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Clinical application of the pO_2–pCO_2 diagram

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Based on the classic, linear blood gas diagram a logarithmic blood gas map was constructed. The scales were extended by the use of logarithmic axes in order to allow for high patient values. Patients with lung disorders often have high arterial carbon dioxide tensions, and patients on supplementary oxygen typically respond with high oxygen tensions off the scale of the classic diagram. Two case histories illustrate the clinical application of the logarithmic blood gas map. Variables from the two patients were measured by the use of blood gas analysis equipment. Measured and calculated values are tabulated. The calculations were performed using the *oxygen status algorithm*. When interpreting the graph for a given patient it is recommended first to observe the location of the marker for the partial pressure of oxygen in inspired, humidified air (I) to see whether the patient is breathing atmospheric air or air with supplementary oxygen. Then observe the location of the arterial point (a) to see whether *hypoxemia* or *hypercapnia* appears to be the primary disturbance. Finally observe the alveolo-arterial oxygen tension difference to estimate the degree of *veno-arterial shunting*. If the mixed venous point (v) is available, then observe the value of the mixed venous oxygen tension. This is the most important indicator of *global tissue hypoxia*.

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Key words: acid-base status; blood gas tensions; hypercapnia; hypoxaemia; supplementary oxygen, veno-arterial shunting.

WALLACE O. Fenn and Hermann Rahn in 1955 (1) used a diagram with the partial pressure of oxygen (pO_2) on the abscissa and the partial pressure of carbon dioxide (pCO_2) on the ordinate to illustrate important relationships between these partial pressures in inspired air (I), mixed expired air (E), end expired air (eE), and ideal alveolar air (A). The latter represents alveolar air from alveoli with the same ventilation/perfusion ratio as the overall pulmonary ventilation/perfusion ratio, while end expired air represents mixed alveolar air from all alveoli. The diagram also illustrates the relationships between the partial pressures in alveolar air and the gas tensions in arterial and mixed venous blood.

The diagram is a useful didactic tool, as elegantly elaborated by John B. West in his classic monograph 'Ventilation/blood flow and gas exchange' from 1965 (2).

The purpose of this report is to re-emphasize the utility of the pO_2–pCO_2 diagram, not only for teaching purposes but also for practical clinical application. In the following, we first describe the theory of the diagram and then illustrate the clinical application using two selected examples. Normal and extreme blood gas values were taken from Siggaard-Andersen (3) and Siggaard-Andersen, Fogh-Andersen, Gøthgen and Larsen (4).

The classic pO_2–pCO_2 diagram

The diagram, shown in Fig. 1, has a linear pO_2 scale as abscissa, extending from 0 to 25 kPa, and a linear pCO_2 scale as ordinate, extending from 0 to 15 kPa. Points 'A', 'eE', 'E', and 'I' fall on a straight line: the 'air line' (Fig. 1). The total distance (A–I) indicates the tidal volume. The relative distances along the line indicate the relative sizes of the alveolar dead space (A–eE) and the total physiological dead space (A–E) as fractions of the tidal volume. The pO_2 of the inspired air is calculated by multiplying the fraction of oxygen in the dry inspired air (FO_2I_{dry}) with the total ambient pressure (Pamb) less the water vapour pressure (pH_2OI):

$$pO_2I = FO_2I_{dry} \cdot (P\text{amb} - pH_2OI) \qquad (1).$$

Notice that 'I' refers to air saturated with water vapour at the temperature of the patient; I_{dry} refers to dry inspired air. The substance fraction of oxygen in dry atmospheric air is 0.2095. Ambient barometric pressure at sea level is 101.325 kPa = 1 atm. The partial pressure of water vapour in humidified inspired air, also called 'saturated water vapour pressure', is 6.27 kPa at 37°C. With these values pO_2I is calculated to be 19.9 kPa.

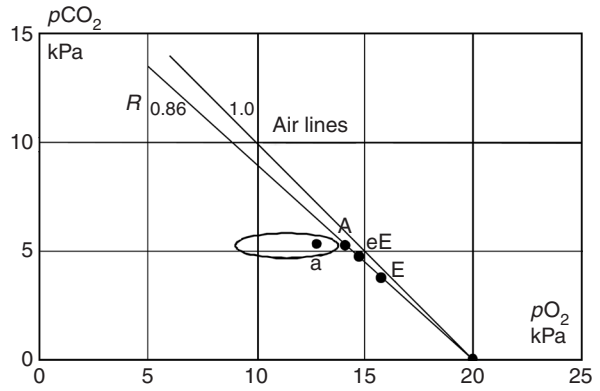

Fig. 1. The pO_2–pCO_2 diagram. Mark 'a' represents arterial blood and the surrounding ellipse the normal area. Mark 'I' indicates humidified inspired air (tracheal air). The position of mark 'A', indicating ideal alveolar air, is calculated from pO_2I and the arterial pCO_2 using the 'alveolar air equation'. The mark 'E' may be plotted when mixed expired air has been analyzed, and the mark 'eE' when a sample of end expiratory air has been analyzed.

The carbon dioxide partial pressure, pCO_2, is 0.03 kPa in atmospheric air, i.e. virtually zero. Therefore the mark 'I' is located on the abscissa ($pCO_2 = 0$ kPa) at a pO_2 of 19.9 kPa.

Mixed total expired air is typically collected in a Douglas bag. End expiratory air, sampled after wash out of all inspired air in trachea and bronchi, represents mixed alveolar air. Ideal alveolar air cannot be sampled. It represents alveolar air from alveoli with the same local ventilation-perfusion ratio as the overall ratio for both lungs.

The pCO_2 and pO_2 of alveolar air are closely correlated: a high pCO_2A is associated with a low pO_2A, and vice versa. The relationship is given by the 'alveolar air equation' which expresses the pO_2 of ideal alveolar air as a function of pCO_2A:

$$pO_2A = pO_2I - pCO_2A \cdot [R^{-1} - FO_2I_{dry} \cdot (R^{-1} - 1)] \quad (2).$$

Not surprisingly, the CO_2/O_2 exchange ratio, R, is a variable in the equation. On a pure carbohydrate diet $R = 1.0$, pure lipid combustion gives $R = 0.70$, and a normal mixed diet gives $R \approx 0.85$. When R is unknown, which is generally the case, a default value of 0.85 is employed. When $FO_2I_{dry} = 1.0$ (i.e. breathing pure oxygen) or $R = 1.0$ then the equation simplifies to $pO_2A = pO_2I - pCO_2A$.

The pCO_2A may be replaced by the pCO_2 of the arterial blood (pCO_2a), the difference being negligible (pCO_2a slightly higher than pCO_2A depending upon the shunt fraction). Hence the alveolar point 'A' may be determined when the arterial pCO_2 has been measured.

The alveolar air equation shows that when pO_2A is plotted as a function of pCO_2A, then the relationship is a straight line with a slope – $[R^{-1} - FO_2dI \cdot (R^{-1} - 1)]$. Hence, when pCO_2A is plotted as a function of pO_2A as in Fig. 1, the slope β is the reciprocal:

$$\beta = -R/[1 - FO_2I_{dry} \cdot (1 - R)] \quad (3).$$

β is the slope of the line connecting the points 'I', 'E', 'eE', and 'A' in Fig. 1. We call this line the 'air line'. When the 'air line' has been established the value of β is calculated as:

$$\beta = -pCO_2eE/(pO_2I - pO_2eE) \quad (4).$$

Rearranging the first equation for β (Eqn 3) provides the R-value as a function of β

$$R = -\beta \cdot (1 - FO_2I_{dry})/(1 + \beta \cdot FO_2I_{dry}) \quad (5).$$

Hence plotting the 'air line' and calculating the slope allows calculation of the R-value, provided $FO_2I_{dry} < 1$; if $FO_2I_{dry} = 1$ then $\beta = -1$ and the denominator becomes zero, i.e. R is undetermined. The slope of the 'air line' numerically approaches the R-value. If $R = 1.0$ then $\beta = -1$ regardless of the value of FO_2I_{dry}. If $FO_2I_{dry} = 1$ then $\beta = -1$ regardless of the value of R. When $R = 0.85$ and $FO_2I_{dry} = 0.2095$, then $\beta = -0.88$, i.e. almost, but not quite the same absolute value as R. Several textbooks ignore the difference between β and R.

For a patient in steady state, an R-value between 0.7 and 1.0 provides information on the composition of the diet. If the patient is not at steady state, the R-value may be lower than 0.7, indicating retention of CO_2 in the body, for example due to hypoventilation or developing metabolic alkalosis. A value above 1.0 indicates excessive elimination of CO_2, for example due to hyperventilation or developing metabolic acidosis.

Mixed expired air is a mixture of alveolar air and inspired air from the physiological dead space. Therefore 'E' falls on the line connecting 'A' and 'I'. The position of 'E' gives a visual impression of the relative size of the physiological dead space, given by the ratio AE/AI.

As shown in Fig. 1, the composition of ideal alveolar air (point A) and mixed alveolar air (end expiratory air) is different, the point 'eE' being somewhat below the point 'A' on the air line. The cause of this difference is that neither ventilation nor blood perfusion is uniform throughout the lungs. The regional pulmonary blood flow per unit lung volume increases from almost zero at the upper parts (apex) to the base of the lungs in a resting, upright person, due to the effect of gravity on the blood. The regional alveolar ventilation of the lung expressed per unit lung volume also increases from the apex to the base – but to a lesser degree. Therefore, the regional ventilation/perfusion ratio (V/Q) decreases from the apex to the base of the lung.

At the apex some alveoli are ventilated but with zero blood flow, i.e. an infinitely high V/Q ratio, equivalent to alveolar dead space (Fig. 2). The average V/Q ratio at the apex is about 3. At the base some alveoli are perfused but with zero ventilation, i.e. a V/Q ratio of zero, equivalent to true shunting. The average V/Q ratio at the base of the lungs is about 0.6. The majority of alveoli are both perfused and ventilated and the overall V/Q ratio is about 0.9, with V about 5 and Q about 5.5 l/min in an adult.

The sigmoid *cumulated* distribution curve (Fig. 2) starts slightly above zero, indicating the fraction of alveoli with zero ventilation (equivalent to a true shunt fraction of about 3%). The curve levels off before reaching 1, indicating the presence of alveoli without any blood flow (equivalent to an alveolar dead space of about 3%). However, in a patient with pulmonary disease, the distribution curve is probably far from log-normal. It may be bi-modal or multi-modal if different parts of the lungs have widely different V/Q ratios.

The V/Q dispersion causes a dispersion of the composition of alveolar air from the individual alveoli. Alveoli with a high V/Q ratio have air compositions approaching that of inspired air. The R-value of these alveoli approaches a value of about 10, because the blood gives off almost all CO_2 (about 23 mmol/l) and takes up O_2 corresponding to $pO_2I = 19.9$ kPa (about 2.3 mmol/l). An R-value of about 10 gives a slope, β, of about -3.5.

Alveoli with a low V/Q ratio have air compositions approaching the gas tensions of mixed venous blood, i.e. pCO_2 about 5.3 kPa and pO_2 about 5.0 kPa for normal values. The slope of the air line, β, therefore is about $5.3/(19.9–5) = -0.356$, corresponding to an R-value of 0.304. Fig. 3 shows the pO_2 and pCO_2 values for all individual alveoli. The values form a curve starting at 'I' with slope -3.5, passing through A, and ending at the point 'v', representing mixed venous blood. The point eE is slightly below the curve because it represents the weighted average of the dispersion of points along the convex curve.

The composition of capillary blood from different alveoli also differs. Assuming that there is complete diffusion equilibrium for carbon dioxide as well as oxygen, the pCO_2 of blood and alveolar air from each individual alveolus must be identical and the same applies to pO_2. Nevertheless, mixed alveolar air (end expiratory air) and mixed blood from all the alveoli will have different pCO_2 as well as pO_2, because the alveolar air predominantly arises from alveoli with a high V/Q ratio, while the blood predominantly arises from alveoli with a low V/Q ratio. Therefore the blood pO_2 is somewhat lower than the alveolar pO_2, while the blood pCO_2 is only slightly higher than the alveolar pCO_2. The composition of mixed blood is more difficult to calculate than that of mixed alveolar air. Mixing two equal volumes of blood with different pO_2 values does not provide the mean value of the two pO_2 values. It provides the mean value of the concentration of total oxygen in the two blood volumes. Due to the curvature of the oxygen binding curve the resulting pO_2 value is lower than the mean pO_2.

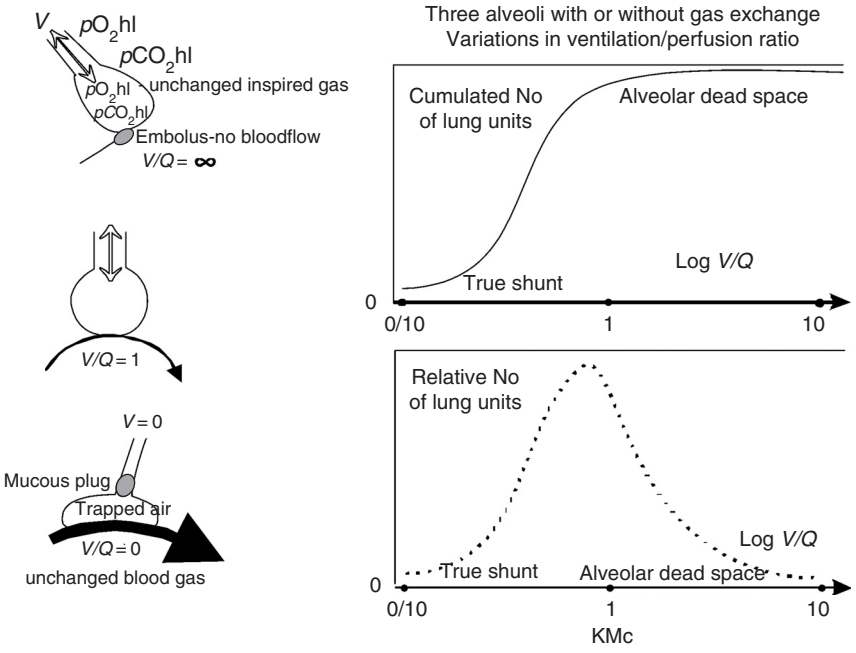

Fig. 2. Ventilation–perfusion dispersion among different alveoli in the upright lung. The upper alveolus is distended while the blood flow is interrupted creating an alveolar dead space. The lower alveolus has no ventilation, but a high blood flow, creating a true shunt. Between these extremes all possible ventilation–perfusion ratios (V/Q) exist. A hypothetical distribution curve for log (V/Q) is shown (dotted curve), as well as the sigmoid-cumulated distribution curve.

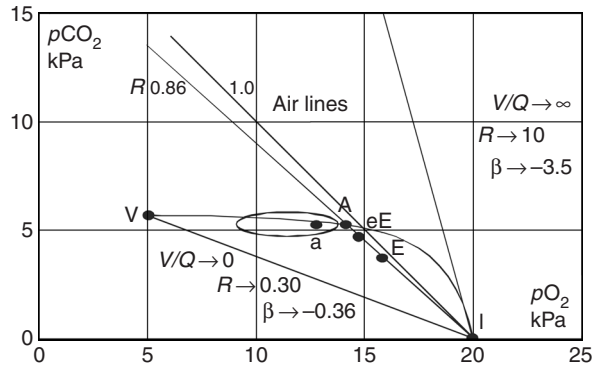

Fig. 3. The pO_2–pCO_2 *diagram with the 'alveolar curve' indicating the composition of alveolar air from individual alveoli. The majority of alveoli have compositions close to the mark 'A' for ideal alveolar air, but alveoli with a very high* V/Q *ratio have values close to the mark 'I', while alveoli with very low* V/Q *ratio have values close to the mark 'v' for mixed venous blood. The mark 'a' indicates the composition of arterial blood. The slope (β) of the air lines is indicated for* $V/Q \rightarrow 0$ *and* $V/Q \rightarrow \infty$.

While the point 'eE' so to say slides down from 'A' along the 'air line' (Fig. 3) in proportion to the *V/Q* dispersion, the arterial point 'a' slides left from the point 'A' along an almost horizontal curve, which gradually bends upwards towards the point 'v'.

The alveolo-arterial pO_2 difference reflects the size of *V/Q* dispersion but also increases with true veno-arterial shunting, i.e. blood bypassing functioning alveoli. In special cases, lack of diffusion equilibrium for oxygen between alveolar air and blood may contribute to an increased alveolo-arterial pO_2 difference. All three causes of hypoxemia are reflected in the physiological (apparent) shunt fraction. The latter is defined as the shunt equation:

$$F_{va} = (ctO_2A - ctO_2a)/(ctO_2A - ctO_2v) \qquad (6).$$

The ctO_2A is the concentration of total oxygen in blood with the same pO_2 and pCO_2 as the alveolar air, ctO_2a is the concentration of total oxygen in arterial blood, and ctO_2v that of mixed venous blood.

The size of the physiological shunt fraction due to *V/Q* dispersion can be estimated by calculation. Assuming that the standard deviation of the logarithmic *V/Q* dispersion is 1, corresponding to a 95% interval of *V/Q* from 1/4 to 4, then the estimated physiological shunt fraction is about 3%. In other words the *V/Q* dispersion accounts for only 50% or less of the normal value for the physiological shunt fraction (5.7–13% in Table 1). A very large *V/Q* dispersion is required to cause a substantial increase in physiological shunt fraction. Therefore the main cause of a high physiological shunt fraction remains to be true veno-arterial shunting, either true intrapulmonary shunting (e.g. atelectases) or true extrapulmonary shunting (e.g. cardiac veins draining into the left atrium or veno-arterial shunting due to congenital cardiac disease).

Model calculation

The *V/Q*-ratio of one lung is assumed to be 2 (two parts of air and one part of blood) and in the other lung 0.5 (one part of air and two parts of blood). Hereby the total *V/Q* is one. Let us further assume that the patient is on a pure carbohydrate diet ($R = 1.0$) and that total diffusion equilibrium is established for both oxygen and carbon dioxide in both lungs.

Calculations show an apparent veno-arterial shunt fraction of 5.2% although no true shunt is present. In the lung with $V/Q = 2$ the *R* equals 1.8 and in the lung with $V/Q = 0.5$ the *R* is 0.6. The total *R* is 1.

Although alveoli with a high *V/Q* ratio have a high CO_2/O_2 exchange ratio (*R*-value) compared with alveoli with a low *V/Q* ratio, there is no relationship between the overall *V/Q* ratio and the overall CO_2/O_2 exchange ratio. The overall CO_2/O_2 exchange ratio depends on the type of food consumed (carbohydrate, protein or fat). The overall *V/Q* ratio depends on the cardiac and the pulmonary function. The CO_2/O_2 exchange ratio remains about 0.85 on a mixed diet regardless of the *V/Q* ratio. The overall *V/Q* ratio as such has only minor clinical interest, but the *V/Q dispersion* among different alveoli is clinically relevant. A large *V/Q* dispersion causes an alveolo-arterial pO_2 difference and an apparent (physiological) veno-arterial shunting even in the presence of complete diffusion equilibrium in each individual alveolus and in the absence of any true veno-arterial shunting.

The logarithmic blood gas map

Patients with obstructive and restrictive lung disorders often have rather high arterial *carbon dioxide* tensions. Patients on supplementary oxygen frequently respond with high *oxygen* tensions. In order to allow for high patient values, we have extended the scales by the use of logarithmic axes (Fig. 4).

The pO_2 axis now extends from 1 to 300 kPa, the pCO_2 axis from 1 to 20 kPa. The mark 'I' for humidified inspired air would fall far below the abscissa at a pCO_2 of 0.03 kPa. Therefore the mark is placed at a level with the alveolar pCO_2 and hence only indicates pO_2I. Another mark at the same level, the mark '*P*amb', indicates ambient barometric pressure. At sea level '*P*amb' will be close to 100 kPa. The position of 'I' in relation to '*P*amb' indicates the fraction of

Table 1

Laboratory data for two patient cases

	Case 1		Case 2		Reference interval
	Measured values				
Temperature,°C	40.0	39.2	38.2	38.0	36.5–37.5
*P*amb, kPa	101.3	101.3	99.8	100.2	97–104
FO_2I_{dry}	0.21	0.40*	0.21	0.40†	0.2095
pO_2eE, kPa	14.3		12.1		14.0–15.5
pCO_2eE, kPa	4.7		7.0		4.0-6-0
pH (37°C)	7.453	7.471	7.347	7.264	7.37–7.43
pCO_2a (37°C), kPa	4.50	4.23	7.55	5.05	4.91–6.16
pO_2a (37°C), kPa	5.6	7.53	5.24	8.41	9.1–12.4
sO_2	0.802	0.904	0.736	0.896	0.945–0.969
*c*tHb, mmol/l	7.8	7.6	7.5	7.7	8.46–10.34
*F*MetHb	0.008	0.005	0.012	0.010	0.001–0.010
*F*COHb	0.015	0.014	0.035	0.014	0.001–0.010
	Calculated values				
pO_2I, kPa	19.7	37.6	19.6	37.4	19.2–20.8
pO_2A, kPa	14.1	32.6	11.3	32.0	13.0–14.5
R	0.85‡	0.85§	0.92¶	0.92**	0.70–1.00
pH	7.41	7.44	7.33	7.25	7.37–7.43
pCO_2a, kPa	5.2	4.7	8.0	5.3	4.91–6.16
pO_2a, kPa	6.9	8.3	5.7	9.0	9.1–12.4
ctH^+Ecf, mmol/l	0.4	0.6	−5.2	8.8	−3.2–+1.8
*F*va	0.37	0.29	0.41	0.30	0.057–0.130
ΔpO_2Aa, kPa	7.2	23.9	5.6	23.0	2–5
pO_2x, kPa	4.2	4.6	3.4	4.9	4.44–5.42

*FO_2I_{dry} was estimated on the basis of a flow of pure oxygen of 8 l/min on a Venturi mask (T. Waldau, personal communication).
†FO_2I_{dry} was set on respirator.
‡¶*R* was calculated from pO_2eE and pCO_2eE (Eqns 4 and 5).
§**R was assumed to remain the same as the previously measured value. The fraction of fetal haemoglobin was taken to be 0.005 (default value for adults).

oxygen in dry inspired air. The extension of the pO_2 scale to 300 kPa allows plotting values during hyperbaric oxygenation.

The point 'a' indicates the pO_2 and pCO_2 values of the arterial blood as measured with a blood gas analyzer but referring to the temperature of the patient. When mixed venous blood has been collected simultaneously with the arterial, the point 'v' indicating mixed venous blood, is also plotted. In the present case, mixed venous blood was not available and the point 'v' is only shown to illustrate possible values. The normal arterial tensions are indicated by the green elliptic area, whereas the normal area for the mixed venous point is merely outlined (Fig. 4). The arterial oxygen extraction tension, pO_2x, is indicated by the red mark 'x'. It predicts the pO_2 of the arterial blood after an oxygen extraction of 2.3 mmol/l. The normal interval for pO_2x is the same as for the mixed venous pO_2 (point 'v'). If the mixed venous pO_2 (blue point 'v') is lower than pO_2x (red mark 'x') then this indicates that the arterio-venous oxygen extraction is higher than 2.3 mmol/l, which is generally due to a low cardiac output. Similarly a blue point 'v' situated at a higher pO_2 than the red 'x' mark indicates a high cardiac output. On the basis of values for fraction of inspired oxygen, barometric pressure, patient temperature, and arterial and mixed venous blood gas data it is possible to calculate the position of the alveolar point 'A'. The alveolo-arterial pO_2 difference is due to physiological veno-arterial shunting and above the A –a interval the value of the calculated shunt fraction is written. If mixed venous blood gas data are not available the calculation is based on a standard value for the arterio-venous oxygen concentration difference of 2.3 mmol/l, the normal mean value at rest.

The position of the 'air curve' is calculated from the alveolar air equation on the basis of a standard value for *R* of 0.85 unless the true value has been measured by respiratory gas analysis.

Extending from the arterial normal area are four reference areas or bands indicating the four main types of blood gas disturbances: primary hypercapnia, primary hypocapnia, primary hyperoxaemia, and primary hypoxemia. The areas of primary hypercapnia and hypocapnia extend upwards and downwards along the hyperbolic alveolar pCO_2-pO_2 curve. An increase in pCO_2 causes a fall in pO_2, while a fall in pCO_2 causes a small rise in pO_2. An example of

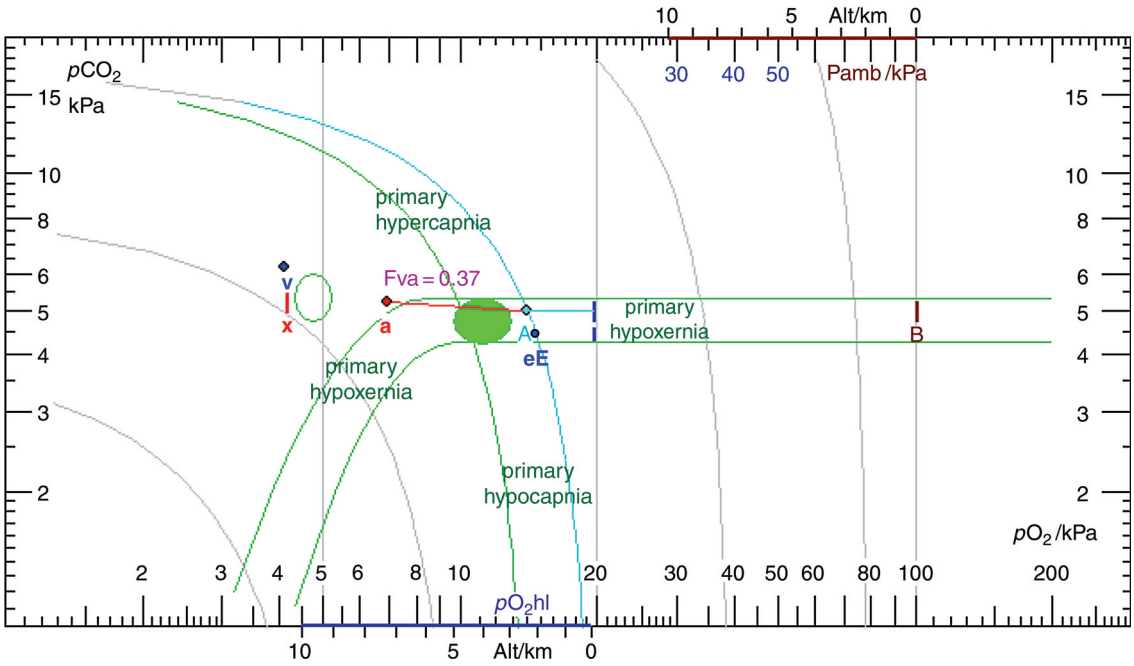

Fig. 4. The logarithmic blood gas map with blood gas data for a 39-year-old patient with pneumonia (case 1). The linear relationship between the alveolar pO_2 *and* pCO_2 *(the 'air line') is now replaced by a hyperbolic curve (the 'air curve'). The green oval area is the reference area for arterial blood. The areas extending up and down from the normal area along the 'air curve' indicate changes in the arterial* pO_2 *with primary changes in* pCO_2*. The area extending to the left of the normal area indicates the fall in* pCO_2 *due to a primary fall in* pO_2*, i.e. the hypoxic respiratory drive. The area extending to the right indicates primary hyperoxaemia, where ventilation and hence* pCO_2 *remains constant. The red arterial point, 'a', for the patient, is located in the area between primary hypoxemia and primary hypercapnia. The point 'A' (light blue colour) represents ideal alveolar air. The dark blue point, 'eE', below the point 'A' on the 'air curve' indicates end expiratory air (mixed alveolar air). Mixed venous blood was not available in this patient. The point 'v', indicating mixed venous blood is only included as an example.*

primary hypercapnia is emphysema where alveolar hypoventilation is dominating while veno-arterial shunting may be less pronounced. An example of primary hypocapnia is hyperventilation due to anxiety or fever. The areas of primary hyperoxaemia and hypoxemia extend horizontally from the normal area with a downward bend of the primary hypoxemia area. An increase in arterial pO_2 due to increased inspired oxygen does not cause any change in ventilation and hence the pCO_2 remains constant. A fall in arterial pO_2, however, due to a low barometric pressure, low inspired oxygen, or veno-arterial shunting causes a stimulation of the peripheral chemoreceptors and hence a hyperventilation with a reduction in pCO_2 as already realized by Rahn & Otis in 1949 (5).

The position of the arterial point in relation to the reference areas and the marks of barometric pressure and inspired oxygen give an immediate visual impression of the causes and severity of disturbances in the blood gases.

When interpreting the graph for a given patient first observe the location of the marker ('I', indicating the pO_2 of humidified inspired air) to see whether the patient is breathing atmospheric air or receives supplementary oxygen. Then observe the location of the arterial point to see whether *hypoxaemia* or *hypercapnia* appears to be the primary and most important disturbance. Finally observe the alveolo-arterial pO_2 difference to estimate the degree of *veno-arterial shunting*. If the mixed venous point is available, then observe the value of the mixed venous pO_2. This is the most important indicator of whole-body or *global tissue hypoxia*.

The following two case histories illustrate the clinical application of the diagram. Variables from the two patients were measured by the use of blood gas analysis equipment. Measured and calculated values are shown in Table 1. The calculations were performed using the oxygen status algorithm (6).

Case 1

A previously healthy male of 39 years was received in the intensive care unit with high fever, cyanosis, dyspnoea and thoracic rales. A chest X-ray showed an infiltrate in the left lung indicating pneumonia. While the patient was breathing ambient air the ventilation was measured to be 14 l/min using a pneumotachograph and a gas monitor, and the end expiratory pO_2 and pCO_2 were measured to be within the reference

interval. An arterial blood sample was obtained from the radial artery and the blood was analyzed with a blood gas analyzer. Measured and calculated data and reference values are given in Table 1.

The blood gas map for this patient (Fig. 4) showed mark 'I' (for pO_2I) at about 20 kPa indicating that he was breathing atmospheric air at a total pressure of approximately 100 kPa (mark 'B'). The *R*-value was calculated from the end expired pO_2 and pCO_2 values to be 0.85 (Eqns 4 and 5). This *R*-value was used for calculation of pO_2 of the ideal alveolar air (Eq. 2). The alveolar pCO_2 value was estimated from the arterial pCO_2 taking the shunt fraction into account by an iterative calculation (6). The alveolar pO_2 and pCO_2 values are illustrated by point 'A' (14.1 and 5.2 kPa, respectively). The arterial point 'a' was well outside to the left of the reference area (9.1–12.4 kPa) due to a high alveolo-arterial pO_2 difference (14.1–6.9 = 7.2 kPa). The physiological veno-arterial shunt fraction was estimated to be 37% (Fig. 4). The shunt fraction was calculated using an arterio-venous oxygen concentration difference of 2.3 mmol/l (the default value used when mixed venous blood was not available for analysis). The oxygen extraction tension was 4.17 kPa, i.e. slightly decreased. Titratable hydrogen ion of the extended extracellular fluid was normal (0.4 mmol/l), see Table 1.

The patient was treated with antibiotics and supplementary oxygen on a face mask with a flow of pure oxygen of 8 l/min (Fig. 5). The patient was lying on his left side with the pneumonic lung as the basal region. The patient's body temperature decreased from 40 to 39.2°C. After 8 h a new arterial sample was taken. A flow of pure oxygen of 8 l/min on a Venturi mask provided a fraction of oxygen in the inspired air of about 0.40 according to tables of the relationship between oxygen flow and fraction of oxygen in the inspired air (T. Waldau, personal communication). The relationship was quite uncertain, however, depending upon the type of face mask.

The arterial pO_2 increased from 6.9 (Fig. 4) to only 8.3 kPa (Fig. 5). The alveolo-arterial pO_2-difference increased markedly, from 7.2 to 23.9 kPa, but the estimated physiological shunt fraction (*F*va) decreased from 0.37 to 0.29 (Table 1). Lung regions are still poorly ventilated or not ventilated at all.

In order to improve the perfusion of the right, healthy lung and increase the ventilation of the left pneumonic lung by dilatation of airways and alveoli, the patient must rest on his right side and not the left.

After 5 days all blood gas values were normal, except the physiological shunt fraction which was still slightly elevated (0.15).

Case 2

A male patient, 58 years old, was brought to the emergency department with dyspnoea and cyanosis. The patient had been smoking 20–40 cigarettes per day

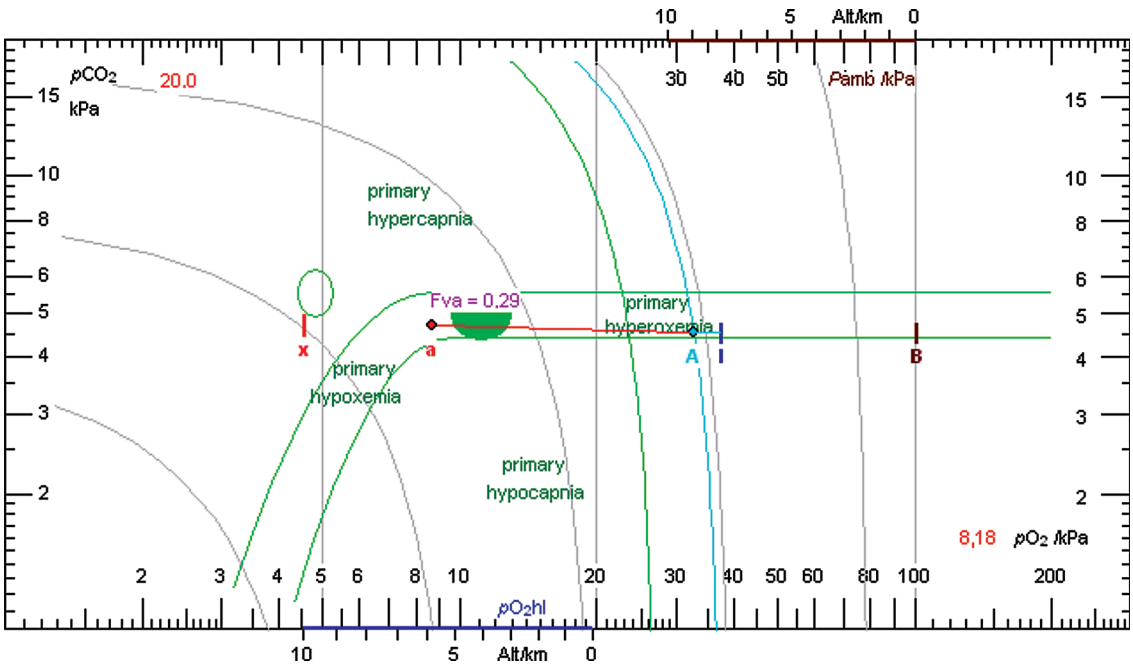

Fig. 5. Case of pneumonia (case 1) treated with antibiotics and supplementary oxygen.

since the age of 15 years. A chest X-ray showed an infiltrate in the right lung suspicious of lung cancer. End expired air pO_2 and pCO_2 were measured (12.1 and 7.0 kPa, respectively) allowing calculation of the CO_2/O_2 exchange ratio R (0.92). Arterial blood gas data showed an oxygen saturation fraction of 0.736 causing the pronounced cyanosis (Table 1).

The blood gas map (Fig. 6) showed the arterial point 'a' close to the area of *primary hypercapnic hypoxia* but with an element of primary hypoxia as well, due to a high physiological shunting. The oxygen extraction tension indicated by the red point 'x' was far below the reference interval, indicating a state of uncompensated hypoxaemia.

After assisted ventilation for 2 days the pCO_2 was within the normal interval, but hypoxaemia persisted although the physiological shunt fraction had decreased from 0.41 to 0.30 (Fig. 7). The oxygen extraction tension indicated by the red mark 'x' was now normal, indicating a state of compensated arterial hypoxaemia. Metabolic acidosis had developed as a complication with titratable hydrogen ion of the extended extracellular fluid of 8.8 mmol/l (Table 1). This could be partly explained by an increased blood lactate of 7.5 mmol/l. The metabolic acidosis gradually disappeared, but the increased physiological shunting and hypoxaemia persisted. The patient died 7 months later with multiple metastases.

Symbols

The respiratory symbols, which conform to the standards published in *Federation Proceedings 9, 602,* 1950, are inconvenient for electronic transfer with their 3 or more levels. We have therefore chosen a modification with up to two levels.

General quantities:

c	concentration of a component in a system
V	volume
P	total pressure
F	substance fraction or volume fraction
pO_2	partial pressure of oxygen for gas mixture
pO_2	*tension* of oxygen for aqueous solution
pCO_2	partial pressure of carbon dioxide for gas mixture
pCO_2	*tension* of carbon dioxide for solution
R	respiratory CO_2/O_2 exchange ratio, respiratory quotient
Q	cardiac output

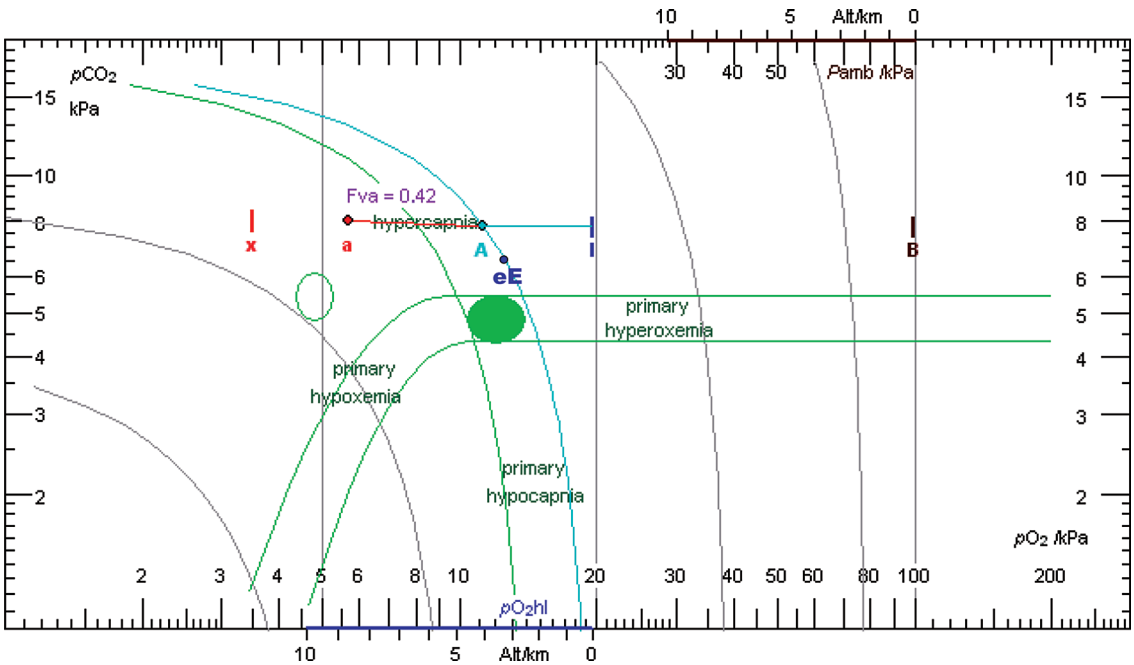

Fig. 6. The logarithmic blood gas map shows blood gas data from a lung patient at admission (case 2). The green curve and a-point indicate mixed primary hypercapnia and primary hypoxaemia.

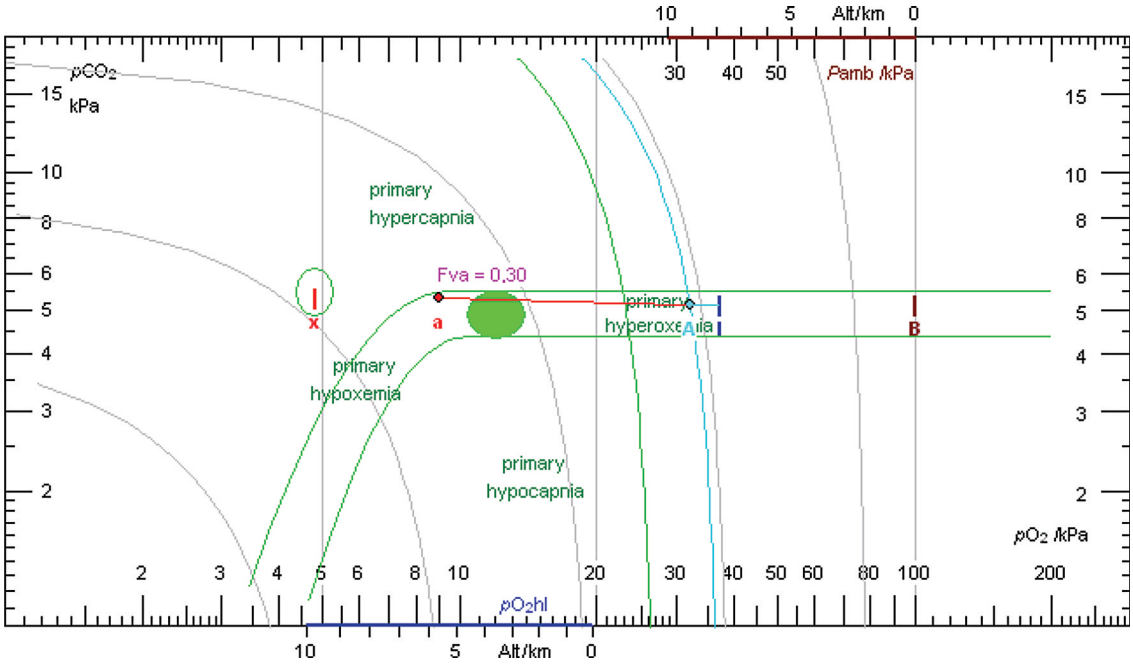

Fig. 7. Case of primary hypercapnic hypoxaemia (case 2) treated with assisted ventilation. After therapy, the patient was normocapnic but still marginally hypoxaemic (point 'a' outside normal area).

V/Q ventilation/perfusion ratio

Symbols for gas phase:

I_{dry} dry inspired air

I humidified inspired air (tracheal inspired air)

E mixed expired air

eE end expired air

A ideal alveolar air from alveoli with the same V/Q as the overall V/Q for both lungs

amb ambient air

Symbols for blood:

a arterial

v mixed venous

x arterial blood after extraction of oxygen (2.3 mmol/l)

Composite symbols

ctH^+Ecf conc. of titratable hydrogen ion in the extended extracellular fluid

FO_2I substance fraction of oxygen in humidified inspired air

pO_2x oxygen extraction tension: pO_2 of the arterial blood after O_2 extraction of 2.3 mmol/l

Fva volume fraction of mixed venous blood in the arterial blood (physiological shunt fraction)

ΔpO_2Aa $pO_2A - pO_2a$

Pamb ambient barometric pressure

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