal SpO $_2$ " with the help of receiver operating characteristic (ROC) curves [18]. The ROC curves plot all possible combinations between the true-positive ratio (sensitivity; y-axis) and the false-positive ratio (1– specificity; x-axis) as one varies the definition of positivity from SpO $_2$ 0 to SpO $_2$ 100%. The axes of this graph both range from 0 to 1 because these are the limits of possible true-positive and false-positive ratio values. ROC curves therefore made it possible to determine the cut-off value corresponding to the best compromise between sensitivity and specificity, defined as the point of the curve closest to the upper left-hand corner ("optimal" SpO $_2$). The SpO $_2$ corresponding to this point was represented by the letter "C" for calculations below. Positive (PPV) and negative (NPV) predictive values of this optimal SpO $_2$ (C) were also calculated for each SaO $_2$ threshold (A) as follows:

PPV = (data points with $SaO_2 \le A$ and $SpO_2 \le C$)/(data points with $SpO_2 \le C$)

PPV = (data points with $SaO_2 \le A$ and $SpO_2 \le C$)/(data points with $SpO_2 \le C$)

Fig. 1 Graphic representation, as described by Bland and Altman [14], comparing pulse oximetry and arterial oxygen saturation. For each data point (black diamonds), the mean value $((SpO_2 + SaO_2)/2)$ figures on the x-axis, and the difference value (SpO₂ to SaO₂) on the yaxis. Black lines represent the 95% confidence interval for SpO_2 (d ± 2s), where d is the bias (mean SpO₂ to SaO₂) and s the precision (standard deviation of the SpO₂ to SaO₂ differences). Bias is -0.02 % and precision is 2.10%



Subgroup analysis

To address the influence of different factors on the accuracy of SpO_2 , we performed a retrospective post hoc analysis, separating the patients on the basis of the type of pulse oximeter, the presence or absence of vasoactive drugs, the presence or absence of assisted ventilation and a SaO_2 above or below 95 %.

Results

Patients

A total of 102 patients were included in the study, representing 323 data points. Median values with 95% confidence intervals of SpO₂ and SaO₂ were 97% (90.4%;100%) and 97.3% (91.1%;100%), respectively. The ranges of SpO₂ and SaO₂ values were (75 %;100 %) and (68.5%;100%), respectively. Modes of ventilation were distributed as follows: spontaneous ventilation 83 patients (200 data points) and assisted ventilation 62 patients (123 data points). Thirteen patients required vasoactives drugs, representing 36 data points. The distribution of oximeters was as follow: 68 patients were monitored with a Hewlett-Packard (202 data points), 21 patients with Ohmeda system (21 data points) and 37 patients with Nellcor system (76 data points). Only 299 data points among the total of 323 were reported in the distribution of oximeters, because description of the oximeter by the nurse was omitted for the other 24 data pairs. The results were similar when only these 299 data points were considered. The sum of patients of the different subgroups is more than 102 because some patients had successive oximeters or a change in the mode of ventilation.

Accuracy of pulse oximetry

For the total population, the mean difference between SpO_2 and SaO_2 values (bias, d) was $-0.02\,\%$, and standard deviation of the differences (precision, s) was 2.1 %. A graphic representation, figuring limits of agreement (d \pm 2s) and individual values, is shown in Fig. 1.

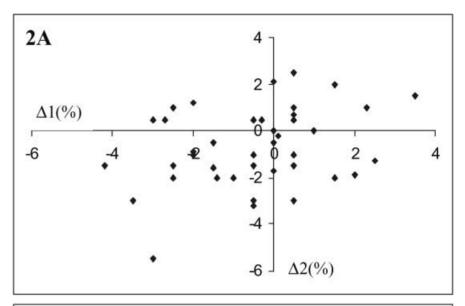
Reproducibility of the difference between pulse oximetry and arterial saturation

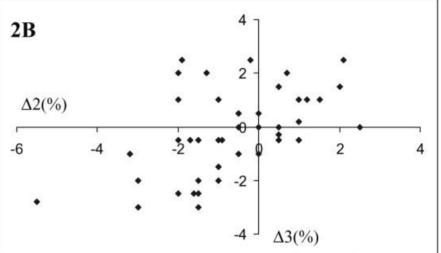
We questioned whether measurement of an initial (SpO₂ to SaO₂) difference (Δ) may be used to evaluate SaO₂ from subsequent SpO₂ values in the time course of an ICU stay. This hypothesis was tested in 45 patients who had more than two ABGs. For each patient, we obtained three (SpO₂ to SaO₂) differences: $\Delta 1$, $\Delta 2$ and $\Delta 3$. We compared $\Delta 2$ versus $\Delta 1$ (Fig. 2a) and $\Delta 3$ versus $\Delta 2$ (Fig. 2b) using a linear regression model. If the Δ had been constant from measurement 1 to measurement 2 and/or from measurement 2 to measurement 3, a low dispersion would have been obtained. However, a large dispersion of the plots in the two axes was observed, indicating that SaO₂ cannot be predicted from SpO₂ more accurately by considering the result of a previous ABG. Thus, the SpO₂ to SaO₂ difference is not reproducible, neither in the magnitude nor in the direction.

Subgroup analysis

Biases were compared among the different subgroups of patients and are represented in Table 1. The accuracy of SpO₂ appeared to be influenced by the type of oximeter, the presence of hypoxemia and the need for vasoactive

Fig. 2 Graphic representation of the reproducibility of pulse oximetry in 45 patients having three data points (SpO₂; SaO₂). The three pulse oximetry to arterial oxygen saturation (SpO₂-SaO₂ = Δ) differences (Δ 1, Δ 2, Δ 3)(%) are represented; (**A**) represents Δ 2 versus Δ 1 and (**B**) represents Δ 3 versus Δ 2





drugs. The bias was not statistically different between patients requiring assisted mechanical ventilation and those breathing spontaneously.

Receiver operating characteristic curves

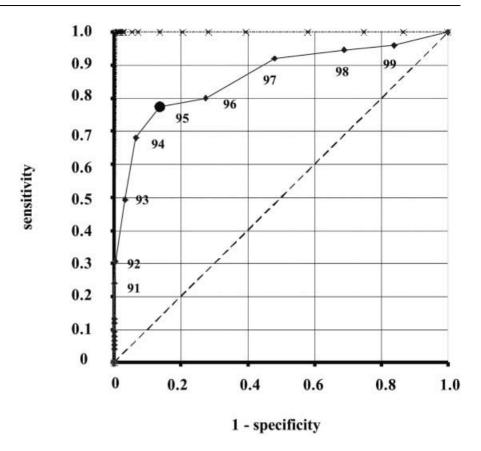
As an example, the ROC curve for a SaO₂ of 95% is represented in Fig. 3. Optimal thresholds for SpO₂, as determined by the ROC curves for each SaO₂ threshold, are represented in Table 2, with respective values of sensitivity, specificity, positive and negative predictive values. The numbers of data points with SaO₂ 90% or less, 92% or less and 95% or less were 13, 22 and 75, respectively. An SaO₂ higher than 90% is frequently recommended as a clinical objective during mechanical ventilation or oxygen therapy: to detect a SaO₂ of 90%

or less, we found that SpO_2 of 89%, 91%, 92%, 93% and 94% had sensitivities of 64%, 71%, 71%, 79% and 86%, respectively, and specificities of 100%, 97%, 95%, 89% and 82%, respectively. For a SpO_2 of 94%, however, the negative predictive value was 99%.

Discussion

Because the deleterious effects of perioperative hypoxemia have been recognized [2, 3], the use of SpO₂ has rapidly become common in operating rooms. A prospective, randomized study of the effect of pulse oximetry on the outcome of anesthesia care in 20,802 surgical patients demonstrated a 19-fold greater increase in the detection of hypoxemia in the oximeter group, and that myocardial ischemia was more common in the control

Fig. 3 Receiver operating characteristic curve for $SaO_2 = 95\%$, representing the sensitivity (y-axis) and (1–specificity) (x-axis) of all cutoff points of SpO_2 to detect a SaO_2 of 95% or less. SpO_2 cutoff values are indicated beside the *black diamonds*. The *black circle* represents the best "compromise" for a SpO_2 target, defined as the point of the curve closest to the upper left-hand corner



group versus the oximetry group [19]. Despite the lack of studies demonstrating a significant reduction in post-operative mortality, it is likely that the use of SpO₂ has made anesthesia safer.

This monitoring was extended to ICUs. Potential applications included increased detection of hypoxemia by continuous monitoring of SpO_2 or titration of FIO_2 in ventilator-dependent patients [7]. Some authors demonstrated a reduction of ABGs through the use of SpO_2 [9,

20], leading to a decrease in complications of arterial punctures and decreased economic costs. However, several studies have pointed out the poor accuracy of SpO₂ in hypoxemic or hemodynamically unstable patients. Finally, few studies have evaluated the accuracy of SpO₂, compared with ABG, in large ICU populations.

Our study included 102 surgical and medical patients and 323 ABGs. We chose to compare SaO₂ and SpO₂ according to the method described by Bland and Altman,

Table 1 Bias (mean SpO₂–SaO₂) (%) of the pulse oximetry in the different subgroups of patients. The bias was significantly increased in patients with SaO₂ 95% or less or on vasoactive drugs. The bias was significantly different in the Hewlett-Packard group

compared with the Nellcor and Ohmeda groups, and also significantly different in the Nellcor group compared with the Ohmeda group (Student's *t*-test)

Parameter	Subgroup	n	Bias (%)	p	Precision (%)
Mode of ventilation	Spontaneous Assisted	200 123	-0.08 0.08	0.48	2.13 2.04
Oximeter	Hewlett-Packard Nellcor Ohmeda	202 76 21	-0.07 0.50 -1.28	< 0.05	2.09 1.63 2.50
Vasoactive drugs	Yes No	36 287	0.70 -0.11	< 0.05	2.06 2.09
SaO ₂ (%)	≤95 % > 95 %	75 248	0.57 -0.20	< 0.05	2.98 1.71

Table 2 Ability of pulse oximetry to detect hypoxemia, defined as $SaO_2(\%)$ below three predefined thresholds. Optimal SpO_2 , representing the best compromise between sensitivity and specificity to detect hypoxemia, was determined graphically upon ROC curves

(cf. Fig. 3). For each SaO_2 threshold, a respective value of optimal SpO_2 is reported, with its sensitivity, specificity, positive and negative predictive values

SaO2 threshold (%)	Optimal SpO ₂ (%)	Sensitivity (%)	Specificity (%)	Positive predictive value (%)	Negative predictive value (%)
95	95	78	86	63	93
92	93	83	91	42	99
90	94	86	82	18	99

because linear regression is not adequate to compare a new measurement technique with an established one. In the total population, the bias (mean difference between SpO₂ and SaO₂) is -0.02% and the precision (standard deviation of the differences) is 2.1 %. This is in agreement with results obtained in previous studies in intensive care. The bias was 1.7% and the precision 1.3% in 35 cardiac surgical patients studied by Bierman et al. [9]. In the study by Ibanez et al. [13] the bias was 2.5% in 24 patients receiving vasoactive drugs. Another study evaluated the reliability of six pulse oximeters in chronic obstructive pulmonary disease with biases varying from 0.4% to 3.6% and precisions from 2.2% to 3.9% [21]. The bias of -0.02% in our study appears to be clinically acceptable, but a precision of 2.1% with a 95% confidence interval of (-4.22%; 4.18%) indicates that a clinically relevant difference may exist between SpO₂ and SaO₂. These results are in accordance with the manufacturers' specifications concerning the performances of the oximeters. Nevertheless, intensive care patients are often hypoxemic and unstable: if the SaO₂ of a patient is 90%, SpO₂ measured by pulse oximetry may vary from 86% to 94%, potentially leading to different diagnostic or therapeutic approaches.

To test the hypothesis that, for a given patient, the difference between SpO₂ and SaO₂ may be predictable over time, we graphically represented successive SaO₂ to SpO₂ differences and found that this difference was poorly reproducible. A reproducible difference would have allowed the avoidance of multiple arterial punctures in the same patient. Two studies have pointed out the potential cost-savings for SpO₂. Bierman observed that the availability of SpO₂ data at the bedside allowed a significant reduction in arterial blood gas utilization [9]. A study realized before and after oximetry in 300 critical care patients found a significant reduction in arterial blood gas determinations with SpO₂, mostly in surgical patients [20]. Although relevant, these results were obtained in a majority of surgical patients, without severe respiratory or hemodynamic impairment. Our results showed that, beyond the large limits of agreement of SpO₂, the SpO₂ to SaO₂ difference may vary greatly for a given patient over time.

Observing the modest accuracy of SpO₂ in the total population, we focused on selected subgroups of pa-

tients. Indeed, several studies have emphasized that severe hypoxemia, shock or type of oximeter may decrease the accuracy of SpO₂. Hannhart et al. have demonstrated lower bias and precision with newer oximeters, compared with older instruments, in chronic obstructive pulmonary patients [21]. In a pediatric study of 66 patients comparing two pulse oximeters, performance of the Ohmeda oximeter deteriorated below an SpO₂ of 75%, whereas the Hewlett-Packard oximeter performed consistently above, as well as below, an SpO₂ of 75%, and was associated with a lower bias [12]. Our study included three pulse oximeters and only patients with acceptable plethysmographic waveform: our results found a lower bias for the Hewlett-Packard instrument compared with the other two, and a lower bias for the Nellcor oximeter compared with the Ohmeda. This emphasized the fact that the accuracy of SpO₂ may also depend of the algorithms used by the manufacturers.

The influence of hemodynamic instability on the accuracy of SpO₂ has been poorly evaluated. The study by Ibanez et al. included 24 patients treated with vasoactive drugs [13]. The bias was 2.5 % and, in 9 of 24 patients, SpO₂ values were at least 4% lower or higher than SaO₂. Despite the lack of a control group, the authors concluded that the reliability of SpO₂ in patients receiving vasoactive drugs is poor, comparing their results with previously published studies. In patients with septic shock, other authors demonstrated a significant underreading of the SpO₂ in the group with low or normal systemic vascular resistance [22]. They hypothesized that this underreading was due to the oximeter's reading the pulsatile venous flow due to the opening of arteriovenous channels in septic patients. Our study also shows a significant increase of the bias in patients receiving vasoactive drugs; this result cannot be explained by a poor signal on the finger probe, because all patients with abnormal plethysmographic waveform were excluded.

An impaired accuracy of SpO_2 at low oxygen saturation has also been emphasized in several studies. Severinghaus, inducing profound, transient hypoxemia in normal volunteers, observed mean errors greater than 6% and a standard deviation greater than 10% with finger probes [23]. Jubran et al., in 54 ventilated patients, found a bias of 1.7% for SaO_2 above 90% and 5.1%

for SaO₂ below 90% (p < 0.0001); the precisions were 1.2% and 2.7%, respectively [7]. Another author concluded that the performance of the Ohmeda pulse oximeter deteriorated below an SpO₂ of 75% in a group of 66 patients with arterial saturation less than 90% [12]. One explanation is thought to be the difficulty of obtaining reliable human calibration data during extreme hypoxia [5]. Other factors are probably implicated, since we found a significant difference in the bias as soon as SaO₂ fell below 95%, not an extremely low value.

In our study the presence of cardiac arrhythmias or anemia was not specifically assessed as a factor influencing the accuracy of pulse oximetry. Indeed, the performance of SpO₂ was examined in 163 patients in a surgical ICU and no difference in bias was observed between patients with or without cardiac arrhythmias [24]. Concerning anemia, one study found a bias of only 0.53 % in patients with a mean hemoglobin level as low as 5.2 g/dl [25]. Another author reported performances of pulse oximetry in severe anemia comparable to those generally reported in non-anemic patients [26]. Moreover, in our study, no patient had a hemorrhagic diathesis and no hemoglobin level below 7 g/dl was noted.

In clinical trials of patients with the acute respiratory distress syndrome (ARDS), SaO₂ was often monitored by pulse oximetry, with a relatively low target range for SpO₂, from 86% to 94% [8, 27]. This target was used to titrate the FIO₂ or the positive end-expiratory pressure. A blood gas analysis was not always available to ensure that an acceptable PaO₂ or SaO₂ was achieved. We focused on predefined thresholds of SaO₂ (90%, 92% and 95%), considered as acceptable goals by sev-

eral authors [15, 16, 17], to determine the sensitivity of SpO₂ to detect a SaO₂ below the threshold. We found a relatively low sensitivity of SpO₂, whatever the SaO₂. To detect a SaO₂ of 90% or less, a SpO₂ of 90% or less was associated with a sensitivity below 70%. Thus, the use of a SpO₂ target of 90% would be associated with a significant risk of undiagnosed hypoxemia. Our results are in agreement with a recently published study [28] on 33 surgical intensive care patients, where 111 values of SpO₂ were compared with corresponding SaO₂s. Using two different oximeters, the authors concluded that a threshold of SpO₂ of 96% was necessary to ensure a SaO₂ higher than 90%.

Our study showed that a SpO₂ threshold of 94% was associated with a negative predictive value of 99% for a SaO₂ of 90%. Thus, 99% of our patients with a SpO₂ higher than 94% had a SaO₂ higher than 90%. If a SaO₂ higher than 90% is considered as a clinical objective in patients receiving mechanical ventilation, changes in the FIO₂ with SpO₂ persistently above 94% does not necessarily require an ABG to avoid severe hypoxemia; this leads to potentially significant economic costs-saving.

In conclusion, our study on a large population of medical intensive care patients showed that the agreement between SpO₂ and SaO₂, although within the limits ensured by the manufacturers, may be clinically insufficient. Large SaO₂ to SpO₂ differences may occur, especially in the most severe patients, with poor reproducibility among sequential measurements. Incorporating a low SpO₂ target in a decision-making process should be done cautiously.

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