The oxygen status of arterial human blood

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The oxygen status of arterial human blood is described at least by four variables: Oxygen partial pressure (pO₂, mmHg), oxygen saturation (SO₂, %), hemoglobin content (cHb, g/dL) and oxygen content (cO₂, mL/dL).

Besides perfusion, however, the oxygen supply of all organs is decisively determined by the mean capillary pO₂, which itself is primarily dependent on the arterial cO₂.

Therefore, the oxygen availability (cardiac output x caO₂, mL/min) may be described by the cO₂ value in arterial blood or those variables who determine the latter one. The diagnostic significance of the O₂ variables of the oxygen status consequently increases in the order of pO₂, SO₂, cHb and cO₂.

In arterial blood, oxygen partial pressure is the result of O₂ diffusion within the lungs into the blood (lung function). Oxygen saturation describes the portion of chemically bound oxygen expressed as O₂Hb in relation to total Hb (Hb + O₂Hb + COHb + MetHb). Oxygen content is the total amount of oxygen in blood chemically bound plus physically dissolved.

Under pathophysiological conditions the diagnostic significance becomes very clear. Disturbances of lung function decreases all three variables, pO₂ (hypoxia), SO₂ (hypoxemia) and cO₂ (hypoxemia), to produce hypoxic hypoxemia.

Carbon monoxide poisoning or methemoglobin formation decreases two variables, SO₂ and cO₂, where the pO₂ remains normal and results in toxic hypoxemia.

Anemia with a decrease in the hemoglobin content lowers cO₂ only, while pO₂ and SO₂ remain normal (anemic hypoxemia).

Key words: Hb content; hypoxemia; hypoxemia; Hb content; O₂ partial pressure; O₂ saturation; O₂ status.

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INTRODUCTION

Oxygen must be constantly renewed via a long and complicated route (see Fig. 1). Apart from the role of the external respiration, the oxygen uptake \( (VO_2) \) is determined mainly by the diffusion of \( O_2 \) from the alveolar space to the blood of the pulmonary capillaries. The driving force for this diffusion process is the difference in partial pressure \( (AP_{O_2}) \). Under physiological conditions the \( O_2 \) partial pressure of arterial blood \( (pO_2) \) reaches, within a few \( mmHg \), the alveolar-arterial \( pO_2 \) difference, \( AaDpO_2 \), the alveolar \( pO_2 \) \( (pAO_2) \), in other words, an almost complete equilibration of the \( pO_2 \) of the blood with that of the neighboring alveolar \( pO_2 \) occurs. The arterial \( pO_2 \) is thus an indicator of whether diffusion of \( O_2 \) into the blood has taken place (see Fig. 1). However, it does not indicate whether this diffusion has led to a physiological (i.e. adequate) \( O_2 \) concentration.

![Image](28x604 to 432x1199)

**Fig. 1.** Schematic representation of the transport of \( O_2 \) from the lungs to the tissues with the associated determining parameters: \( O_2 \) uptake in the lung \( (AP_{O_2}) \), \( O_2 \) supply via the bloodstream \( (cO_2) \) and \( O_2 \) diffusion from the blood into the tissues \( (AP_{O_2}) \).

Oxygen is subsequently transported to all organs and tissues by the circulation, a convective transport maintained by the action of the heart. The amount of \( O_2 \) supplied to the organism by the blood (the \( O_2 \) availability, \( AO_2 \)) is determined not only by the blood flow, i.e. by the cardiac output, but also by the \( O_2 \) concentration in the arterial blood \( (cO_2) \). Thus, in contrast to the \( O_2 \) uptake, \( O_2 \) transport is essentially determined by the \( O_2 \) concentration \( (cO_2) \) and not by the \( pO_2 \).

The microcirculation is equipped with an extremely large surface allowing gas diffusion over a short distance, so that \( O_2 \) reaches all tissue cells as efficiently as possible. The driving force for this diffusive transport is also in this case the \( O_2 \) partial pressure difference \( (AP_{O_2}) \), here between the capillary blood \( (pCO_2) \) and the cells of the tissue \( (pCO_2) \).

**ASSESSMENT OF OXYGEN SUPPLY**

A complete assessment of the transport of \( O_2 \) from the alveoli to the individual cells requires knowledge of the following:

- The arterial \( O_2 \) partial pressure \( pO_2 \) for assessing the function of the lungs (respiration) or of artificial ventilation.
- The cardiac output \( (CO_2) \) and/or the organ perfusion \( (Q) \) for describing the \( O_2 \) availability.
- The arterial \( O_2 \) concentration \( cO_2 \) for determining the \( O_2 \) availability, and
- The capillary \( O_2 \) partial pressure \( pCO_2 \) for assessing the supply of \( O_2 \) to the tissue.

However, since neither the cardiac output (or perfusion) nor the capillary blood can generally be approached diagnostically, the assessment of \( O_2 \) availability (\( O_2 \) transport) must be based upon the arterial blood alone. Thus the arterial \( O_2 \) concentration, together with the \( O_2 \) content curve, are predictive of the state of the capillary \( O_2 \) partial pressure. Since a physiological \( cO_2 \) can only occur if the \( pO_2 \) function and the \( cO_2 \) and \( cHb \) \( (O_2 \) binding of the blood) lie in the normal range, the arterial \( O_2 \) concentration \( (cO_2) \) can be considered to be a global value incorporating the \( pO_2 \), \( cO_2 \), and \( cHb \). An arterial \( O_2 \) concentration in the physiological range therefore guarantees an adequate \( cO_2 \) supply from the standpoints of both \( cO_2 \) and \( pO_2 \). However, in
rare cases an inadequate supply of O\textsubscript{2} via the capillaries may occur despite the presence of a physiological arterial O\textsubscript{2} concentration, i.e. when the O\textsubscript{2} content curve is altered pathologically to the left and the capillary pO\textsubscript{2} is significantly lowered.

For some theoretical purposes it is most appropriate to assess the O\textsubscript{2} supply to a tissue or the organism from data on venous (for tissue) or mixed venous (for the organism) blood rather than from arterial blood (O\textsubscript{2} availability). In practice, however, this is difficult, since all the above-mentioned parameters (pO\textsubscript{2}, sO\textsubscript{2}, etc.) can only be evaluated if further data are known: the venous values are dependent upon both the blood flow (Q) and the O\textsubscript{2} consumption (VO\textsubscript{2}).

In the case of mixed venous blood (whole organism), diagnosis is further complicated by the fact that only the average of the O\textsubscript{2} consumption and perfusion for all organs can be estimated. A change in O\textsubscript{2} consumption of a single organ or interruption of its blood supply will lead to practically no change in any O\textsubscript{2} status parameters from the mixed venous point of view.

Furthermore, mixed venous blood in practice is very difficult to obtain (catheter within the pulmonary artery).

Finally, calculation of mixed venous parameters, e.g. uncompensated mixed venous pO\textsubscript{2}, includes all mentioned restrictions, i.e. only a mean value dependent upon both unknown cardiac output and oxygen consumption together with a probably uncertain calculation procedure related to hemoglobin affinity (p50).

**PHYSIOLOGY OF THE ARTERIAL O\textsubscript{2} STATUS**

All the above-mentioned parameters, O\textsubscript{2} concentration, O\textsubscript{2} saturation, O\textsubscript{2} partial pressure as well as Hb concentration, can together be referred to as the O\textsubscript{2} status [6].

The relationship between these parameters is illustrated in Fig. 2. Via the so-called O\textsubscript{2} binding curve the arterial partial pressure (p\textsubscript{O\textsubscript{2}}, mmHg, kPa) determines the arterial O\textsubscript{2} saturation of hemoglobin (sO\textsubscript{2}, %). This gives the percentual or fractional proportion of oxygenated hemoglobin (O\textsubscript{2}Hb) in relation to the total amount of hemoglobin in the blood.

When the O\textsubscript{2} binding ability of Hb is normal, the O\textsubscript{2} saturation can reach approximately 96 % in arterial blood. When the O\textsubscript{2} binding ability is altered, e.g. in the presence of methemoglobin (MetHb) or carbonylhemoglobin (COHb), the maximal level of O\textsubscript{2} saturation must be correspondingly lower. Since virtually all humans have about 0.5 - 2 % of their Hb in the form of MetHb and 1 - 2 % as COHb, approximately 1.5 - 3 % of hemoglobin will be present in the deoxygenated form (Hb); this explains the physiological value of 96 % for the sO\textsubscript{2} in arterial blood (satO\textsubscript{2}).

![Diagram](image-url)

**FIG. 2. Parameters determining the O\textsubscript{2} status of the blood with their interrelationships.**

However, for methodological reasons, in addition to the O\textsubscript{2} saturation of hemoglobin (O\textsubscript{2}Hb as a percentage of total Hb), a partial O\textsubscript{2} saturation (pO\textsubscript{2}Hb) % can be defined when the percentual or fractional proportion of O\textsubscript{2}Hb is considered in relation to the sum of O\textsubscript{2}Hb plus Hb alone. The term "partial" is used here since only a portion of the total hemoglobin (i.e. that available for O\textsubscript{2} transport) is taken into consideration.

The terms "O\textsubscript{2} saturation" (sO\textsubscript{2}) related to total Hb and "partial O\textsubscript{2} saturation" (pO\textsubscript{2}Hb) related to O\textsubscript{2}Hb plus Hb alone are to be preferred to "fractional" (sO\textsubscript{2}) and "functional" (pO\textsubscript{2}Hb) saturation [3].

The relationship between O\textsubscript{2} saturation as a measure of chemically bound O\textsubscript{2} and the O\textsubscript{2} partial pressure is referred to as the O\textsubscript{2} binding curve. It is not only describes the
binding of O₂ to hemoglobin (O₂ uptake in the lungs) but also the release of O₂ from hemoglobin ("O₂ dissociation curve") as can be imagined in the capillaries. Examples of O₂ binding curves, i.e. pSO₂ (%) as a function of pO₂ (mmHg), are shown in Fig. 3. With the exception of the fetus (arterial pO₂ only 25-30 mmHg), the arterial blood (pO₂ approx. 90 mmHg) reaches an O₂ saturation of around 98% in all cases shown. During the subsequent (capillary) O₂ release, the fetal O₂ binding curve, as well as that of a smoker, shows a leftward shift (increased affinity) whereas that of an anemic patient is shifted to the right (decreased affinity) compared to the normal O₂ binding curve. Whereas the leftward shift seen in the fetus (in this case the O₂ uptake in the placenta is of prime importance) leads to a desirable effect, i.e. facilitation of O₂ uptake, this is undesirable in the case of smokers since a deterioration in O₂ release to the tissues results from the loading of HB with CO. The rightward shift of the O₂ binding curve seen in anemic patients fulfills a useful purpose since the release of O₂ to the tissues is facilitated. With the exception of the special case of the fetus, the organism reacts to a deterioration in O₂ supply to the capillaries within 6-12 hours by an increase in the 2,3-DPG concentration in the erythrocytes, resulting in a rightward shift of the O₂ binding curve. This shift is not noticeable in arterial blood, i.e., it only occurs to the extent that no significant decrease in O₂ saturation (hypoxigenation) can arise. Such a rightward shift therefore cannot be diagnosed in arterial blood, i.e., pO₂ and pO₂ remain normal.

On the other hand, if the O₂ binding curve is depicted in terms of SO₂ (%) as a function of pO₂ (mmHg) as shown in Fig. 4, a change is observed in arterial blood. The arterial O₂ saturation now shows a value of about 96% (with the exception of the fetus). However, this is not the case for smokers, where the maximal SO₂ can be only approximately 84% if, as assumed here, the COHb concentration is 15%. Clearly, this manner of depicting the O₂ binding curve,
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![Image]

FIG. 5. O2 content curves of blood, presented as O2 content (cO2, mL/dL) as a function of O2 partial pressure (pO2, mmHg) for normal subjects (N), fatal blood (F), anemic patients (A) and a smoker (S) (female with 15% COHb). The diagrammatically accessible ranges (arterial, mixed venous) are shown as continuous lines, the capillary, inaccessibility, ranges as broken lines. The half saturation pressure is indicated by (a).

I.e. sO2 v.s. pO2 has a greater information content than pO2 vs. cO2.

Thus the two O2 saturation parameters differ significantly in their diagnostic evidence. The O2 saturation that only takes into account the amount of Hb available for O2 transport ("available Hb") pO2, represents a value that compares well with the pO2, i.e. it is suitable only for assessing the function of the lungs. Even more clearly, the pO2, gives the percentage proportion of O2Hb related to the sum of O2Hb plus Hb, without necessitating knowledge of the concentrations of Hb and those Hb derivatives that are not available for O2 transport.

The O2 saturation parameter that takes into account the total Hb (sO2) will always change if the function of the lungs and the O2 binding ability of Hb are affected.

If the O2 saturation (sO2) is to be used as a basis for calculating the concentration of chemically bound O2, the sO2 (as a fraction) must be multiplied by the hemoglobin concentration (E) and the so-called Hufner number (Fig. 2). The latter represents the theoretically maximum amount of O2 (sO2 100%) that can be bound to 1 g Hb. This has the value 1.39 mL/g.

Apart from the large proportion of chemically bound O2 there is a smaller amount of physically dissolved O2 in the blood that can be estimated from the O2 partial pressure and the O2 solubility. The O2 concentration (O2 content) of the blood consists of the sum of chemically bound plus physically dissolved O2 (sO2 + COHb) and is generally expressed in mL/dL (see Fig. 2). The normal value derived from an sO2 of 96%, a EbHb of 15 g/dL and 0.3 mL/dL physically dissolved O2 is 20.3 mL/dL.

The relationship between the O2 concentration of blood (sO2 otherwise known as the O2 content) and the O2 partial pressure (pO2, mmHg) can be referred to as the O2 content curve. Thus the O2 content consists of the sum of the chemically bound and physically dissolved O2, the partial pressures of which are in equilibrium with one another.

The O2 content curve is shown in Fig. 5 for the same samples as in Fig. 3 and 4 (O2 binding curve). It is clear that the O2 content curve allows the differences between fatal blood, normal blood from both men and women, the blood of a smoker (female with 15% COHb) and that of an anemic patient to be distinguished.

The O2 content curve describes a relationship of the arterial O2 status and the prediction of the situation occurring in the microcirculation, i.e. the condition of the supply of O2 to the tissues via the capillaries.

It is apparent that possible changes in all parameters that affect the arterial O2 content, namely:
- pO2
- sO2
- and
- the Hb
are evident in the arterial O2 status, especially in connection with the later release of O2 to the tissue.

The O2 content curve demonstrates especially well the two physiological adaptation mechanisms for improving the O2 supply to
FIG. 6. O₂ content curves of blood, cO₂, for normal
blood (N) and anemia (A) during hypoxemia. Al-though the proportion of chemically bound O₂ remains constant (cO₂ = 98 %), the amount of physically dissolved O₂ increases steadily with pO₂.

the tissue:
- an increase in the Hb concentration,
  here shown in the case of the fetus, and
- a rightward shift in the O₂ binding curve, here shown in the case of an anemic patient.
At the same time the undesirable negative effects seen in a smoker are also described;
- a leftward shift in the O₂ binding curve and
- a decrease in the effective Hb concentration.
A special case, that can be used at the same time to illustrate the O₂ content curve, is shown in Fig. 6. This concerns the O₂ content curve in the arterial blood following administration of pure oxygen. At pO₂ values above around 150 mmHg, in this case with an O₂ saturation (sO₂) of approximately 98 % (Methb and COHb in the physiological range), the O₂ concentration increases linearly with increasing pO₂. This linear increase in O₂ concentration represents an increase only in physically dissolved O₂; the amount of chemically bound O₂ remains constant. In the case of the anemic patient it is clear that with a decrease in the amount of chemically bound O₂ (decreased cHb) the proportion of physically dissolved O₂ compared to total O₂ content (cHb/dl) steadily increases.

The diagnostic value of the parameters described above, i.e., O₂ partial pressure (pO₂), O₂ saturation (sO₂) and O₂ concentration (cO₂), therefore differs. The pO₂ will always be altered in cases of im-paired lung function or when the inspired pO₂ is modified. A decrease in saO₂ will additionally occur when the O₂ binding ability of Hb or the O₂ affinity of Hb (O₂ binding curve) are impaired. Finally, a change in caO₂ records all the changes de-scribed plus those in the Hb concentration. The diagnostic value thus increases consi-derably in the order: pO₂, saO₂, caO₂.

PATHOPHYSIOLOGY OF THE ARTERIAL O₂ STATUS

To prevent complication of the terminology of the various pathological situations, the following definition should be used:
- a decrease in pO₂ is defined as hypoxia,
- a decline in sO₂ is defined as hypoxegenation, and
- a reduction in cO₂ is referred to as hypoxemia.
Since the critical, global parameter of arterial blood, the O₂ concentration, is deter-mined by all the above-mentioned parameters, this should be used as a compre-hensive term.
Hypoxemia can be defined according to its origin (see Fig. 2): hypoxic hypoxemia is characterized by a decrease in pO₂, saO₂ and caO₂; in toxic hypoxemia the pO₂ is normal but the saO₂ and caO₂ are reduced; and in anemic hypoxemia both pO₂ and saO₂ are normal but the caO₂ is lowered.
All disturbances of lung function, of the external respiration or artificial ventilation can lead to a decrease in arterial pO₂ (hypoxia) and thus to hypoxemia. The severity of this hypoxic hypoxemia depends upon the degree to which the pO₂ falls. The theoretically possible case of a hypox-
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**Figure 7:** O₂ content curves of blood, cO₂, as a function of pO₂, in three different forms of hypoxemia at approximately the same O₂ concentration. The differences in maximum response (hypoxic hypoxia, pO₂ = 26 mmHg) and position of the O₂ content curve (toxic hypoxia, 50% COHb) compared with the normal (acute) or slightly altered (chronic) O₂ curve in anemia (anemic hypoxia, cHb = 8 g/dL) clearly illustrate why hypoxemias of different origins are tolerated to such different extents. The arterial and mixed venous ranges are shown as solid lines, the capillary ranges as broken lines.

**Figure 8:** O₂ content curves of blood, cO₂ (mL/dL) as a function of pO₂ (mmHg), for normal subjects (NS) in comparison to that from a blood bag (BB) after four days' storage from a non-smoker (NS) or a smoker (SS) with 10% COHb. A hypoxia with a pO₂ of 26 mmHg is compared with a CO intoxication with 50% COHb and an anemia with an Hb concentration of 8 g/dL. These O₂ content curves compellingly illustrate clinical experience and make it clear why the same degree of hypoxemia of different origins must have different consequences. Although an anemic hypoxemia of this severity can be survived without difficulty, the same degree of hypoxic hypoxia can only be tolerated under extreme conditions; a toxic hypoxia (CO intoxication) can hardly be considered conducive to life.

The O₂ supply to the tissue depends not only on the capillary O₂ concentration but is also determined by the associated O₂ partial pressure, which is the driving force for the diffusion of O₂ from capillary blood into the tissue (cf. Fig. 1).

If a displacement of the oxygen content curve at all has a clinical relevance, then only in those cases where:
- a marked leftward shift (CO intoxication) or
- a combination of more than one
leftward shifts of different causes (CC treatment together with loss of 2,3-
Diphosphoglycerate) are to be expected.

The latter one may be observed in the case of stored blood taken from a smoker. Such a typical example [4] is shown in Fig. 8.

During storage of blood within a blood bag (after dilution by 12.5 % with the saline preservation solution) the erythrocytes lose their 2,3-DPG which results in a marked leftward shift of the O₂ content curve.

If the blood donor would be a smoker, here assumed with 10 % COHb, an additional leftward shift of the O₂ content curve must be seen. Such a combination may lead to an inadequate O₂ supply, especially to the myocardium with its high arterio-venous O₂ difference.

NEw METHODS FOR THE DETERMINATION OF VARIABLES OF THE ARTERIAL O₂ STATUS

Only a few remarks will be made here according to new methods in the field of the described parameters. All methods of different principles for the variables of the O₂ status are listed in Table I. New methods available today are the hemoximeters, the pulse oximeters and the Oxystat procedure.

Hemoximeters, e.g. 2,500 Ciba-Corning or OSM3 Radiometer, are multi-wavelength blood oximeters for the in vitro measurement of total Hb content (cHb), all Hb derivatives (COHb, MetHb and O₂ saturation (sO₂), together with some derived parameters (chemically bound O₂ content, partial O₂ saturation (pO₂)).

For some reasons, e.g. sample volume, accuracy, blood with FbF, we propose the OSM3 oximeter [2].

Pulse oximeters try to measure arterial partial O₂ saturation (pO₂) in vivo and continuously.

Using only two wavelengths they are unable to differentiate between all possible Hb derivatives like O₂Hb, Hb, COHb and MetHb. Therefore, in relation to the used algorithms they can measure only the partial O₂ saturation in the range of 98 % (normoxia) down to about 75 % (hypoxia). The results of a test of five pulse oximeters of different producers under normoxic and hypoxic conditions for 20 subjects (smokers and non-smokers) demonstrate the fact that (with one exception) pulse oximeters are able to measure pO₂ with an accuracy of ±2 % [1].

However, the pulse oximeter of Radiometer obviously tries to measure O₂ saturation (sO₂) using other algorithms with the consequence of a slight underestimation in the case of non-smokers as well as a small overestimation in the case of smokers [1].

The Oxystat method [6] is a photometric in vitro procedure using disposable cuvettes for the determination of oxygen as well as of hemoglobin content together with a battery-operated mini-photometer. The oxygen cuvette is based on a photometric O₂ determination, in which the oxygen reacts quantitatively with a specific and highly sensitive reagent (alkaline solution of catechol with iron ions). The hemoglobin cuvette uses the new developed reaction solution (alkaline hematin D-375). A special dosing attachment for both cuvettes was developed that can be affixed to a rectangular disposable cuvette (see Fig. 9A). About 20 µl of blood (ear lobe, fingertip) fill the lumen of the dosing system by capillary action. By displacing the piston, exactly 10 µl are measured and inserted, with the exclusion of air, into the inner space of the cuvette and thus into contact with the liquid reaction. Photometry is thus simplified as follows: application of a drop of blood, absorption measurement, displacement of the piston, shaking, second absorption measurement.

Using a photometer with a built-in calculator (see Fig. 9B) the following values for the O₂ status are obtained quickly from the measured absorbance differences and recorded:
<table>
<thead>
<tr>
<th>Variable</th>
<th>Method</th>
<th>Principle</th>
</tr>
</thead>
<tbody>
<tr>
<td>$P_{O_2}$ (mmHg)</td>
<td>Blood Gas Anal. Transcutaneous</td>
<td>$O_2$ electrode, in vivo $O_2$ electrode, in vivo</td>
</tr>
<tr>
<td>$S_{O_2}$ ($S_{O_2}$ (fractions))</td>
<td>Hemoxyteter</td>
<td>Photometry, in vivo Calculation ($O_2H$, $O_3O_2$, $O_4$ Cap)</td>
</tr>
<tr>
<td>$pO_2$ ($S_{O_2}$ (mmHg))</td>
<td>Blood Gas Anal. Pulse Oximeter</td>
<td>Calculation ($pO_2$, $O_2BC$) Photometry, in vivo</td>
</tr>
<tr>
<td>$cHb$ (g/dL)</td>
<td>Various Methods</td>
<td>Photometry Conductometry</td>
</tr>
<tr>
<td>$cO_2$ ($mL/dL$)</td>
<td>Van Slyke</td>
<td>Manometry, in vivo Galvanometry, in vivo</td>
</tr>
<tr>
<td>$O_2$ (chem.bld.) (mL/DL)</td>
<td>Hemoxyteter</td>
<td>Calculation ($cHb$, $S_{O_2}$)</td>
</tr>
</tbody>
</table>

**FIG. 9 A.** Schematic presentation of a dosing system affected to a disposable cuvette according to Wolf & Zander. A volume of exactly 10 μL of blood takes place into the cuvette with the exclusion of air, when capillary force causes blood (about 20 μL) to enter the lumen, the centre of which, a moveable piston, can be displaced. Tipping the cuvette then washes the content of the lumen into the liquid reagent.

**FIG. 9 B.** The Oximat photometer. A battery-operated mini-photometer for measuring the absorbance difference of a $Hb$ and an $O_2$ cuvette, before and after injection of 10 μL of blood in each case. A built-in calculator gives the $Hb$ concentration (g/dL), the $O_2$ concentration (mL/dL) and calculates the $O_2$ saturation (%) from these two values.
O₂ content (cO₂, ml/dl), Hb concentration (cHb, g/dl), and O₂ saturation (cO₂ %, calculated). Since a crystalline standard exists for both procedures, the O₂ cuvette and the Hb cuvette, which allows gravimetric calibration, an accuracy of 2 % is obtained in both procedures.

Using the Osystat method, consisting of ready-made disposable cuvettes (for O₂ and Hb) with a dosing attachment and a battery-operated mini-photometer with built-in calculator, all the necessary data for the O₂ status (cO₂, cHb, sO₂) can be obtained quickly and simply anywhere with an accuracy and reproducibility of ±2 %.

The user can perform an optimal quality control of the entire procedure - namely, cuvettes, dosing system, photometer and calculator - using concentrated solutions (Hb, O₂) of defined composition, which are treated the same way as blood. This way the quality is optimally guaranteed.

Since only 30 - 40 µL (1 - 2 drops) of blood are required for a complete analysis, the puncturing of arteries is unnecessary. With a few exception (extreme drop in blood pressure), the arterial blood can be obtained from the "arterIALIZED" ear lobes.

REFERENCES