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The oxygen status of arterial human blood

R. ZANDER

Institute of Physiology and Pathophysiology, Mainz University (FRG)

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The oxygen status of arterial human blood is described at least by four variables: Oxygen partial pressure (pO_2, mmHg), oxygen saturation (sO_2, %), hemoglobin content (cHb, g/dL) and oxygen content (cO_2, mL/dL).

Beside perfusion, however, the oxygen supply of all organs is decisively determined by the mean capillary pO_2 which itself is primarily dependent on the arterial cO_2.

Therefore, the oxygen availability (cardiac output x caO_2, mL/min) may be described by the cO_2 value in arterial blood or those variables who determine the latter one. The diagnostic significance of the O_2 variables of the oxygen status consequently increases in the order of pO_2, sO_2 (cHb) and cO_2.

In arterial blood, oxygen partial pressure is the result of O_2 diffusion within the lungs into the blood (lung function). Oxygen saturation describes the portion of chemically bound oxygen expressed as O_2Hb in relation to total Hb (Hb + O_2Hb + 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, pO_2 (hypoxia), sO_2 (hypoxygenation) and cO_2 (hypoxemia), to produce hypoxic hypoxemia.

Carbon monoxide poisoning or methemoglobin formation decreases two variables, sO_2 and cO_2, where the pO_2 remains normal and results in toxic hypoxemia.

Anemia with a decrease in the hemoglobin content lowers cO_2 only, while pO_2 and sO_2 remain normal (anemic hypoxemia).

Key words: Hb content; hypoxemia; hypoxygenation; hypoxia; O_2 content; O_2 partial pressure; O_2 saturation; O_2 status.

Reprints: R. Zander, Institut für Physiologie und Pathophysiologie, Universität Mainz, Saarstrasse 21, D-6500 Mainz, FRG.

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, ($\dot{V}O_2$) 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 (ΔpO_2). Under physiological conditions the O_2 partial pressure of arterial blood (paO_2) reaches, within a few mmHg (alveolar-arterial pO_2 difference, $AaDO_2$), 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.

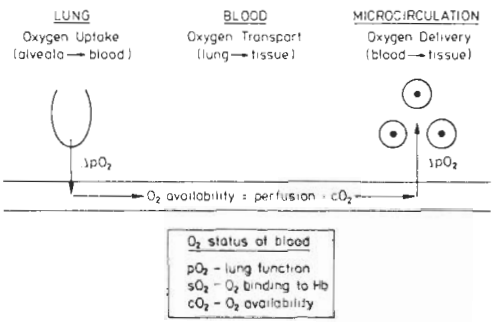

FIG. 1. Schematic representation of the transport of O_2 from the lungs to the tissues with the associated determining parameters: O_2 uptake in the lung (ΔpO_2), O_2 supply via the bloodstream (cO_2) and O_2 diffusion from the blood into the tissues (ΔpO_2).

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, $\dot{A}O_2$) 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 (caO_2). Thus, in contrast to the O_2 uptake, O_2 transport is essentially determined by the O_2 concentration (caO_2) and not by the paO_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 (ΔpO_2), here between the capillary blood (pcO_2) and the cells of the tissue (ptO_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 paO_2 for assessing the function of the lungs (respiration) or of artificial ventilation,
- the cardiac output (C.O.) and/or the organ perfusion ($\dot{Q}$) for describing the O_2 availability,
- the arterial O_2 concentration caO_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 caO_2 can only occur if the paO_2 (lung function) and the saO_2 and cHb (O_2 binding of the blood) lie in the normal range, the arterial O_2 concentration (caO_2) can be considered to be a global value incorporating the paO_2, saO_2 and cHb.

An arterial O_2 concentration in the physiological range therefore guarantees *per se* an adequate capillary O_2 supply from the standpoints of both cO_2 and pO_2. However, in

rare cases an inadequate supply of O_2 via the capillaries may occur despite the presence of a physiological arterial O_2 concentration, i.e. when the O_2 content curve is altered pathologically to the left and the capillary pO_2 is significantly lowered.

For some theoretical purposes it is most appropriate to assess the O_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_2 availability). In practice, however, this is difficult, since all the above-mentioned parameters (pO_2, sO_2, cO_2) can only be evaluated if further data are known: the venous values are dependent upon both the blood flow ($\dot{Q}$) and the O_2 consumption ($\dot{Q}O_2$).

In the case of mixed venous blood (whole organism), diagnosis is further complicated by the fact that only the average of the O_2 consumption and perfusion for all organs can be estimated. A change in O_2 consumption of a single organ or interruption of its blood supply will lead to practically no change in any O_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_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_2 STATUS

All the above-mentioned parameters, O_2 concentration, O_2 saturation, O_2 partial pressure as well as Hb concentration, can together be referred to as the O_2 status [6].

The relationship between these parameters is illustrated in Fig. 2. Via the so-called O_2 binding curve the arterial partial pressure (paO_2, mmHg, kPa) determines the arterial O_2 saturation of hemoglobin (saO_2, %). This gives the percentual or fractional proportion of oxygenated hemoglobin (O_2Hb) in relation to the total amount of hemoglobin in the blood. When the O_2 binding ability of Hb is normal, the O_2 saturation can reach approximately 96 % in arterial blood. When the O_2 binding ability is altered, e.g. in the presence of methemoglobin (MetHb) or carboxyhemoglobin (COHb), the maximal level of O_2 saturation must be correspondingly lower. Since virtually all humans have about 0.5 - 1 % 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_2 in arterial blood (saO_2).

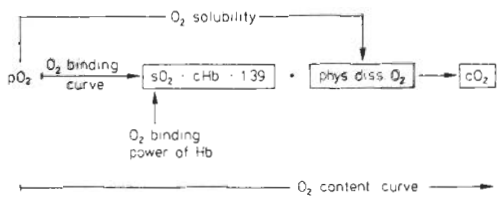

FIG. 2. Parameters determining the O_2 status of the blood with their interrelationships.

However, for methodological reasons, in addition to the O_2 saturation of hemoglobin (O_2Hb as a percentage of total Hb), a partial O_2 saturation (psO_2, %) can be defined when the percentual or fractional proportion of O_2Hb is considered in relation to the sum of O_2Hb plus Hb alone. The term "partial" is used here since only a portion of the total hemoglobin (i.e. that available for O_2 transport) is taken into consideration.

The terms "O_2 saturation" (sO_2) related to total Hb and "partial O_2 saturation" (psO_2) related to O_2Hb plus Hb alone are to be preferred to "fractional" (sO_2) and "functional" (psO_2) saturation [3].

The relationship between O_2 saturation as a measure of chemically bound O_2 and the O_2 partial pressure is referred to as the O_2 binding curve. It not only describes the

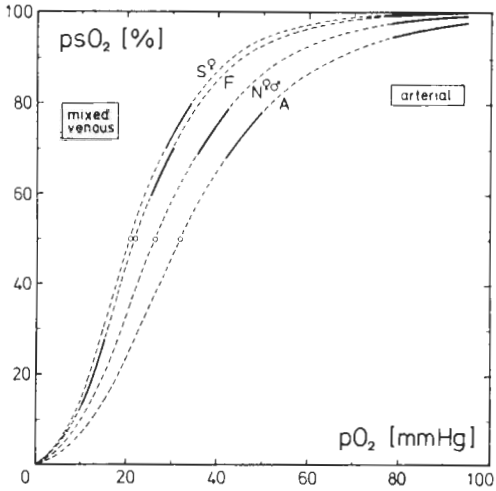

FIG. 3. O_2 binding curves of blood, presented as partial O_2 saturation (psO_2, %) as a function of the O_2 partial pressure (pO_2, mmHg) for normal subjects (N), fetal blood (F), anemic patients (A) and a smoker (S) (female with 15 % COHb). The diagnostically accessible ranges (arterial, mixed venous) are shown as continuous lines, the capillary, inaccessible ranges as broken lines. The half saturation pressure is indicated by (o).

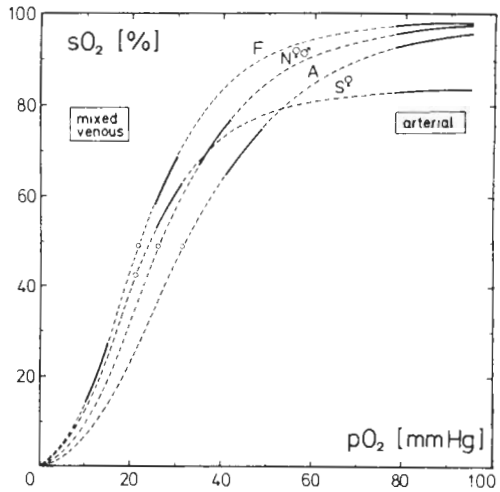

FIG. 4. O_2 binding curves of blood, presented as O_2 saturation (sO_2, %) as a function of O_2 partial pressure (pO_2, mmHg) for normal subjects (N), fetal blood (F), anemic patients (A) and a smoker (S) (female with 15 % COHb). The diagnostically accessible ranges (arterial, mixed venous) are shown as continuous lines, the capillary, inaccessible ranges as broken lines. The half saturation pressure is indicated by (o).

binding of O_2 to hemoglobin (O_2 uptake in the lungs) but also the release of O_2 from hemoglobin ("O_2 dissociation curve") as can be imagined in the capillaries. Examples of O_2 binding curves, i.e. psO_2 (%) as a function of pO_2(mmHg) are shown in Fig. 3. With the exception of the fetus (arterial pO_2 only 25-30 mmHg), the arterial blood (pO_2 approx. 90 mmHg) reaches an O_2 saturation of around 98 % in all cases shown. During the subsequent (capillary) O_2 release, the fetal O_2 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 O_2 binding curve. Whereas the leftward shift seen in the fetus (in this case the O_2 uptake in the placenta is of prime importance) leads to a desirable effect, i.e. facilitation of O_2 uptake, this is undesirable in the case of smokers since a deterioration in O_2 release to the tissues results from the loading of Hb with CO. The rightward shift of the O_2 binding curve seen in anemic patients fulfills a useful purpose since the release of O_2 to the tissues is facilitated. With the exception of the special case of the fetus, the organism reacts to a deterioration in O_2 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 O_2 binding curve. This shift is not noticeable in arterial blood, i.e. it only occurs to the extent that no significant decrease in O_2 saturation (hypoxygenation) can arise. Such a rightward shift therefore cannot be diagnosed in arterial blood, i.e. paO_2 and saO_2 remain normal.

On the other hand, if the O_2 binding curve is depicted in terms of sO_2 (%) as a function of pO_2 (mmHg) as shown in Fig. 4, a change is observed in arterial blood. The arterial O_2 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 sO_2 can be only approximately 84 % if, as assumed here, the COHb concentration is 15 %. Clearly, this manner of depicting the O_2 binding curve,

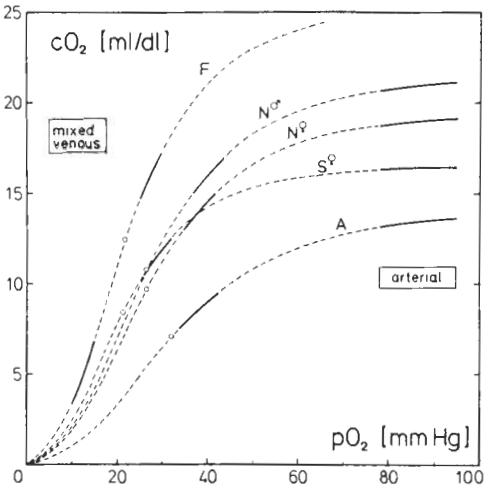

FIG. 5. O_2 content curves of blood, presented as O_2 content (cO_2, mL/dL) as a function of O_2 partial pressure (pO_2, mmHg) for normal subjects (N), fetal blood (F), anemic patients (A) and a smoker (S) (female with 15 % COHb). The diagnostically accessible ranges (arterial, mixed venous) are shown as continuous lines, the capillary, inaccessible ranges as broken lines. The half saturation pressure is indicated by (o).

i.e. sO_2 v.s. pO_2, has a greater information content than psO_2 vs. pO_2.

Thus the two O_2 saturation parameters differ significantly in their diagnostic evidence. The O_2 saturation that only takes into account the amount of Hb available for O_2 transport ("available Hb"), psO_2, represents a value that compares well with the paO_2, i.e. it is suitable only for assessing the function of the lungs. Even more clearly, the psO_2 gives the percentual proportion of O_2Hb related to the sum of O_2Hb plus Hb, without necessitating knowledge of the concentrations of Hb and those Hb derivatives that are not available for O_2 transport.

The O_2 saturation parameter that takes into account the total Hb (sO_2) will always change if the function of the lungs and the O_2 binding ability of Hb are affected.

If the O_2 saturation (sO_2) is to be used as a basis for calculating the concentration of chemically bound O_2, the sO_2 (as a fraction) must be multiplied by the hemoglobin concentration (cHb) and the so-called Hüfner number (Fig. 2). The latter represents the theoretically maximum amount of O_2 (sO_2 100 %) that can be bound to 1 g Hb. This has the value 1.39 mL/g.

Apart from the large proportion of chemically bound O_2 there is a smaller amount of physically dissolved O_2 in the blood that can be estimated from the O_2 partial pressure and the O_2 solubility. The O_2 concentration (O_2 content) of the blood consists of the sum of chemically bound plus physically dissolved O_2 (cO_2) and is generally expressed in mL/dL (see Fig. 2). The normal value derived from an sO_2 of 96 %, a cHb of 15 g/dL and 0.3 mL/dL physically dissolved O_2 is 20.3 mL/dL.

The relationship between the O_2 concentration of blood (cO_2, otherwise known as the O_2 content) and the O_2 partial pressure (pO_2, mmHg) can be referred to as the O_2 content curve. Thus the O_2 content consists of the sum of the chemically bound and physically dissolved O_2, the partial pressures of which are in equilibrium with one another.

The O_2 content curve is shown in Fig. 5 for the same examples as in Fig. 3 and 4 (O_2 binding curve). It is clear that the O_2 content curve allows the differences between fetal 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.

Only the O_2 content curve allows a description of the arterial O_2 status and the prediction of the situation occuring in the microcirculation, i.e., the condition of the supply of O_2 to the tissues via the capillaries.

It is apparent that possible changes in all parameters that affect the arterial O_2 content, namely

- pO_2,
- sO_2, and
- cHb

are evident in the arterial O_2 status, especially in connection with the later release of O_2 to the tissue.

The O_2 content curve demonstrates especially well the two physiological adaptation mechanisms for improving the O_2 supply to

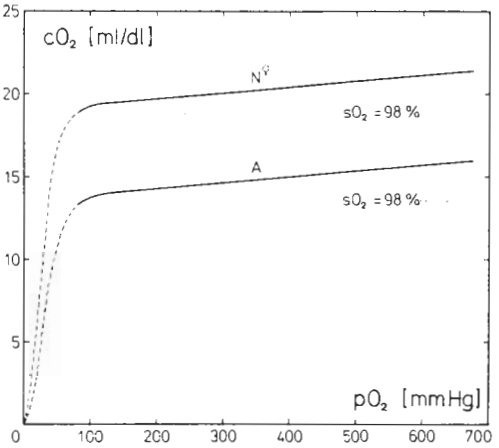

FIG. 6. O_2 content curves of blood, cO_2, for normal blood (N) and anemia (A) during hyperoxia. Although the proportion of chemically bound O_2 remains constant (sO_2 = 98 %), the amount of physically dissolved O_2 increases steadily with pO_2.

the tissue:

- an increase in the Hb concentration, here shown in the case of the fetus, and
- a rightward shift in the O_2 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 O_2 binding curve and
- a decrease in the effective Hb concentration.

A special case, that can be used at the same time to illustrate the O_2 content curve, is shown in Fig. 6. This concerns the O_2 content curve in the arterial blood following administration of pure oxygen. At paO_2 values above around 150 mmHg, in this case with an O_2 saturation (sO_2) of approximately 98 % (MetHb and COHb in the physiological range), the O_2 concentration increases linearly with increasing pO_2. This linear increase in O_2 concentration represents an increase only in physically dissolved O_2; the amount of chemically bound O_2 remains constant. In the case of the anemic patient it is clear that with a decrease in the amount of chemically bound O_2 (decreased cHb) the proportion of physically dissolved O_2 compared to total O_2 content (mL/dL) steadily increases.

The diagnostic value of the parameters described above, i.e. O_2 partial pressure (paO_2), O_2 saturation (saO_2) and O_2 concentration (caO_2), therefore differs. The paO_2 will always be altered in cases of impaired lung function or when the inspired pO_2 is modified. A decrease in saO_2 will additionally occur when the O_2 binding ability of Hb or the O_2 affinity of Hb (O_2 binding curve) are impaired. Finally, a change in caO_2 records all the changes described plus those in the Hb concentration. The diagnostic value thus increases considerably in the order: paO_2, saO_2, caO_2.

PATHOPHYSIOLOGY OF THE ARTERIAL O_2 STATUS

To prevent complication of the terminology of the various pathological situations, the following definition should be used:

- a decrease in pO_2 is defined as hypoxia,
- a decline in sO_2 is defined as hypoxygenation, and
- a reduction in cO_2 is referred to as hypoxemia.

Since the critical, global parameter of arterial blood, the O_2 concentration, is determined by all the above-mentioned parameters, this should be used as a comprehensive term.

Hypoxemia can be defined according to its origin (see Fig. 2): hypoxic hypoxemia is characterized by a decrease in paO_2, saO_2 and caO_2; in toxic hypoxemia the paO_2 is normal but the saO_2 and caO_2 are reduced; and in anemic hypoxemia both paO_2 and saO_2 are normal but the caO_2 is lowered.

All disturbances of lung function, of the external respiration or artificial ventilation can lead to a decrease in arterial pO_2 (hypoxia) and thus to hypoxemia. The severity of this hypoxic hypoxemia depends upon the degree to which the paO_2 falls.

The theoretically possible case of a hypoxy-

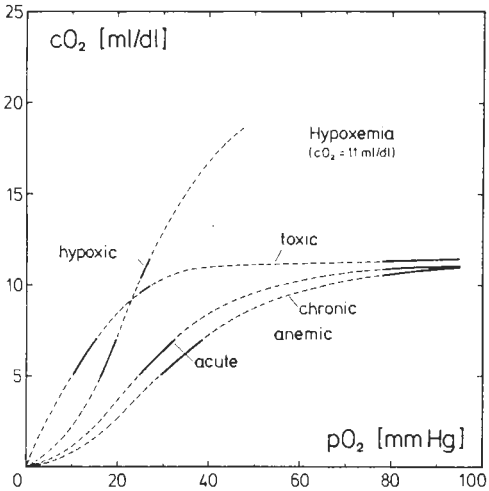

FIG. 7. O_2 content curves of blood, cO_2 as a function of pO_2, in three different forms of hypoxemia at approximately the same O_2 concentration. The differences in maximum response (hypoxic hypoxemia, paO_2 = 26 mmHg) and position of the O_2 content curve (toxic hypoxemia, 50 % COHb) compared with the normal (acute) or slightly altered (chronic) O_2 curve in anemia (anemic hypoxemia, 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.

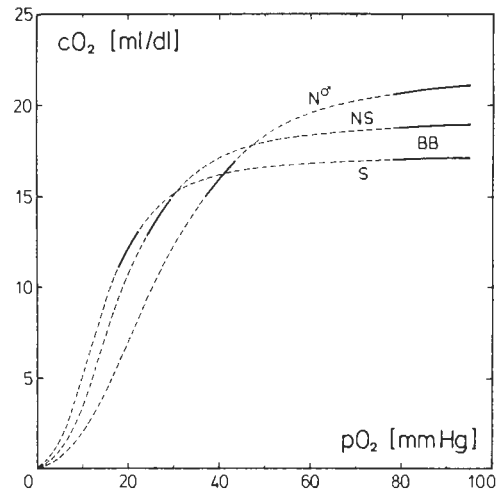

FIG. 8. O_2 content curves of blood, cO_2 (mL/dL) as a function of pO_2 (mmHg), for normal subjects (N) in comparison to that from a blood bag (BB) after four days storage from a non-smoker (NS) or a smoker (S) with 10 % COHb.

genation with normal paO_2 resulting from a rightward shift in the O_2 binding curve can practically be ruled out. It is much more common that such a hypoxygenation (paO_2 normal) is of toxic origin.

In carbon monoxide intoxication (smoke poisoning) or chronic exposure to CO as occurs in tobacco smokers, different proportions of Hb are reversible occupied by CO. An increase in MetHb concentration will always arise when oxidizing substances are able to convert hemoglobin (Fe^{++}) to hemiglobin (Fe^{+++}) i.e. MetHb.

Finally, a change in hemoglobin concentration must also lead to hypoxemia, in this case referred to as anemic.

The O_2 content curves of these three possible forms of arterial hypoxemia are illustrated for clarity in Fig. 7. As an example, forms of hypoxemia have been chosen which lead, with different origins, to the same decrease in arterial O_2 concentration to a value around 11 mL/dL. A hypoxia with a paO_2 of 26 mmHg is compared with a CO intoxication with 50 % COHb and an anemia with an Hb concentration of 8 g/dL. These O_2 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 hypoxemia can only be tolerated under extreme conditions; a toxic hypoxemia (CO intoxication) can hardly be considered conductive to life.

The O_2 supply to the tissue depends not only on the capillary O_2 concentration but is also determined by the associated O_2 partial pressure, which is the driving force for the diffusion of O_2 from capillary blood into the tissue (cf. Fig. 1).

If a displacement of the oxygen content curve at all has a clinical relevance, than only in those cases where

- a marked leftward shift (CO intoxication) or
- a combination of more than one

leftward shifts of different causes (CO treatment together with loss of 2,3-DPG) is given.

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 acidic preservation solution) the erythrocytes lose their 2,3-DPG which results in a marked leftward shift of the O_2 content curve.

If the blood donor would be a smoker, here assumed with 10 % COHb, an additional leftward shift of the O_2 content curve must be seen. Such a combination may lead to an inadequate O_2 supply, especially to the myocardium with its high arterio-venous O_2 difference.

NEW METHODS FOR THE DETERMINATION OF VARIABLES OF THE ARTERIAL O_2 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 O_2 status are listed in Table I. New methods available today are the hemoxymeters, the pulse oxymeters and the Oxystat procedure.

Hemoxymeters, e.g. 2.500 Ciba-Corning or OSM3 Radiometer, are multi-wavelength blood oxymeters for the *in vitro* measurement of total Hb content (cHb), all Hb derivatives (COHb, MetHb) and O_2 saturation (sO_2), together with some derived parameters (chemically bound O_2 content, partial O_2 saturation (psO_2)).

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

Pulse oxymeters try to measure arterial partial O_2 saturation (psO_2) in vivo and continuously.

Using only two wavelengths they are unable to differentiate between all possible Hb derivatives like O_2Hb, Hb, COHb and MetHb. Therefore, in relation to the used algorithms they can measure only the partial O_2 saturation in the range of 98 % (normoxia) down to about 75 % (hypoxia). The results of a test of five pulse oxymeters of different producers under normoxic and hypoxic conditions for 20 subjects (smokers and non-smokers) demonstrate the fact that (with one exception) pulse oxymeters are able to measure psO_2 with an accuracy of ± 2 % [1].

However, the pulse oxymeter of Radiometer obviously tries to measure O_2 saturation (sO_2) 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 O_2 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-575). 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 a 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 simplied as follows: application of a drop of blood, absorbance measurement, displacement of the piston, shaking, second absorbance measurement.

Using a photometer with a built-in calculator (see Fig. 9B) the following values for the O_2 status are obtained quickly from the measured absorbance differences and recorded:

TABLE I. Methods for the determination of variables of the O_2 status.

Variable		Method	Principle
pO_2	(mmHg)	Blood Gas Anal. Transcutaneous	O_2 electrode, *in vitro* O_2 electrode, *in vivo*
sO_2 (sO_2 (frac))	(%)	Hemoxymeter Oxystat	Photometry, *in vitro* Calculation (cHb, cO_2, O_2 Cap.)
psO_2 (sO_2 (func))	(%)	Blood Gas Anal. Pulse Oxymeter	Calculation (pO_2, O_2BC) Photometry, *in vivo*
cHb	(g/dL)	Various Methods	Photometry Conductometry
cO_2	(mL/dL)	Van Slyke Lex-O_2-Con Oxystat	Manometry, *in vitro* Galvanometry, *in vitro* Photometry, *in vitro*
cO_2 (chem.bd.)	(mL/dL)	Hemoxymeter	Calculation (cHb, sO_2)

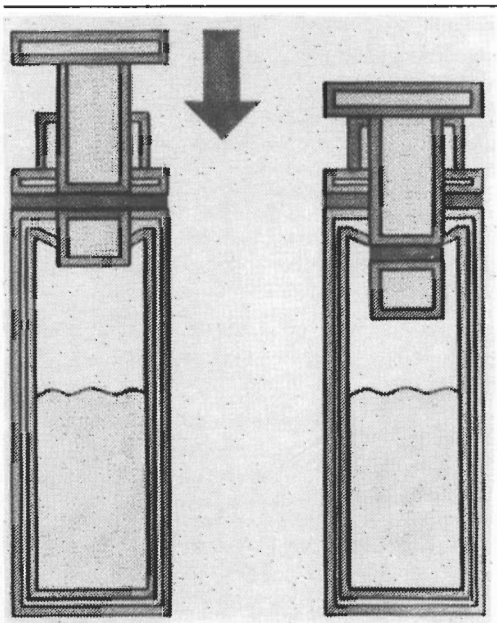

FIG. 9 A. Schematic presentation of a dosing system affixed to a disposable cuvette according to Wolf & Zander. A volume of exactly 10 μL of blood takes place into the cuvette with the exclusion of air, when capillary force causes blood (about 20 μL) to enter the lumen, the centre of which, a moveable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

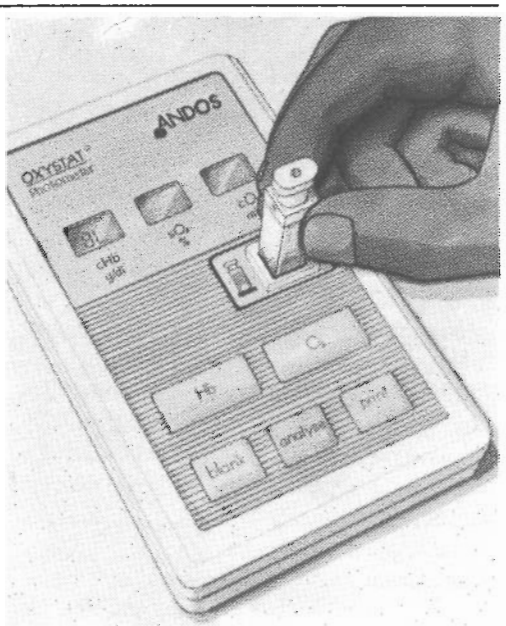

FIG. 9 B. The Oxystat photometer. A battery-operated mini-photometer for measuring the absorbance difference of a Hb and an O_2 cuvette, before and after injection of 10 μL of blood in each case. A built-in calculator gives the Hb concentration (g/dL), the O_2 concentration (mL/dL) and calculates the O_2 saturation (%) from these two values.

O_2 content (cO_2, mL/dL)
Hb concentration (cHb, g/dL), and
O_2 saturation (sO_2, %, calculated).

Since a crystalline standard exists for both procedures, the O_2 cuvette and the Hb cuvette, which allows gravimetric calibration, an accuracy of 2 % is obtained in both procedures.

Using the Oxystat method, consisting of ready-made disposable cuvettes (for O_2 and Hb) with a dosing attachment and a battery-operated mini-photometer with built-in calculator, all the necessary data for the O_2 status (cO_2, cHb, sO_2) can be obtained quickly and simply anywhere with an accuracy and reproducibility of ±3 %.

The user can perform an optimal quality control of the entire procedure - namely, cuvette, dosing system, photometer and calculator - using concentrated solutions (Hb, O_2) 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.

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