

## Comparison between Calculated O<sub>2</sub> 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 O<sub>2</sub> saturation (psO<sub>2</sub>, %) of arterial blood from the measured values for pO<sub>2</sub>, pH and pCO<sub>2</sub> using formulas derived by Kelman, Severinghaus or Siggaard-Andersen [1]. A common feature of all these methods is that an actual O<sub>2</sub> binding curve is derived from the acid-base status data, which together with the measured pO<sub>2</sub> permit calculation of the psO<sub>2</sub> (%). Since calculation by the BGA does not allow the actual concentrations of COHb and MetHb to be taken into account, values are obtained for sO<sub>2</sub> that represent the partial sO<sub>2</sub> (psO<sub>2</sub>). 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 BGA since no data are available concerning shifts in the O<sub>2</sub> binding curve that might result in changes in acid-base status. For this reason, the O<sub>2</sub> saturation calculated by a representative blood gas analyzer (Ciba Corning 178) is compared with the actual sO<sub>2</sub> 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 O<sub>2</sub> saturation (psO<sub>2</sub>) and the true O<sub>2</sub> saturation (sO<sub>2</sub>).

### *Methods*

Venous blood (heparinized) from 6 smokers and 8 non-smokers was first adjusted to various BE values ( $-10$ ,  $\pm 0$ ,  $+10$  mmol/l) at 37 °C in a tonometer (IL 237, Instrumentation Laboratory) and then equilibrated using a gas mixing apparatus (Ciba Corning 192) with CO<sub>2</sub> partial pressures of 20, 40 and 60 mmHg, and O<sub>2</sub> partial pressures of 27, 40 and 90 mmHg. The O<sub>2</sub> saturation was on the one hand calculated for each blood sample from the values measured in triplicate for pO<sub>2</sub>, pCO<sub>2</sub> and pH using a blood gas analyzer (Ciba Corning 178) and on the other hand measured with an oxymeter (CO-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 O<sub>2</sub> saturation, and the measured O<sub>2</sub> saturation. Calculation led to a slight underestimation of the sO<sub>2</sub> that represented around 1% in the upper and 2% in the lower saturation range.

In the smokers this agreement between calculated and measured O<sub>2</sub> saturation was no longer observed; the true O<sub>2</sub> 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  $1.3 \pm 0.5\%$ , the smokers (blood taken in the morning) showed a mean COHb concentration of  $7.5 \pm 1.4\%$ .

### *Discussion*

As was to be expected, the blood gas analyzer can only calculate the O<sub>2</sub> saturation correctly when besides Hb and O<sub>2</sub>Hb no COHb or MetHb are present. In non-smokers a relatively good agreement is therefore obtained between measured and calculated sO<sub>2</sub>, since in this case the partial and true sO<sub>2</sub> values are practically the same. Compared to the saturation values expected for blood samples with a BE of  $\pm 0$  mmol/l and a pCO<sub>2</sub> of 40 mmHg (corresponding to a so-called standard O<sub>2</sub> binding curve [3]), no significant differences were observed in non-smokers after equilibration with a given pO<sub>2</sub> (data not shown). For instance, the O<sub>2</sub>

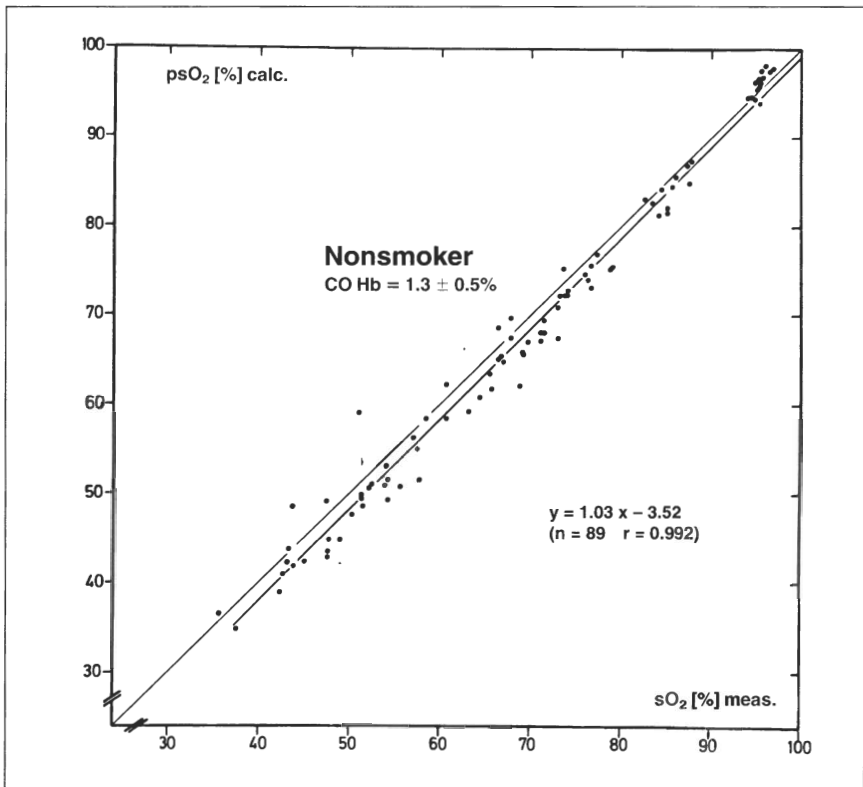


Fig. 1. Comparison between the  $psO_2$  calculated from the  $pO_2$ ,  $pCO_2$  and  $pH$  using a blood gas analyzer (calculated  $psO_2$ , %), and the actual  $sO_2$  measured in a CO-oximeter (measured  $sO_2$ , %) in blood obtained from non-smokers (mean COHb concentration  $1.3 \pm 0.5\%$ ). The line drawn assuming identical values for  $psO_2$  and  $sO_2$  is shown together with the regression line for the calculated  $psO_2$ . Each symbol represents the mean of three individual values.

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 permit the calculation of  $sO_2$  with a blood gas analyzer despite the slight underestimation of  $sO_2$  obtained by calculation (cf. fig. 1).

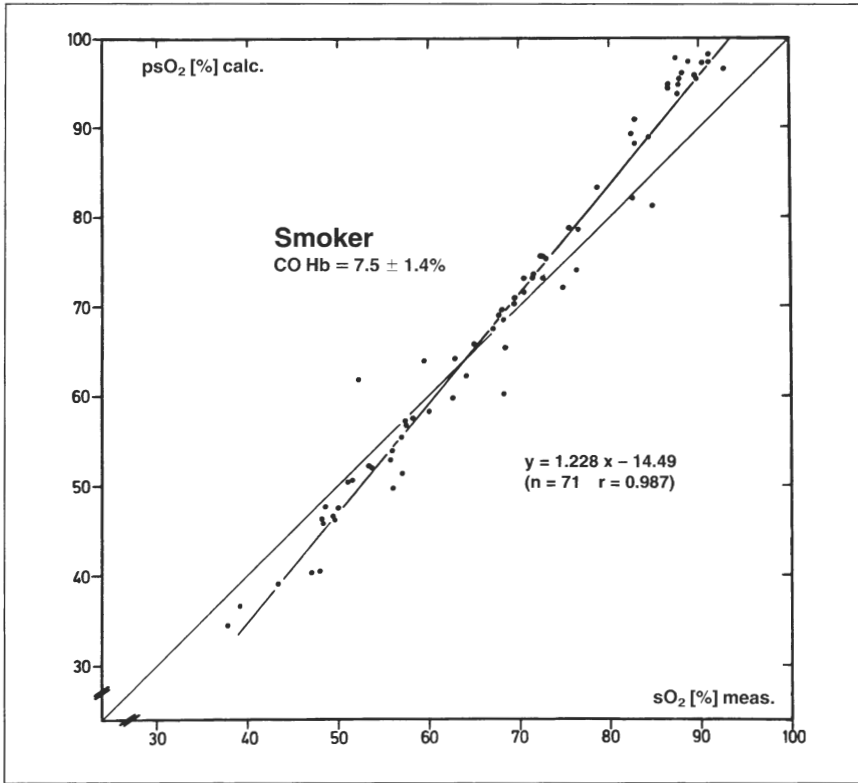


Fig. 2. Comparison between the psO<sub>2</sub> calculated from the pO<sub>2</sub>, pCO<sub>2</sub> and pH using a blood gas analyzer (calculated psO<sub>2</sub>, %), and the actual sO<sub>2</sub> measured in a CO-oxymeter (measured sO<sub>2</sub>, %) in blood obtained from smokers (mean COHb concentration 7.5 ± 1.4%). The line drawn assuming identical values for psO<sub>2</sub> and sO<sub>2</sub> is shown together with the regression line for the calculated psO<sub>2</sub>. Each symbol represents the mean of three individual values.

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 sO<sub>2</sub> (CO-oxymeter) of such blood samples, dropped from 97% to 89%, when these had been equilibrated at pCO<sub>2</sub> = 40 mmHg and BE ± 0 mmol/l with a pO<sub>2</sub> of 90 mmHg. Calculation of the sO<sub>2</sub>, 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 89%. This overestimation of the  $sO_2$  in the upper saturation range obtained by calculation in smokers is due to the fact that only the so-called partial  $sO_2$  ( $psO_2$ ) can be calculated, and not the true  $sO_2$ .

The second problem encountered in calculating the  $psO_2$  from the  $pO_2$ ,  $pCO_2$  and  $pH$  is the fact that only the shift in the  $O_2$  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  $O_2$  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  $pO_2$  of 27 mmHg ( $pCO_2 = 40$  mmHg,  $BE = \pm 0$  mmol/l) should show an  $sO_2$  of 46% in the case of a normal  $O_2$  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 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  $sO_2$  on the assumption of a standard  $O_2$  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  $O_2$  binding curve, only a standard  $O_2$  binding curve can be used as a basis for calculation.

The relatively 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  $pO_2$ ,  $pCO_2$  and  $pH$ , all of which display experimental scatter, these variations can accumulate in the final calculation.

The comparison by Breuer et al. [1] of measured and calculated values of  $O_2$  saturation can only in part be referred to here. Thus, these authors compared the partial, calculated  $sO_2$  (based upon a total of three calculation formulas) with the values measured in the Hem-oxymeter OSM 2 (Radiometer), also representing the partial  $sO_2$ . Since this older equipment only uses two wavelengths it can optimally only measure the partial  $sO_2$  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 Ciba Corning)  $O_2$  saturation values since the actual measured values are not presented

(the report that the calculated sO<sub>2</sub> is 2.7% higher than the measured sO<sub>2</sub> cannot be assigned to any particular range; no information is given on cCOHb).

### *Summary*

The (partial) O<sub>2</sub> saturation calculated by a blood gas analyzer from measured values for pO<sub>2</sub>, pCO<sub>2</sub> and pH is compared with the actual sO<sub>2</sub> values measured in smokers and non-smokers using a CO-oxymeter 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<sub>2</sub> in the lower saturation range, compared to the measured (true) values.

### *References*

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