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Oxygen and acid-base parameters of arterial and mixed venous blood, relevant versus redundant

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A complete pH and blood gas analysis of arterial and mixed venous blood may comprise more than forty different quantities. We have selected sixteen, including patient temperature. The arterial oxygen tension group includes the oxygen tension, fraction of oxygen in inspired air, and fraction of mixed venous blood in the arterial (total physiological veno-arterial shunting). The haemoglobin oxygen capacity group includes effective haemoglobin concentration and fractions of carboxy- and methaemoglobin. The haemoglobin oxygen affinity group includes half-saturation tension and estimated 2,3-diphosphoglycerate concentration of erythrocytes. In a neonatal care unit fraction of fetal haemoglobin need to be included. The arterial oxygen extractivity is measured as the oxygen extraction tension, which indicates the degree of compensation among the oxygen tension, capacity, and affinity. The mixed venous group includes mixed venous oxygen tension, and, when measured, cardiac output, and oxygen consumption rate. The acid-base status includes blood pH, arterial carbon dioxide tension, and extracellular base excess. Other quantities such as haemoglobin oxygen saturation, respiratory index, total oxygen concentration (oxygen content), oxygen extraction fraction, oxygen delivery, and several others, provide no essential additional clinical information and are therefore redundant.

Key words: acid-base status; blood gas analysis; carbon dioxide; intensive care; metabolic rate; mixed venous blood; pH;

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Oxygen tension or oxygen saturation are often the only oxygen parameters reported with an arterial blood gas analysis, and therapy is based on simple rules of thumb, such as giving supplementary oxygen if the tension is less than 10.7 kPa (80 mmHg) or the saturation less than 90 %.

A pH-blood gas analyzer, now standard equipment in all intensive care units, measures the pO_2, pCO_2, and pH of the blood (at 37 °C), and often measures the ambient pressure as well for automatic calibration of the gas sensors. A multi wavelength haemoximeter (also called CO-oximeter) measures the concentrations of oxy-, deoxy-, carboxy-, met-, and sulfhaemoglobin, and sometimes the concentration of fetal haemoglobin may be derived. From these directly measured quantities it is possible to get a series of calculated quantities. We have a freely available computer program for this purpose: the Oxygen Status Algorithm (2,3). At present the program collects data on-line only from analyzers of the ABL5xx series combined with an OSM™3 HAEMOXIMETER™ (Radiometer, Copenhagen) (4). Up to 2000 consecutive measurements may be accumulated. Some of the calculations require supplementary information such as patient temperature and fraction of inspired oxygen. The program generates a *patient status report* with a list of twenty six quantities with reference values, a list of laboratory diagnoses, and a graphical display of the oxygen status (the oxygen graph) and the acid-base status (the acid-base chart). The program also generates a condensed *cumulated patient report*. Given the option of calculating more than forty oxygen related parameters, the question of redundancy becomes pertinent.

The purpose of the present text is to show the lay-out of our patient report with cumulated pH-blood gas data (Table 1) and discuss the relevance of the various quantities. The data are grouped in relation to the arterial oxygen status, mixed venous blood, and acid-base status. The arterial oxygen status is subdivided into quantities related to the oxygen tension, haemoglobin oxygen capacity, haemoglobin oxygen affinity, and oxygen extractivity (a new term we propose).

TEMPERATURE

Patient temperature is important in relation to gas tensions and pH. According to the *pH-stat approach*, pH and pCO_2 are regulated to be constant *in vivo* as in hibernating animals, by accumulation or excretion of CO_2 as temperature decreases or increases. Hence, reference values for pH and pCO_2 remain constant when reported at the actual patient temperature. According to the *α-stat approach*, blood pH and pCO_2 change with temperature to the same extent *in vivo* and *in vitro* as in poikilothermic animals. Hence, reference values remain constant when pH and pCO_2 are reported at a fixed standard temperature of 37 °C, regardless of patient temperature. This controversy regarding pH and pCO_2 reference values does not apply to pO_2. Failure to take a patient temperature of 36 °C into account when calculating alveolar pO_2 may result in a negative value for the functional veno-arterial shunting where it should have been as high as 10 %. For this reason the authors prefer to report all values at body temperature and adjust reference values accordingly. A more detailed analysis of temperature effects is given elsewhere (3).

ARTERIAL OXYGEN TENSION

Arterial oxygen tension:

pO_2a is the first of a triad of quantities which determine the oxygen extractivity (see later). The causes of change in pO_2a are changes in ambient pressure, fraction of inspired oxygen, arterial carbon dioxide tension, and functional veno-arterial shunting, all described in the following. The pO_2a is essential to evaluate the risk of oxygen toxicity especially in the newborn. Hypoxaemia is synonymous with a decreased pO_2a.

Table 1. Cumulated patient report generated with the Oxygen Status Algorithm computer program. The lay out was designed to provide a condensed overview of the oxygen status and the acid-base status of the blood with a logical grouping of the many quantities. The columns show the values as a function of time with the most recent data at top. pCO_2 is flagged with "R" when the patient is on respirator. The reference values shown in the header refer to the temperature of the last measurement and indicate the normal mean value. The patient had a coronary bypass operation with haemodilution and cooling. The first measurement (on April 21) was made just before extracorporeal circulation, the second just after, the third in the recovery room. The patient developed a large anterior transmural infarction and was unable to maintain an adequate cardiac output. On April 23 an intra-aortic balloon pump was inserted. The mixed venous pO_2 was consistently decreased due to the low cardiac output. The oxygen extraction tension also tended to be slightly decreased due to the low haemoglobin concentration. The tissue hypoxia resulted in a progressive and fatal metabolic acidosis.

Patient: Anonymous. Male, 65 y. Oxygen Status Algorithm: Cumulated Patient Report

		O2 TENSION			CAPACITY			AFFINITY		EXTRA	MIXED VENOUS			ACID BASE		
Sample time	TPt	**pO2a**	FO2	Fva	**ceHb**	FCO	FMet	**p50**	cDPG	**px**	pO2	V'B	RMR	pHa	pCO2a	cBE
yymmdd hh:mm	C	kPa	%	%	mM	%	%	kPa	mM	kPa	kPa	L/min	%	1	kPa	mM
References:	38.5	9.5	21	11	9.2	0.5	0.5	3.8	5	5.2	5.0	5.2	100	7.42	5.2	1
950423 13:20	38.5	**16.0**	100	20	**7.3**	0.7	0.1	**4.6**	6	**6.0**	4.6	3.3	118	7.23	2.9R	-16
950423 06:20	38.0	**10.5**	100	26	**7.5**	0.7	0.1	**4.1**	5	**5.0**	4.0	3.5	124	7.33	3.7R	-10
950423 03:30	38.0	**12.1**	100	20	**7.5**	0.7	0.1	**3.9**	5	**5.0**	3.5	3.5	149	7.36	3.7R	-8
950423 01:15	37.5	**6.9**	100	35	**7.1**	0.7	0.1	**4.1**	5	**4.1**	2.7	5.5	226	7.28	5.2R	-7
950422 22:00	36.9	**7.5**	100	37	**6.2**	0.7	0.1	**4.2**	5	**4.1**	3.3	3.6	117	7.31	7.3R	-5
950422 20:15	36.9	**11.3**	100	22	**5.5**	0.7	0.1	**3.8**	5	**4.1**	3.0	2.9	111	7.32	4.8R	-6
950422 18:05	36.9	**16.7**	100	18	**5.6**	0.7	0.1	**4.4**	5	**5.0**	3.0	3.1	133	7.20	6.3R	-8
950422 11:10	36.9	**10.8**	100	20	**6.4**	0.4	0.9	**3.0**	1	**3.7**	2.6	2.6	105	7.30	5.2R	-6
950422 08:15	38.3	**10.8**	100	22	**6.4**	0.4	0.9	**3.5**	1	**4.1**	2.9	2.9	106	7.23	6.2R	-7
950421 14:55	37.6	**22.5**	100	19	**6.4**	0.4	0.9	**3.6**	4	**4.6**	3.9	3.1	96	7.34	6.0R	-1
950421 11:00	32.0	**28.3**	100	21	**5.7**	0.4	0.9	**2.2**	4	**2.7**	2.5	3.3	148	7.49	3.6R	-2
950421 09:10	36.8	**56.3**	100	14	**6.9**	0.3	0.7	**2.9**	4	**4.3**	3.8	3.1	74	7.52	4.0R	5

Ambient pressure:
*P*amb is rarely reported with routine blood gas measurements, even though a low pressure (high altitude) is one of the causes of arterial hypoxaemia. Changes in local barometric pressure are too small to be clinically relevant.

Fraction of oxygen in dry inspired air:
FO_2dI (FiO_2) should be reported routinely. When the patient receives supplementary oxygen on a mask, the fraction of oxygen should be estimated on the basis of an empirical relationship between oxygen flow and FO_2dI (5). It should be recalled that a considerable uncertainty in an elevated FO_2dI has little influence on the calculated veno-arterial shunting, when the alveolar oxygen tension falls on the horizontal part of the oxygen binding curve.

Oxygen tension of humidified inspired air:
pO_2hI is calculated as $FO_2\text{dI} \cdot (P\text{amb} - pH_2O)$, where pH_2O is partial pressure of saturated water vapour at patient temperature. It is redundant to report pO_2hI in addition to the fraction of oxygen in inspired air.

Oxygen tension in alveolar air:
pO_2A is calculated from the alveolar air equation as $pO_2\text{hI} - pCO_2\text{a} \cdot (R_Q^{-1} - FO_2\text{dI} \cdot (R_Q^{-1} - 1))$, where R_Q is the expiratory CO_2/O_2 exchange ratio and pCO_2a is the carbon dioxide tension of the arterial blood at the temperature of the patient. When R_Q=1 or FO_2dI=1, the equation simplifies to $pO_2\text{A} = pO_2\text{hI} - pCO_2\text{a}$. pO_2A is needed as an intermediary in the calculation of the functional veno-arterial shunt, but is not important in itself.

Respiratory CO_2/O_2 exchange ratio:
R_Q, is needed for calculation of the alveolar pO_2. It may be measured continuously with CO_2 and O_2 sensors in the airway (6), but usually the value is taken to be 0.86 and not specified in the cumulated report.

Fraction of mixed venous blood in the arterial:
The total physiological v-a shunt, *F*va or *F*shunt, is calculated as $(ctO_2\text{A} - ctO_2\text{a})/(ctO_2\text{A} - ctO_2\text{v})$. ctO_2 is the concentration of total oxygen in blood in equilibrium with the alveolar air (A), in arterial blood (a), and in mixed venous blood (v), respectively. When mixed venous blood is unavailable ctO_2v is replaced by ctO_2a – 2.3 mmol/L. Zetterström used 2.2 mmol/L (7).

Even with this approximation, the shunt estimate is a better indicator of pulmonary function than the difference DpO_2Aa (= $pO_2\text{A}–pO_2\text{a}$) or the ratios $pO_2\text{a}/FO_2$dI, $pO_2\text{a}/pO_2$A, $DpO_2\text{Aa}/pO_2$a, all proposed as useful parameters, mostly because they are easier to calculate than the shunt (8).

Arterial haemoglobin oxygen saturation:
sO$_2$a, defined as the fraction of oxyhaemoglobin in the sum of oxy- and deoxyhaemoglobin, does not appear among the relevant quantities in the cumulated report (Table 1). Haemoglobin is a natural colour indicator for pO$_2$, and sO$_2$ is primarily an indirect measure of pO$_2$, albeit necessary for calculation of p_{50} and several other quantities. sO$_2$a may be reported in the *patient status report* if it is needed for comparison with simultaneous pulse oximeter monitoring.

The fraction of oxyhaemoglobin in total haemoglobin, sometimes erroneously called oxygen saturation, is totally redundant (9, 10).

HAEMOGLOBIN OXYGEN CAPACITY

Concentration of effective haemoglobin:
*c*eHb is the second of the triad of quantities determining the oxygen extractivity. It is the sum of oxy- and deoxyhaemoglobin and equivalent to the haemoglobin oxygen binding capacity. When arterial and mixed venous blood are measured simultaneously the best accuracy of the calculations is obtained by using the mean value of the effective haemoglobin concentration for both. The same applies to the fractions of carboxy- and methaemoglobin.

In the older literature haemoglobin oxygen binding capacity was reported in vol%, as the volume of oxygen (mL, STP) bound to haemoglobin in 100 mL of fully oxygenated blood.

Concentration of total haemoglobin:
*c*tHb is redundant when the concentration of effective haemoglobin is reported. Sometimes the *haematocrit* is specifically requested as a rheological parameter. Many blood gas analyzers calculate the haematocrit by simply dividing the total haemoglobin concentration with a standard value for the mean cell haemoglobin concentration (20 or 21 mmol/L).

Fraction of carboxyhaemoglobin in total haemoglobin:
*F*COHb is a relevant quantity since an increased *F*COHb not only reduces the haemoglobin oxygen binding capacity but also increases the binding affinity. Slightly increased carboxyhaemoglobin is often observed in critically ill patients due to haemoglobin sequestration.

Fraction of methaemoglobin in total haemoglobin:
*F*MetHb reduces the effective haemoglobin concentration and increases the oxygen binding affinity. It is rarely much increased in patients in the intensive care unit although a small increase may be seen in patients treated with nitrites.

Increased sulfhaemoglobin is observed so rarely that it is omitted from the cumulated report.

Concentration of total oxygen in the arterial blood:
*c*tO$_2$a, often called the “oxygen content”, is an important intermediary result in the calculation of several of the other quantities, e.g. the v-a shunt and the oxygen consumption rate, but does not itself contribute to the interpretation of the oxygen status.

HAEMOGLOBIN OXYGEN AFFINITY

Half-saturation tension:
p_{50} is the oxygen tension which provides 50 % oxygen saturation at the actual pH and pCO$_2$. It is the third of the triad of quantities which determine the oxygen extractivity. The half-saturation tension has been neglected, mostly because a potent, non-toxic drug for easy and rapid manipulation is still awaiting. It should be recalled, however, that decreasing pH from 7.5 to 7.2, which increases p_{50} from 3.2 to 4.3 kPa, has the same effect on the oxygen extractivity as increasing inspired oxygen from 21 % to 100 %, or increasing the haemoglobin concentration from 7.0 to 12.0 mmol/L.

The half-saturation tension can only be calculated if the oxygen saturation is less than 97 %, and if the haemoximeter is not calibrated with the utmost accuracy this limit reduces to 95 % or even 90 %. For this reason, whenever possible, p_{50} and *c*DPG are derived from measurements of mixed venous, central venous, or peripheral venous blood where oxygen saturation is usually lower.

Other measures of the haemoglobin oxygen affinity, such as p_{10}, p_{70}, or p_{90} (pO$_2$ at sO$_2$ values of 10 %, 70 %, and 90 %, respectively) or $s_{3.6}$ or s_5 (sO$_2$ at pO$_2$ values of 3.6 kPa and 5.0 kPa, respectively) are redundant.

Concentration of 2,3-diphosphoglycerate in erythrocytes:
*c*DPG is one of the determinants of the haemoglobin oxygen affinity. It is estimated on the basis of the deviation of the corrected half-saturation tension from its normal target value after correcting for temperature, pH, pCO$_2$, carboxyhaemoglobin, methaemoglobin, and fetal haemoglobin. It is a matter of temperament whether one prefers the corrected p_{50} or the estimated *c*DPG. If estimation of *c*DPG (or corrected p_{50}) returns an unrealistic value the presence of a haemoglobin variant with abnormal oxygen affinity should be contemplated (provided analytical error has been ruled out).

Fraction of fetal haemoglobin:
*F*HbF increases the haemoglobin oxygen affinity and should be reported in a neonatal care unit. Since measurement of fetal haemoglobin requires some manipulation (complete oxygenation of the blood with pH control) before measurement in the haemoximeter, it is customary to assume a default value of 0.5 % fetal haemoglobin for adults.

OXYGEN EXTRACTIVITY

Oxygen extraction tension of the arterial blood:
p_x is a measure of the "oxygen extractivity" of the arterial blood, the property which allows tissues to extract the usual amount of oxygen (about 2.3 mmol per litre) without a fall in oxygen tension below the usual venous level (about 5 kPa). The cells are only able to extract enough oxygen if the end capillary pO_2 remains above a critical value which ensures a sufficient diffusion gradient. A convenient quantitative measure of the oxygen extractivity is the oxygen extraction tension, defined as the oxygen tension measured in the arterial blood (at the actual patient temperature) after extraction of 2.3 mmol of oxygen per litre blood (11). A decreased p_x may be due to a decreased arterial pO_2, a decreased *ce*Hb, or a decreased p_{50}. However, a decreased arterial pO_2 may be compensated by an increased *ce*Hb and/or increased p_{50}; and vice versa. The p_x indicates whether a disturbance in one of the triad (the arterial pO_2, the effective haemoglobin concentration, and the p_{50}) is compensated or not. A decreased arterial pO_2 should not automatically trigger supplementary oxygen. If the oxygen extraction tension is normal the hypoxaemia is compensated and the patient should not require extra oxygen, unless it is decided to raise the p_x to a supra-normal value to compensate for an insufficient cardiac output.

It is possible to define oxygen extraction tensions based on extraction of other amounts of oxygen than 2.3 mmol/L. As in the case of p_{10}, p_{70}, or p_{90} as measures of the haemoglobin oxygen affinity such additional oxygen extraction tensions as measures of the oxygen extractivity are redundant.

Concentration of extractable oxygen:
Bryan-Brown and coworkers in 1973 were the first to suggest a measure of the oxygen extractivity: the concentration of *consumable* oxygen in the arterial blood, defined as the amount of oxygen which can be extracted per litre of arterial blood at an oxygen tension of 20 torr (2.7 kPa)(12,13). We defined the concentration of extractable oxygen, c_x, as the amount of oxygen which can be extracted at 5 kPa (at 37 °C) (11). The concentration of consumable oxygen, c_x and p_x are all closely correlated. Since we have chosen p_x as measure of the oxygen extractivity the others become redundant.

The same applies to the Oxygen compensation factor, Q_x, defined as a standard extractable oxygen (2.3 mmol/L) divided by the actual c_x (11). Q_x may be interpreted as the factor by which the cardiac output should increase in order to compensate for a decreased oxygen extractivity.

MIXED VENOUS BLOOD AND CARDIAC OUTPUT

Mixed venous oxygen tension:
pO_2v is an important indicator of the average end capillary oxygen tension and hence important for the average diffusion gradient for oxygen from haemoglobin to cytochrome aa_3 (14). Therapy should be directed towards maintaining the mixed venous pO_2 above a critical value, usually about 3.5 kPa. The critical value may, however, be higher in cases of dys-perfusion (tissue oedema, micro embolism, arterio-venous shunting, or inhibition of the cytochromes) or hypermetabolism (fewer, oxygen debt, shivering, or increased muscle tonus) (11,14). The mixed venous pO_2 does not provide any information on individual organs.

The pO_2 of central venous blood is a useful parameter although it cannot replace the mixed venous pO_2; it is flagged with a "c" in the cumulated report. Normally pO_2 of the upper caval blood is slightly lower than that of the lower caval blood and the difference between the two might be an indicator of pathologic flow distribution.

Mixed venous oxygen saturation:
sO_2v is redundant when the mixed venous oxygen tension is reported. However, if the mixed venous oxygen saturation is monitored continuously with an optical catheter tip sensor, it may be decided to report the mixed venous saturation from the haemoximeter for comparison or in vivo calibration of the catheter tip sensor.

Concentration of total oxygen in mixed venous blood:
ctO_2-,v is an important intermediary for calculation of the veno-arterial shunting and the oxygen consumption rate, but does not add information in itself. The same applies to the arterio-venous oxygen concentration difference, $DctO_2av$, and the oxygen extraction fraction, $DctO_2av/ctO_2a$. None of them are included in the cumulated report (Table I).

Total carbon dioxide in mixed venous blood:
$ctCO_2v$ could be a useful intermediary for calculation of the CO_2/O_2 exchange ratio: $R_Q = DctCO_2va/DctO_2av$. Unfortunately, $DctCO_2va$ is too inaccurate for this purpose, being a small difference between two large numbers, both subject to a considerable inaccuracy.

Cardiac output:
$V'B$ may be measured by thermodilution using a Swan-Ganz catheter. It is one of the important physiological functions and necessary for calculation of the oxygen consumption rate.

Amount of substance rate of oxygen consumption:
$\acute{n}O_2$ is calculated as the product of cardiac output and arterio-venous oxygen concentration difference: $\acute{n}O_2 = V'B \cdot DctO_2av$. The result appears as mmol/min. The amount of oxygen may be expressed as volume (at standard temperature and pressure), and the result, $V'O_2$, is then expressed in mL/min. Oxygen consumption is strongly dependent on body size and is often reported relative to body mass or relative to body surface area. In the latter case patient height is needed in addition to weight, to be able to calculate surface area from the DuBois equation. Sometimes the amount of energy produced in the combustion process is calculated using a standard value of 450 kJ/mol, although the accurate value depends

on the fuel (carbohydrate, fat, protein, or alcohol); the result is then reported as the metabolic rate in W, W/kg, or W/m^2. Even when related to body weight or body surface area, the reference values depend on age, sex, and body temperature. In view of all the different ways of expressing the oxygen consumption rate it seems appropriate to report the result relative to the normal basal oxygen consumption rate taking age, sex and temperature into account, R_{MR}. Even thus expressed, the value is difficult to interpret: does a decreased value, of say 75 %, indicate that the patient needs more oxygen or is it merely a result of pharmaceutical sedation? Or does an increased value, of say 150 %, indicate that the patient is repaying an oxygen debt, or does it indicate that he is hyperactive and needs sedation? More experience is needed to evaluate the benefit of routine determination of the oxygen consumption rate.

Amount of substance rate of oxygen delivery:
$\acute{n}O_2$flow is calculated as the product of the cardiac output and the concentration of total oxygen in the arterial blood: $\acute{n}O_2\text{flow} = V\text{'B} \cdot c\text{t}O_2\text{a}$. The result appears in mmol/min. The amount of oxygen may also be expressed as volume (standard temperature and pressure) and the conventional symbol then is Do_2 with the unit mL/min. As with the oxygen consumption rate the result can be expressed relative to body mass or body surface area. Although this quantity has received considerable attention in the literature it is not among the relevant quantities. Oxygen consumption rate divided with oxygen delivery is the same as fractional oxygen extraction, another redundant quantity.

ACID-BASE STATUS

Hydrogen ion activity (negative logarithm):
pHa, is the indispensable measure of acidaemia or alkalaemia. It is so entrenched in our daily life as a measure of the acidity of lotions and beverages that it seems futile to try to replace it by the hydrogen ion concentration.

Carbon dioxide tension of the arterial blood:
$p\text{CO}_2\text{a}$ is the measure of alveolar ventilation and of a respiratory acid-base disturbance. Hypercapnia or hypocapnia are important causes of change in the arterial $p\text{O}_2$.

Concentration of titratable base in extracellular fluid:
cBEcf, also called the extracellular base excess, is the measure of a metabolic acid-base disturbance, i.e. accumulation of non-volatile acid or base in the extracellular fluid. It is defined as the titratable base when titrating a model of the extracellular fluid to a pH = 7.40 at $p\text{CO}_2$ = 5.33 kPa at 37 °C (without changing the total oxygen concentration). The model of extracellular fluid is defined as the blood diluted three fold with its own plasma. In actual practise cBEcf is calculated from the pH, $p\text{CO}_2$, and ctHb using the Van Slyke equation. The extracellular base excess remains virtually constant during acute respiratory changes, where the whole blood base excess or the plasma base excess change slightly and in opposite direction, due to a redistribution of hydrogen ions. The extracellular base excess is the most relevant indicator of metabolic acidosis or alkalosis (15). Base excess ought to be called hydrogen ion deficit, or the sign should be reversed and the quantity called the hydrogen ion excess.

Concentration of buffer base and strong ion difference:
Buffer base was defined by Singer and Hastings (16) as the sum of the "strong" cations minus the sum of the strong "anions". This equals the sum of the buffer anions (minus the sum of the buffer cations, if any). Plasma buffer base was later renamed "strong ion difference" (SID) (17). Buffer base is now obsolete (15).

Concentration of bicarbonate in plasma:
$c\text{HCO}_3^-$, is sometimes used as an indirect measure of accumulation of acid or base. Since cBEcf is a more direct measure of a metabolic acid-base disturbance the bicarbonate concentration is redundant for this purpose. It is needed, however, for calculation of the anion gap, and then belongs to the group of electrolytes: sodium, potassium, and chloride.

Standard bicarbonate:
The standard bicarbonate, defined by Jørgensen and Astrup as the plasma bicarbonate concentration of blood equilibrated with a gas mixture with a $p\text{CO}_2$ of 5.33 kPa (at 37 °C and full oxygenation), is obsolete. The same applies to the related quantity, standard pH, and older quantities such as the CO_2 combining power.

Concentration of lactate:
$c\text{Lac}^-$ supplements the base excess as a measure of metabolic acidosis, specifically lactic acidosis. When the pH-blood gas analyzer is equipped with a lactate sensor, lactate should appear on the cumulated report along with the base excess.

Anion gap or undetermined anions:
$c\text{UA}^-$ is defined as the sum of measured cations minus the sum of measured anions in the plasma, often calculated simply as $c\text{Na}^+ + c\text{K}^+ - c\text{Cl}^- - c\text{HCO}_3^-$. This approximately equals the concentration of albuminate plus other organic anions, such as lactate or hydroxybutyrate. With the introduction of combined pH-blood gas-electrolyte analyzers calculation of the anion gap becomes very easy. However, if blood lactate has been measured with a lactate sensor, the anion gap seems to be unnecessary.

CONCLUSION

The many oxygen parameters and acid-base quantities derived on the basis of routine arterial blood sampling and

measurement may seem to cause more confusion than clarification. This is probably the first reaction to our cumulated report, which contains as many as sixteen quantities in addition to date and time, in spite of our attempts to reduce the number. The second reaction may be consternation that the oxygen saturation, the oxygen content, oxygen delivery, extraction fraction, respiratory index, etc. are missing among the relevant quantities. The third reaction should be a systematic effort to evaluate the clinical usefulness of the more elaborated pH-blood gas analysis. This involves a considerable teaching programme for the staff of the intensive care unit and a systematic quality assessment of the proposed cumulated patient report.

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