Comparison between Calculated O₂ Saturation Values and Those Determined Directly In Vitro (Blood Gas Analyzer vs. Oxymeter)

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Introduction

Most blood gas analyzers (BGA) on the market today offer the possibility of calculating the so-called partial O₂ saturation (pO₂, %) of arterial blood from the measured values for pO₂, pH and pCO₂ using formulas derived by Kelman, Severinghaus or Siggaard-Andersen [1]. A common feature of all these methods is that an actual O₂ binding curve is derived from the acid-base status data, which together with the measured pO₂ permits calculation of the pSO₂ (%). Since calculation by the BGA does not allow the actual concentrations of COHb and MetHb to be taken into account, values are obtained for sO₂ that represent the partial sO₂ (pO₂). This describes the amount of Oxy-Hb as a percentage of the sum of only oxy- plus deoxy-hemoglobin. In addition, there is a danger of erroneous calculations by the WGA since no data are available concerning shifts in the O₂ binding curve that might result in changes in acid-base status. For this reason, the O₂ saturation calculated by a representative blood gas analyzer (Ciba Corning 175) is compared with the actual sO₂ measured in vitro using a modern multi-wavelength oxymeter (CO-oxymeter 2500, Ciba Corning). This comparison of methods can also be considered to be a practical example of the diagnostic value of the partial O₂ saturation (pO₂) and the true O₂ saturation (sO₂).
Methods

Venous blood (heparinized) from 6 smokers and 8 non-smokers was first adjusted to various BE values (-10, ±10, +10 mmol/l) at 37°C in a tonometer (ILL 237, Instrumentation Laboratory) and then equilibrated using a gas mixing apparatus (Ciba Corning 102) with CO₂ partial pressures of 20, 40 and 60 mmHg, and O₂ partial pressures of 27, 40 and 60 mmHg. The O₂ saturation was on the one hand calculated for each blood sample from the values measured in triplicate for pO₂, pH and PO₂ using a blood gas analyzer (Ciba Corning 176) and on the other hand measured with an oximeter (CO₂-oxymeter 2500; Ciba Corning). Means were calculated from three separate determinations for each sample.

Results

The results are presented separately for smokers and non-smokers in figures 1 and 2; each symbol represents the mean from three separate measurements.

In the non-smokers, good agreement was obtained between the calculated, i.e. partial O₂ saturation, and the measured O₂ saturation. Calculation led to a slight underestimation of the O₂ that represented around 1% in the upper and 2% in the lower saturation range. In the smokers this agreement between calculated and measured O₂ saturation was no longer observed; the true O₂ saturation was clearly overestimated in the upper and underestimated in the lower saturation range. In contrast to the mean COHB concentration in non-smokers of only 1.3 ± 0.5%, the smokers (blood taken in the morning) showed a mean COHB concentration of 7.5 ± 1.4%.

Discussion

As was to be expected, the blood gas analyzer can only calculate the O₂ saturation correctly when besides Hb and O₂-Hb no COHB or MetHb are present. In non-smokers a relatively good agreement is therefore obtained between measured and calculated O₂, since in this case the partial and true O₂ values are practically the same. Compared to the saturation values expected for blood samples with a BE of ±10 mmol/l and a pCO₂ of 40 mmHg (corresponding to a so-called standard O₂ binding curve [3]), no significant differences were observed in non-smokers after equilibration with a given pO₂ (data not shown). For instance, the O₂-
saturation at a $pO_2$ of 27 mmHg gave a mean value of 52.8% compared with an expected value of between 50 and 51% based upon the standard $O_2$ binding curve; the value calculated from the blood gas analyzer data was, instead of 52.8%, just 50.9% (cf. regression line in fig. 1).

Thus the methods used and the data obtained in the absence of COHb and MetHb permitted the calculation of $sO_2$ with a blood gas analyzer despite the slight underestimation of $sO_2$ obtained by calculation (cf. fig. 1).
The situation changes in more than one sense in the presence of significant concentrations of COHb or MetHb (MetHb has not been investigated here). In contrast to the non-smokers investigated (COHb 1.3%), the smokers displayed a mean COHb concentration of 7.5% (in blood samples taken in the morning). As was to be expected, the measured true sO₂ (CO-oximeter) of such blood samples, dropped from 97% to 89%, when these had been equilibrated at pCO₂ = 40 mmHg and BE ± 0 mmol/l with a pO₂ of 90 mmHg. Calculation of the sO₂, however, must
lead, on the basis of the regression line shown in figure 2, to a significant overestimation, i.e. of 94.8% instead of the actual value of 99%. This overestimation of the SO2 in the upper saturation range obtained by calculation in smokers is due to the fact that only the so-called partial SO2 (pSO2) can be calculated, and not the true SO2.

The second problem encountered in calculating the pO2 from the pO2, pCO2 and pH is the fact that only the shift in the O2 binding curve which is due to changes in the acid-base status can be taken into account. In the case of the smokers, however, there was an independent leftward shift of the O2 binding curve due to CO, which should be described by a characteristic calculated factor (not presented here).

Blood samples that have been equilibrated at a pO2 of 27 mmHg (pCO2 = 40 mmHg, BE = ± 0 mmol/l) should show an SO2 of 46% in the case of a normal O2 binding curve and a COHb concentration of 7.5%. The actual mean value measured in the CO-oxymeter was 54.3%, indicating a significant leftward shift. However, instead of the m-measured value of 54.3%, the blood gas analyzer yields a value of 52.2% (cf. regression line in fig.32) since it calculates the SO2 on the assumption of a standard O2 binding curve. The underestimation of the saturation in the lower saturation range in smokers is therefore due to the fact that, instead of the actual O2 binding curve, only a standard O2 binding curve can be used as a basis for calculation.

The relative large scatter of the data shown in figures 1 and 2 is not so much due to variation among the values measured in the CO-oxymeter, but rather to that of the values calculated by the blood gas analyzer. Since calculation of the saturation is based upon measured values for pO2, pCO2 and pH, all of which display experimental scatter, these variations can accumulate in the final calculation.

The comparison by Breezer et al. [1] of measured and calculated values of O2 saturation can only in part be referred to here. Thus, these authors compared the partial, calculated SO2 (based upon a total of three calculation formulas) with the values measured in the Hem-oxymeter OSM2 (Radiometer), also representing the partial SO2. Since this older equipment only uses two wavelengths it can optimally only measure the partial SO2 if COHb cannot be detected photometrically.

The comparison of methods published by Marian et al. [2] does not allow a comparison between calculated (blood gas analyzer IL 1302, Instrumentation Laboratory) and measured (CO-oxymeter Cibus Corning) O2 saturation values since the actual measured values are not presented.
(the report that the calculated \( sO_2 \) is 2.7% higher than the measured \( sO_2 \) cannot be assigned to any particular range; no information is given on cCOHb).

Summary

The (partial) \( O_2 \) saturation calculated by a blood gas analyzer from measured values for \( pO_2 \), \( pCO_2 \), and \( pH \) is compared with the actual \( sO_2 \) values measured in smokers and non-smokers using a CO-oximeter after equilibration of blood samples. Whereas good agreement was found between calculated and measured saturation values in non-smokers, calculation of the saturation in smokers leads to a significant overestimation in the upper saturation range and to a significant underestimation of the \( sO_2 \) in the lower saturation range, compared to the measured (true) values.

References