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Oxygen status algorithm, version 3, with some applications

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The Oxygen Status Algorithm is a computer program which uses measurements from a pH & blood gas analyzer and a haemoximeter to calculate the oxygen status and the acid-base status of the arterial blood. Version 3 features on-line data collection from the analyzer; storage of up to 2000 patient cases in a Lotus 123 file format; printing of a Cumulated Patient Report in addition to the Patient Status Report; combination of arterial and mixed venous data for calculation of the shunt and the oxygen consumption rate (when cardiac output is keyed in); calculation of reference values for fetal haemoglobin for newborns (when gestational age is keyed in).

Examples of applications answer the following questions:

1) Does hyperventilation improve the oxygen supply to the tissues? No, for a normal person a slight hypoventilation with a pCO_2 of 8.5 kPa provides a maximal oxygen extraction tension.

2) What is the optimal hyperventilation at the top of Mt. Everest (ambient pressure 33 kPa)? Hyperventilation to a pCO_2 of about 1.4 kPa provides a maximal oxygen extraction tension of 2.4 kPa for an unacclimatised person.

3) Which change in haemoglobin oxygen affinity would be equivalent to a decrease in arterial pO_2 to 6.3 kPa? The oxygen extraction tension would decrease to 4.0 kPa and the same value would be caused by a decrease in half-saturation tension to 2.8 kPa, a decrease which could be due to a moderate alkalaemia (pH = 7.54) combined with a moderately decreased 2,3-diphosphoglycerate concentration (3.4 mmol/L).

4) Is temperature correction of the measured pO_2 and pCO_2 to the actual body temperature needed? Yes, for example, omitting temperature correction even when the patient temperature is only slightly decreased to 36 °C would result in a negative value for the calculated arterio-venous shunt fraction when the actual value, using temperature correction, is 11%.

5) Does the alpha-stat approach of pCO_2 and pH regulation in hypothermia, where pH is allowed to rise as in blood *in vitro,* cause a fall in mixed venous pO_2 below the critical value? No, although the mixed venous pO_2 will be lower than with the pH-stat approach (constant pH at body temperature), it remains above the critical mixed venous pO_2 level.

The program is intended for clinical routine use as well as teaching purposes. It has context sensitive help as well as an extensive help index. A number of "demo" cases are provided with annotations in a separate file.

Key words: acid-base status of the blood; blood gases; carbon dioxide; clinical physiology; computer programs; intensive care; metabolism; physiology;

The Oxygen Status Algorithm is a computer program which uses measurements from a pH & blood gas analyzer and a haemoximeter (1). The former measures pH, pCO_2, and pO_2, the latter measures oxygen saturation, fractions of carboxy- and methaemoglobin, and concentration of total haemoglobin. The program calculates the oxygen status and the acid-base status of the blood and display the results in the *oxygen graph* ($\log pO_2$, ctO_2), the *acid-base chart* (pH, $\log pCO_2$), and the *gas map* ($\log pO_2$, $\log pCO_2$). The results, including the first two graphs, may be printed in a *patient status report.*

The Oxygen Status Algorithm, version 1, was ready in 1988. Version 2 has been distributed since 1992 with a "blue manual" in a large number by courtesy of Radiometer, Copenhagen. Version 3 has been distributed in a limited number by the authors.

The present text describes version 3 with emphasis on the modifications in relation to previous versions and some examples of calculations with results of general physiological interest are provided. The text is not intended to replace the program manual, which is distributed in a text file together with the program itself.

VERSION 3 FEATURES

On-line data collection:

We have only had opportunity to make on-line connection to analyzers of type ABL5xx combined with OSM3, *e.g.* ABL520 with built-in haemoximeter (Radiometer, Copenhagen). The ABL must be provided with a RSI100 communication interface card (Radiometer, Copenhagen). Instructions for on-line connection are given in the help text of the Oxygen Status Algorithm. The program has been running on-line for more than two years in our intensive care unit and the experiences are described elsewhere (2).

Data storage in spreadsheet file:

When the program is first started, a default data file named OsaDat.WK1 with 20 default cases is automatically created. We have adopted the WK1 format used by the popular Lotus 123 spreadsheet program, thus enabling the user to read from and write to the OSA data file with any compatible program. While the values may be changed, it is essential that the cell formatting is not modified within the range that holds the data used and generated by the OSA program. Outside this range,

the user may perform any desired calculations, and these parts of the spreadsheet will not be modified by the OSA program. Different file names and sizes are created and accessed using switches on the command line; >osa /f=MYDAT /n=2000 for example opens or creates a datafile named MYDAT.WK1 containing 2000 default cases. The file is 'circular' in the sense that new cases are written into the next record until the total number of records is reached, at which time the file should be saved before overwriting previous cases. A 1.4 Mbyte diskette will hold more than 1000 cases.

Cumulated patient report:

The data file provides data for a cumulated patient report containing all the previous measurements on a given patient. The lay-out of the cumulated report has previously been described (3).

Selection of relevant quantities:

It is possible to calculate more than forty quantities on the basis of simultaneous measurements of arterial and mixed venous blood. It is therefore necessary to decide which quantities are relevant and which are dispensable. We have selected sixteen quantities as indispensable in the cumulated report (3). Several quantities are intermediary results, useful for the understanding of the relationships among the variables, but not needed in the printed reports (*e.g.* the $p\mathrm{O_2}$hI and $p\mathrm{O_2}$A). Other quantities contain redundant information, for example p_x, c_x and Q_x, which are all measures of the oxygen extractivity of the arterial blood. We have selected p_x as the most instructive, and the other two have been removed from the display, but they are still calculated and stored in the Lotus 123 file. We have included calculation of the concentration of total carbon dioxide in whole blood for the purpose of estimating the $\mathrm{CO_2/O_2}$ exchange ratio, R_Q, when simultaneous arterial and mixed venous measurements have been made.

Combining arterial and mixed venous data:

When simultaneous measurements are performed on arterial and venous blood it is possible to combine the measurements and plot the venous point in the arterial graphs or vice versa. The total functional veno-arterial shunting is then calculated using the actual arterio-venous total oxygen concentration difference rather than a default value of 2.3 mmol/L.

Extrapolation to 50 % oxygen saturation and calculation of p_{50} and *c*DPG require an oxygen saturation of less than 97 %, and, if the haemoximeter is not calibrated to the utmost accuracy, this limit need to be reduced to 95 % or less. For this reason it is preferable to use venous blood, which usually has an oxygen saturation of less than 90 %, for calculation of *c*DPG. *c*DPG of the arterial blood is assumed to equal the value for venous blood, which is used automatically for calculating the arterial p_{50} and the arterial $s\mathrm{O_2}$.

We also assume that *c*tHb, *F*COHb and *F*MetHb are identical in arterial and venous blood. Peripheral venous blood must be obtained without venous stasis to avoid haemoconcentration. Thus, when simultaneous measurements are performed on arterial and venous blood we substitute the measured *c*tHb, *F*COHb, and *F*MetHb values with the mean of the arterial and venous values.

It has been suggested that pH and $p\mathrm{CO_2}$ of the mixed venous blood, or rather the a-v differences, should contain valuable information (4). Thus, if the veno-arterial $p\mathrm{CO_2}$ difference is increased this should indicate accumulation of non-volatile acid in the tissues. We find it more relevant to calculate the $\mathrm{CO_2/O_2}$ exchange ratio, R_Q, in order to reveal tissue accumulation or liberation of $\mathrm{CO_2}$. Further studies are needed to substantiate this thesis, and for this purpose we have included the calculation of R_Q in the program. It should be added that the calculation of the veno-arterial total $\mathrm{CO_2}$ concentration difference is rather imprecise, being a small difference between two large values.

When the cardiac output has been measured, for example by thermodilution using the pulmonary artery catheter also used for mixed venous blood sampling, calculation of the oxygen consumption rate is possible. Interpretation of the oxygen consumption rate is difficult in critically ill patients whether expressed as mmol/min or mL/min, and whether related to body weight (per kg) or body surface area (per $\mathrm{m^2}$) (3). Reference values vary with patient age, sex, body size, and temperature. For this reason we also express the result in relation to the normal mean value taking all these variables into account.

Reference values for fetal haemoglobin:

Reference values for fetal haemoglobin adjusted for gestational age have been included. The calculation is based on the data published by Wimberley (5).

Printer support:

We have added printer support for the HP DeskJet 500 and HP Laserjet 4.

EXAMPLES OF CALCULATIONS

The following examples reveal some interesting and surprising findings. Other examples have been published elsewhere (6). All the calculations require iterative solution of several physiological equations, and the results are difficult to achieve without a computer program.

Influence of ventilation on oxygen supply:

Does hyperventilation improve the oxygen supply to the tissues? Most people think so: hyperventilation increases the alveolar and the arterial $p\mathrm{O_2}$. But at the same time $p\mathrm{CO_2}$ decreases, pH increases, and p_{50} decreases: oxygen is bound

tighter to haemoglobin. We have calculated the oxygen extraction tension as a function of pCO_2 for a normal person, and the conclusion is, that it is better to hypoventilate (Fig. 1).

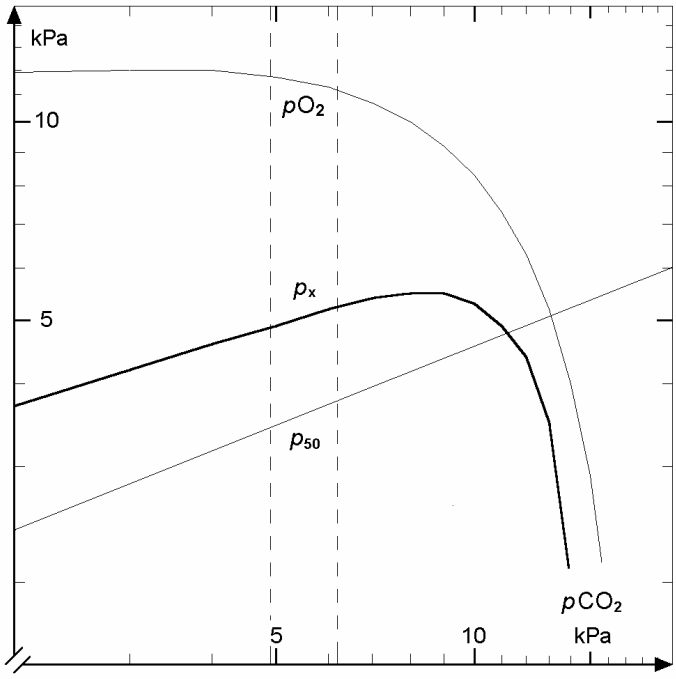

Fig. 1. Arterial oxygen tension (pO_2), half-saturation tension (p_{50}), and oxygen extraction tension (p_x) as functions of the arterial carbon dioxide tension (pCO_2) during acute hyper- and hypoventilation in a normal 25 year old male (double logarithmic plot). The dashed vertical lines indicate the reference interval for pCO_2. The maximal oxygen extraction tension is achieved at a pCO_2 of 8.5 kPa.

The pCO_2 which provides the highest p_x is 8.5 kPa. The advantage of a slight hypercapnia disappears if the hypercapnic acidosis is compensated with return of pH towards normal. Similarly, a patient with chronic respiratory insufficiency and a pCO_2 of 8 kPa does not benefit from hypoventilation but might benefit from hyperventilation.

High altitude:

What is the optimal ventilation at high altitude? The low arterial pO_2 stimulates the chemoreceptors to hyperventilation which tends to increase the pO_2. At the same time, however, the haemoglobin oxygen affinity increases due to pH increase. We have calculated the optimal pCO_2 for a normal person at the top of Mt. Everest where the barometric pressure is about 33 kPa (Fig. 2) (7). We find that a maximal oxygen extraction tension of 2.4 kPa is obtained at a pCO_2 of 1.4 kPa. If the person acclimatises, primarily by compensating partly for the respiratory alkalaemia and increasing the haemoglobin concentration, then a maximal oxygen extraction tension of 3.0 kPa is obtained at a pCO_2 of 1.0 kPa. Direct measurements of expired air from mountaineers near the summit have indicated an arterial pCO_2 of about 1.5 kPa, i.e. very close to the value, which provides the maximal oxygen extraction tension.

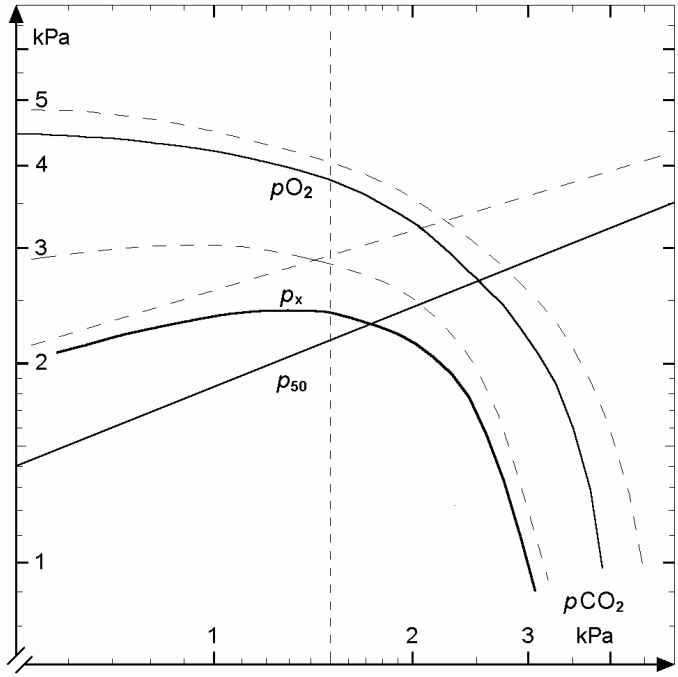

Fig. 2. Arterial oxygen tension (pO_2), half-saturation tension (p_{50}), and oxygen extraction tension (p_x) as functions of the arterial carbon dioxide tension (pCO_2) at the top of Mt. Everest, where the pO_2 of the humidified inspired air is as low as 5.6 kPa. The solid curves indicate the functions for a normal unacclimatized person with *F*va=6 %, R_Q=0.86, *c*DPG=5 mmol/L, *c*tHb=9.3 mmol/L, and *c*BEcf=0 mmol/L. A maximum for p_x of 2.4 kPa is observed at pCO_2 about 1.4 kPa. The dashed curves indicate the effect of acclimatization and refer to a person with *F*va=4 %, R_Q=1.0, *c*DPG=6 mmol/L, *c*tHb=10.5 mmol/L, and *c*BEcf=-10 mmol/L. A new maximum for p_x of 3.0 kPa is observed at pCO_2 about 1.0 kPa. The vertical dotted line at 1.5 kPa indicates the arterial pCO_2 of mountaineers near the summit.

Comparison of hypoxaemic -, anaemic -, and high-affinity tissue hypoxia:

The arterial oxygen extractivity is the property of the blood which allows extraction of a given amount of oxygen with a limited fall in pO_2. A normal oxygen extractivity allows an oxygen extraction of 2.3 mmol/L while the pO_2 falls to approximately 5 kPa. If the oxygen extractivity is reduced extraction of 2.3 mmol/L results in a pO_2 lower than 5 kPa, or less oxygen may be extracted before the pO_2 reaches 5 kPa (8,9). We have selected the oxygen extraction tension (p_x, see List of quantities) as the most appropriate quantitative measure of the oxygen extractivity. The oxygen extractivity is determined by a triad of quantities: the arterial pO_2, the haemoglobin oxygen binding capacity, and the haemoglobin oxygen binding affinity (measured by the half-saturation tension). The half-saturation tension, p_{50}, is a neglected parameter, mostly because an effective, non-toxic drug to increase the p_{50} is still awaiting. Nevertheless, the p_{50} is as influential in determining the oxygen extractivity as the arterial pO_2 and the haemoglobin concentration. We have calculated the changes needed in arterial pO_2, haemoglobin concentration, or p_{50}, in order to produce a reduction in p_x from 5 to 4 kPa in a normal adult (Fig. 3). The arterial pO_2 should fall from 11.4 to 6.3 kPa, for example due to an increase in veno-arterial shunting from 6 to 38 %. The haemoglobin concentration should fall from 9.0 to 5.9 mmol/L. The half-saturation tension should fall from 3.6 to 2.8 kPa, which could be due to a metabolic alkalosis (*c*BEcf=12 mmol/l, pH=7.54) with decreased 2,3-di-phosphoglycerate concentration (3.4 mmol/L). The patient with the low half-saturation tension would not receive due attention unless the p_{50} is reported routinely and recognized as equally important as the arterial oxygen tension and the haemoglobin concentration.

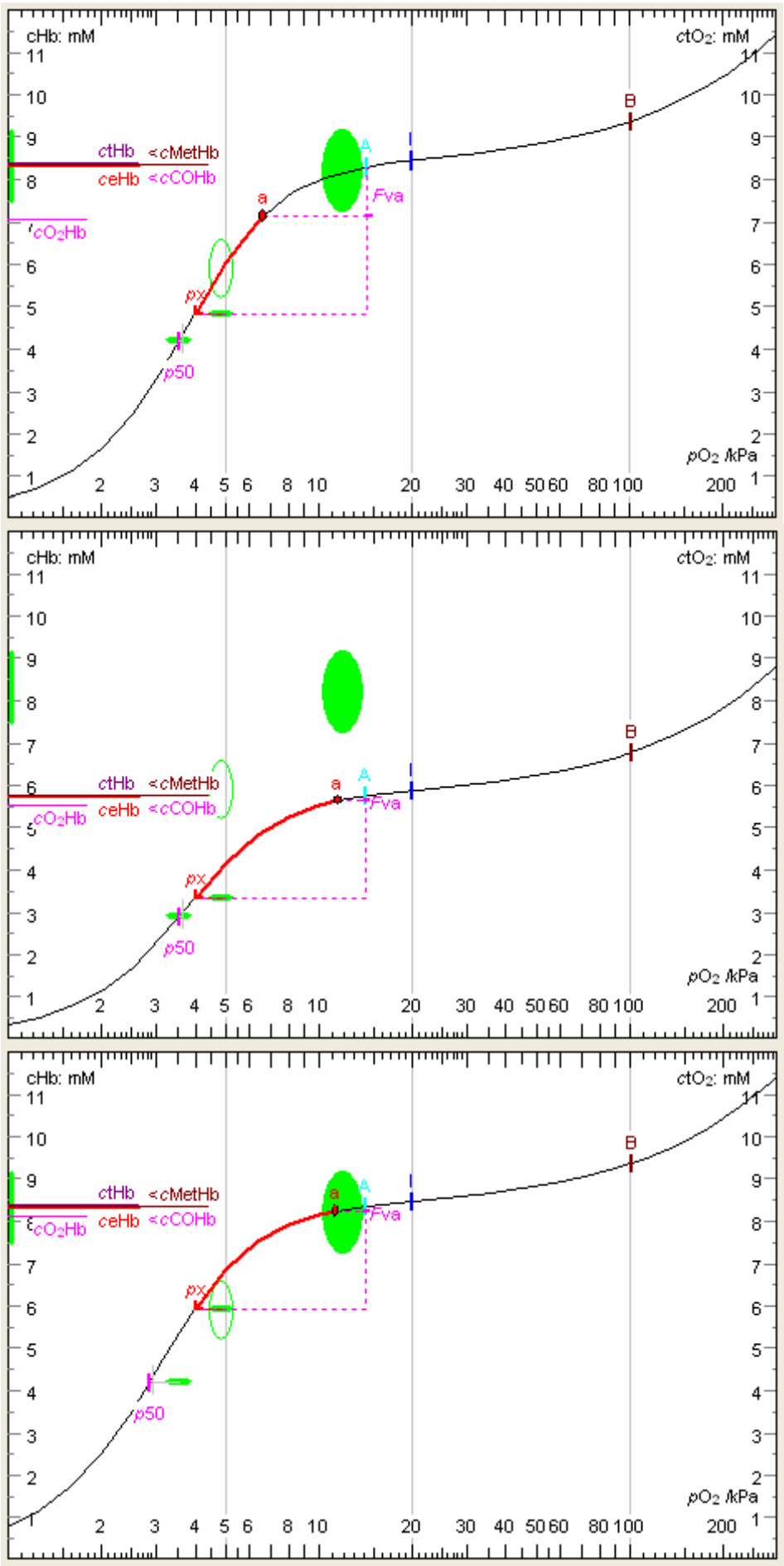

Fig. 3. Oxygen graph illustrating the same reduction in oxygen extractivity (to p_x = 4 kPa) due to 1) hypoxaemia (decreased arterial pO_2, due to increased shunting, 0.34), 2) anaemia (decreased haemoglobin oxygen binding capacity, due to decreased total haemoglobin, 5.78 mmol/L), and 3) high oxygen affinity (decreased half-saturation tension, due to decreased 2,3-

diphosphoglycerate, 2.0 mmol/L).

Temperature effects:

Patient temperature is often neglected in relation to pH-blood gas analysis. The following example illustrates the necessity of taking the patient temperature into account when calculating the veno-arterial shunt (Fig. 4). The values measured at 37 °C were: pH = 7. 240, pCO_2 =10.2 kPa, pO_2= 8.8 kPa, sO_2= 0.865, ctHb = 9.2 mmol/L, FCOHb = 0.005, FMetHb=0.004. FO_2dI was 0.21, R_Q assumed to be 0.86, FHbF assumed to be 0.005. The fraction of mixed venous blood in the arterial blood, Fva, was calculated to be negative. When the correct patient temperature of 36 °C is keyed in, the shunt is calculated to be 11 %, normal for a 50 year old person.

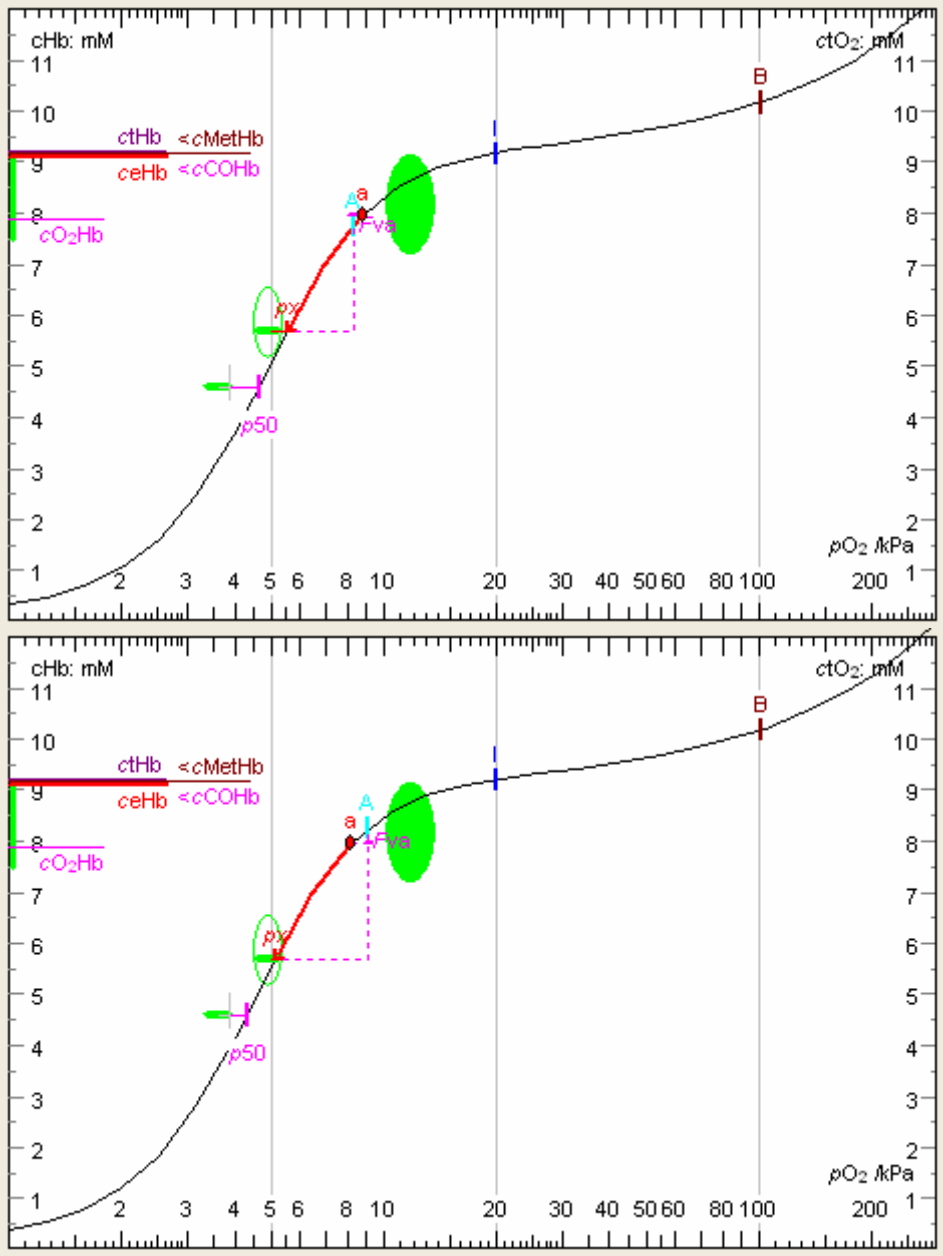

Fig. 4. Oxygen graph illustrating the influence of patient temperature on the shunt calculation. The upper curve refers to 37 °C, the lower curve to the actual patient temperature of 36 °C. Notice that, at 37 °C, the alveolar pO_2 (A) is lower that the arterial (a), resulting in a negative value for the calculated veno-arterial shunt fraction. Referring the values to 36 °C, results in a higher alveolar than arterial pO_2 and the estimated shunt turns out to be 11 %.

The practise of ignoring patient temperature in connection with pH-blood gas analysis is a consequence of the alpha-stat theory of pH and pCO_2 regulation during hypothermia. According to the *α-stat approach* (10), blood pH and pCO_2 change with temperature to the same extent *in vivo* and *in vitro*, as it does in poikilothermic animals. Hence, referencevalues remain constant when pH and pCO_2 values are reported at a fixed standard temperature of 37 °C, regardless of the patient temperature. According to the pH-*stat approach*, pH and pCO_2 should be regulated to be constant *in vivo* as in hibernating animals, by CO_2 accumulation or liberation as temperature decreases or increases. Hence, reference values for pH and pCO_2 remain constant when values are reported at the actual patient temperature.

We have calculated the mixed venous pO_2 as a function of temperature using the alpha-stat as well as the pH-stat approach. We have assumed that the arterio-venous oxygen concentration difference remains constant (2.3 mmol/L), which

means that the cardiac output falls in direct proportion to the fall in oxygen consumption rate. The results are shown in Fig. 5. The critical mixed venous pO_2 is about 3.5 kPa at 37 °C. Below this value the diffusion gradient becomes too small to allow the necessary oxygen diffusion and the oxygen consumption rate falls (9). We assume that the critical mixed venous pO_2 falls 9 %/°C as temperature falls, due to the decrease in oxygen consumption rate, but rises 1 %/°C due to an increase in the oxygen permeability coefficient, hence the change in mixed venous pO_2 is calculated using: $\mathrm{d}\ln pO_2 v_{crit}/\mathrm{d}T = 0.08$.

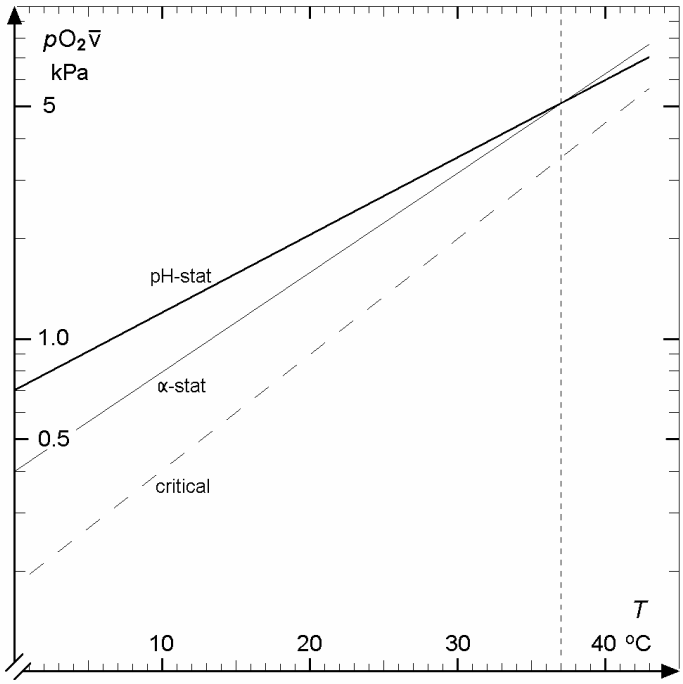

Fig. 5. Mixed venous oxygen tension as a function of temperature for a normal person breathing air. Comparison of the alpha-stat and the pH-stat approach. The dashed line shows the critical mixed venous pO_2. Although the mixed venous pO_2 remains above the critical mixed venous level with both the alpha-stat and the pH-stat approach, the safety margin is greater with the pH-stat approach, especially at very low body temperatures. The graph confirms similar calculations by Willford, Hill, and Moores (11).

The mixed venous pO_2 remains above the critical level with both the alpha-stat and the pH-stat approach, but the safety margin is greater with the pH-stat approach, and that is the reason why the hibernating warm blooded animals prefer the pH-stat regulation. For example, at a body temperature of 10 °C the estimated critical mixed venous pO_2 is 0.4 kPa, the mixed venous pO_2 with the alpha-stat approach 0.8 kPa, and with the pH-stat approach 1.2 kPa. If the cardiac output is halved in relations to the already reduced value, then the mixed venous pO_2 would fall below the critical level with the alpha-stat approach, but not with the pH-stat approach, where a reduction of the cardiac output to one third would be tolerated.

The relationship between temperature, metabolic rate, ventilation, and pCO_2 is derived in the following. The rate of CO_2 elimination in the lungs ($V'CO_2$) is proportional to the alveolar (≈ arterial) pCO_2 and the alveolar ventilation ($V'A$):

$$V'CO_2 = V'A \cdot pCO_2a/Pamb,$$

where Pamb is the ambient pressure. Differentiating with respect to temperature (T) after logarithmic transformation gives:

$$\mathrm{d}\ln V'CO_2/\mathrm{d}T = \mathrm{d}\ln pCO_2a/\mathrm{d}T + \mathrm{d}\ln V'A/\mathrm{d}T.$$

Many studies of the effect of body temperature on the metabolic rate indicate that $\mathrm{d}\ln V'CO_2/\mathrm{d}T \approx 0.09$. If the alveolar ventilation changes in direct proportion to the change in metabolic rate, i.e. $\mathrm{d}\ln V'A/\mathrm{d}T = 0.09$, then pCO_2a remains constant independent of changes in temperature. So, an hibernating animal reduces ventilation in direct proportion to the reduction in metabolic rate to achieve the pH stat approach.

To achieve the α-stat approach, where $\mathrm{d}\ln pCO_2a/\mathrm{d}T = 0.048$ (the temperature coefficient for whole blood in vitro), the change in ventilation with temperature must be: $\mathrm{d}\ln V'A/\mathrm{d}T = 0.042$. This means that during hypothermia the alveolar ventilation falls less than the fall in general metabolic rate. Poikilothermic animals therefore need to maintain a *relative* hyperventilation during hypothermia. The energy requirement of ventilation normally accounts for less than 4 % of total energy requirements, so this relative hyperventilation only requires a few per cent extra energy.

These calculations refer to chronic steady state with equal CO_2 production and elimination. In case of a very rapid fall in body temperature both hibernating and poikilothermic animals by necessity follow the α-stat approach.

Precision of calculations:

The precision of the calculated quantities is difficult to derive analytically from the precision of the measured quantities. It is easy, however, to perform a Monte Carlo simulation, which involves generating a data file with a large number of identical cases and then replace each measured quantity with random numbers with a normal distribution with the desired mean value and a standard deviation equal to the analytical standard deviation. Such random distributions are easily

generated with a spread sheet program and inserted in the Lotus 123 data file. All cases in the file are then marked to be recalculated, and when the Oxygen Status Algorithm is started with this file it automatically calculates all the derived quantities. Reopening the data file in the spread sheet program allows a rapid calculation of mean values and standard deviations for all quantities. Some of the results are shown in Table 1.

The between run variation is twice as large as the within run variation for the spectrometric measurements, indicating that variation of calibration of the haemoximeter is a major source of variation. Once the haemoximeter is calibrated it should not be recalibrated, unless quality control reveals a need for recalibration. We have previously published data for the within run precision and found a slightly lower standard deviation for sO_2 (0.0012) but slightly higher for pO_2 (1.2 kPa) than reported by the manufacturer (Table 1) (12). The sO_2 level and standard deviation are critical for the precision of the calculated p_{50}, cDPG, and p_x values. If thestandard deviation of the sO_2 measurement is 0.002 and the sO_2 level 0.97, then the standard deviation of the estimated cDPG is 0.5 mmol/L. At lower sO_2 levels the standard deviations of these calculated quantities rapidly diminish.

Table 1. Analytical standard deviations of measured and calculated quantities for three levels of oxygen saturations and oxygen tensions. Precision of the measured quantities is specified by the manufacturer, Radiometer Medical, for the ABL520 analyzer: s_0 is repeatability or within-run variation; s_B is between-run variation, calculated as $\sqrt{(s_0^2 + s_D^2)}$, where s_D is calibration variation. The precision of the measured quantities is independent of the oxygen saturation level. The standard deviation of FO_2dI was assumed to be 0.05 at the elevated levels but zero for atmospheric air. TPt and R_Q were taken to be 37 °C and 0.86, respectively, without variation. The precision of the calculated quantities was obtained by Monte Carlo simulation with 500 data sets.

FO_2dI		0.70	0.51	0.21	0.70	0.51	0.21
sO_2		0.97	0.95	0.90	0.97	0.95	0.90
pO_2, kPa		12.3	10.1	7.8	12.3	10.1	7.8
p_x, kPa		5.0	4.8	4.4	5.0	4.8	4.4
Level		s_0			s_B		
Measured:							
pO_2	(see above)	0.07			0.08		
pCO_2	5.3 kPa	0.05			0.07		
pH	7.40	0.002			0.003		
ctHb	9.0 mmol/L	0.12			0.17		
sO_2	(see above)	0.002			0.004		
FCOHb	0.006	0.002			0.004		
FMetHb	0.006	0.002			0.004		
Calculated:							
Fva	0.25	0.014	0.014	0.004	0.016	0.017	0.008
p50	3.5 kPa	0.11	0.08	0.05	0.24	0.14	0.08
cDPG	5.0 mmol/L	0.5	0.3	0.2	1.0	0.6	0.3
px	(see above)	0.14	0.09	0.06	0.29	0.15	0.08
ceHb	8.9 mmol/L	0.12	0.12	0.12	0.17	0.17	0.17
cBEcf	0.0 mmol/L	0.25	0.25	0.25	0.35	0.35	0.35

The precision of the measured quantities, especially the sO_2, needs to be the highest possible in order to obtain an acceptable precision for the calculated parameters. The large imprecision of the calculated p_{50}, cDPG, and p_x at high sO_2 values must be taken into account in the clinical interpretation of these quantities.

Correlation between the analytical error of the measured variables presents a problem. For example, errors in pCO_2 and pH are likely to be negatively correlated, because a negative error in pCO_2 due to loss of CO_2 is associated with a positive error in pH. Similarly errors in pO_2 and sO_2 tend to be positively correlated. Ignoring such correlations, the Monte Carlo simulation overestimates the standard deviation of base excess, p_{50}, cDPG, and p_x The number of cases need to be as high as 500 to provide a reproducible estimate of the standard deviations with a coefficient of variation of less than 5 %.

NEXT VERSION

The described program is written in Pascal (Turbo Pascal version 6.0, Borland International) with extensive use of 'tool boxes' (Object Professional, Turbo Power Software, and Asynch Plus, Blaise Computing) to ensure state-of-the-art programming techniques for all standard tasks. The resulting program is very robust, and after several years of use in an intensive care unit there remains no known bugs.

Evolution of computer hardware require corresponding changes in the oxygen status algorithm software to make use of improvements, such as higher screen or printer resolutions. This hardware specific programming is integrated in newer operating systems which also add a graphical user interface. We have chosen Microsoft Windows as environment for the

next version of the Oxygen Status Algorithm, which thereby gains support for most of the present and future hardware platforms. We can therefore put emphasis on the development of improved algorithms, together with better interpretive and modelling features. A prototype version programmed in Visual Basic (Microsoft) is well under way, but to obtain a reasonably fast program also on smaller computers, the final program will be written in a compiled language.

The next version will allow communication with analytical instruments and central hospital databases through 'dynamic data exchange'. Simultaneous display of several graphs and data sets will allow easy comparison and analysis of patient data over time, while the ability to test therapeutic measures using the mouse directly on the graphs will greatly improve the modelling capabilities. The continuous re-calculations result in smoothly moving graphs, which visualize the complex physiological dependencies. The latter may prove especially valuable when using the program as a teaching aid, where also a 'textbook' style help file and correct display of italic, super and subscripts in quantities is desired.

Computer graphics will be an essential element in every aspect interpretation and teaching in the future. For this reason it is a challenge to develop a useful application which can be an aid in the routine clinical diagnosis, as well as a tutorial aid in the teaching of the rather complex relationships between pH and blood gas variables.

CONCLUSIONS

Version 3 of the Oxygen Status Algorithm is a useful tool for calculation of the relationships among the many variables related to the oxygen status and the acid-base status of the blood. It is useful in the routine laboratory for on-line data collection and generation of detailed laboratory reports, which assists the physician in the intensive care unit. It is useful as a teaching aid, in the pregraduate physiology program as well as the post graduate follow up courses in anaesthesia and intensive care. The use of computer programs as teaching aids in the daily routine as well as in formal courses will undoubtedly be more and more common in the future. This medium is especially suitable for constant revision and improvement, and it is certain that version 3 of the Oxygen Status Algorithm, which we distribute freely at the mailing cost, is not the last version.

LIST OF QUANTITIES

*c*BEcf	concentration of titratable base in extracellular fluid.
*c*DPG	concentration of 2,3-diphosphoglycerate (in erythrocytes).
*c*eHb	concentration of effective haemoglobin (in blood) (haemoglobin oxygen binding capacity).
*c*tHb	concentration of total haemoglobin (in blood).
ctO_2	concentration of total oxygen (in blood).
c_x	concentration of extractable oxygen (in blood).
*F*COHb	fraction of carboxyhaemoglobin (in total haemoglobin).
*F*HbF	fraction of foetal haemoglobin (in total haemoglobin).
*F*MetHb	fraction of methaemoglobin (in total haemoglobin).
FO_2dI	fraction of oxygen in dry inspired air.
*F*va	fraction of mixed venous blood in arterial (total functional veno-arterial shunting).
pCO_2	tension of carbon dioxide (in blood).
pH	negative logarithm of hydrogen ion activity.
pO_2	tension of oxygen (in blood).
pO_2hI	partial pressure of oxygen in humidified inspired air.
pO_2A	partial pressure of oxygen in alveolar air.
pO_2a	tension of oxygen in arterial blood.
pO_2v	tension of oxygen in mixed venous blood.
p_x	extraction tension (of oxygen in arterial blood), defined as the oxygen tension resulting from extracting 2.3 mmol of oxygen per litre of blood.
p_{50}	half-saturation tension (of oxygen in blood).
Q_x	oxygen compensation factor (of arterial blood).
R_Q	CO_2/O_2 exchange ratio (respiratory quotient).
sO_2	saturation fraction of oxygen in haemoglobin.
*T*Pt	patient temperature.

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