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Acidosis
base excess
hypoproteinemia
anemia
acid-citrate dextrose
THAM
pH

Abnormal Base Excess Curves

Theoretical Studies with a Digital Computer

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Extract

A mathematical model of the Base Excess curve of blood was developed and then programmed into a digital computer coupled to an automatic plotter. Values for serum albumin, globulin and mean corpuscular hemoglobin concentrations were fed to the computer and theoretical Base Excess curves were generated. Normal values gave a curve which closely approximated the experimentally derived curve of Siggaard-Andersen. When values characterizing hypoproteinemia, hypochromia or tris-(hydroxymethyl)-aminomethane buffering were introduced, Base Excess curves were produced which varied, often markedly, from the traditional curve.

Speculation

Base Excess determinations which employ currently published nomograms or slide rules are subject to significant error when blood is seriously hypoproteinemic, hypochromic, or buffered with tris-(hydroxymethyl)-aminomethane.

Introduction

In 1960, Siggaard-Andersen and Engel [6] introduced a new laboratory determination for evaluating the metabolic component of the acid-base status of the blood. This determination, called the Base Excess, has come to be widely used by clinicians, particularly pediatricians. Base Excess is now defined as the titratable acid or base in a blood or plasma sample determined by titration with strong, nonvolatile acid or base to a normal reference point (pH 7.40; P_{CO_2}, 40 mm Hg at 38°) [9]. The direct determination of Base Excess by titration of blood under the conditions stated is a delicate and time-consuming procedure. Consequently, curve and alignment nomograms [7, 8] and a slide rule [5] have been developed which allow indirect determination of Base Excess by laboratory methods which are easier to perform. These graphic aids are each based on the same data from Siggaard-Andersen's titration experiments on four normal adult blood samples [7].

If a blood sample is normal, determination of Base Excess from a nomogram is satisfactory. On the other hand, if ionic strength, plasma protein concentration, mean corpuscular hemoglobin concentration (MCHC) or the intracellular-extracellular distribution of hydrogen and bicarbonate ions deviates from normal, the Base Excess curve for that blood sample should be distorted [9] and should vary in its shape and scaling at all points except the one at which Base Excess is defined as zero (see above).

Siggaard-Andersen has considered how abnormalities of ionic strength affect the Base Excess curve [7] but curves for hypoproteinemic or hypochromic sam-

ples have never been published. BATTAGLIA and MESCHIA [1] have recently found that blood which has been collected in acid-citrate dextrose (ACD) solution and buffered to physiologic pH with tris-(hydroxymethyl)-aminomethane (THAM), has an abnormal intracellular-extracellular hydrogen ion distribution. Such blood, recently advocated for exchange transfusion [4], should also give rise to abnormal Base Excess curves.

The purpose of this study was to examine, theoretically, the validity of nomogram-determined Base Excess values in the special situations referred to above. Substantial experimental work will have to be done to confirm or refute the findings.

Methods

A mathematical model of the Base Excess curve was developed (see Appendix). By inserting values for the serum albumin and globulin concentrations and the MCHC, the coordinates of any number of points on the Base Excess curve could be calculated. All calculations were performed on an IBM 7044 digital computer. Magnetic tapes, generated by the computer, were used to drive a Calcomp 563 automatic plotter which scaled, labelled and plotted the Base Excess curves on that pH-log P_{CO_2} coordinate system on which all isobicarbonate lines have a slope of −1. The pK′ was assumed to remain constant at 6.100. α, the solubility factor for CO_2 was taken as 0.0301.

Hypothetical data were submitted to the computer for a) normal blood, b) bloods with abnormal serum protein concentrations, c) bloods with abnormal MCHC's and d) blood which had been collected with ACD solution and buffered to physiologic pH with THAM.

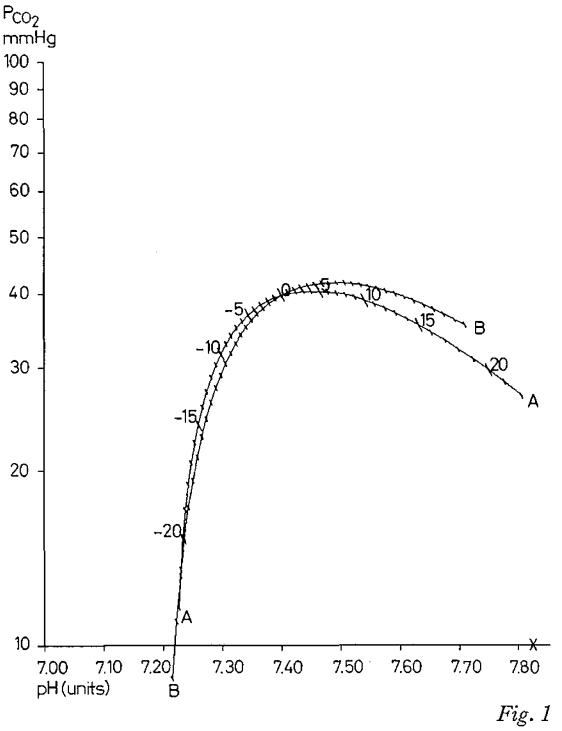


Fig. 1

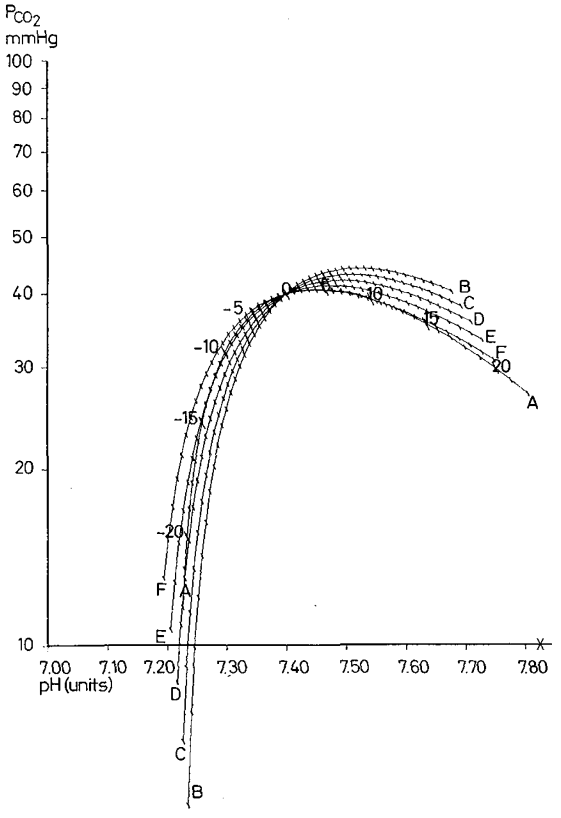


Fig. 2

Fig. 1. The normal Base Excess curve. Curve A is that published by SIGGAARD-ANDERSEN [7]. Curve B is that curve which derives from the model when the serum protein concentrations, MCHC, and intracellular-extracellular hydrogen ion distributions are normal.

Fig. 2. Theoretical Base Excess curves for blood samples of differing serum protein concentrations. Curve A is SIGGAARD-ANDERSEN's [7] and assumes a total serum protein concentration of 7.2 g/100 ml. Curves B, C, D, E and F assume total serum protein concentrations of 1, 4, 7, 10 and 13 g/100 ml, respectively.

Results

The Normal Base Excess Curve

Figure 1 shows the experimentally determined Base Excess curve of SIGGAARD-ANDERSEN [7] superimposed upon the theoretically normal Base Excess curve from the proposed model. Both curves are based on a constant MCHC of 33.4 and a constant total serum protein concentration of 7.2 g/100 ml at an albumin/globulin ratio of 1.6 to 1.

The Effect of Abnormal Serum Protein Concentrations

Figure 2 shows SIGGAARD-ANDERSEN's curve superimposed on a series of theoretical Base Excess curves based on serum protein concentrations of 13, 10, 7, 4 and 1 g/100 ml, each at an albumin/globulin ratio of 1.6 to 1. The MCHC was held constant at 33.4.

The Effect of Abnormal MCHC

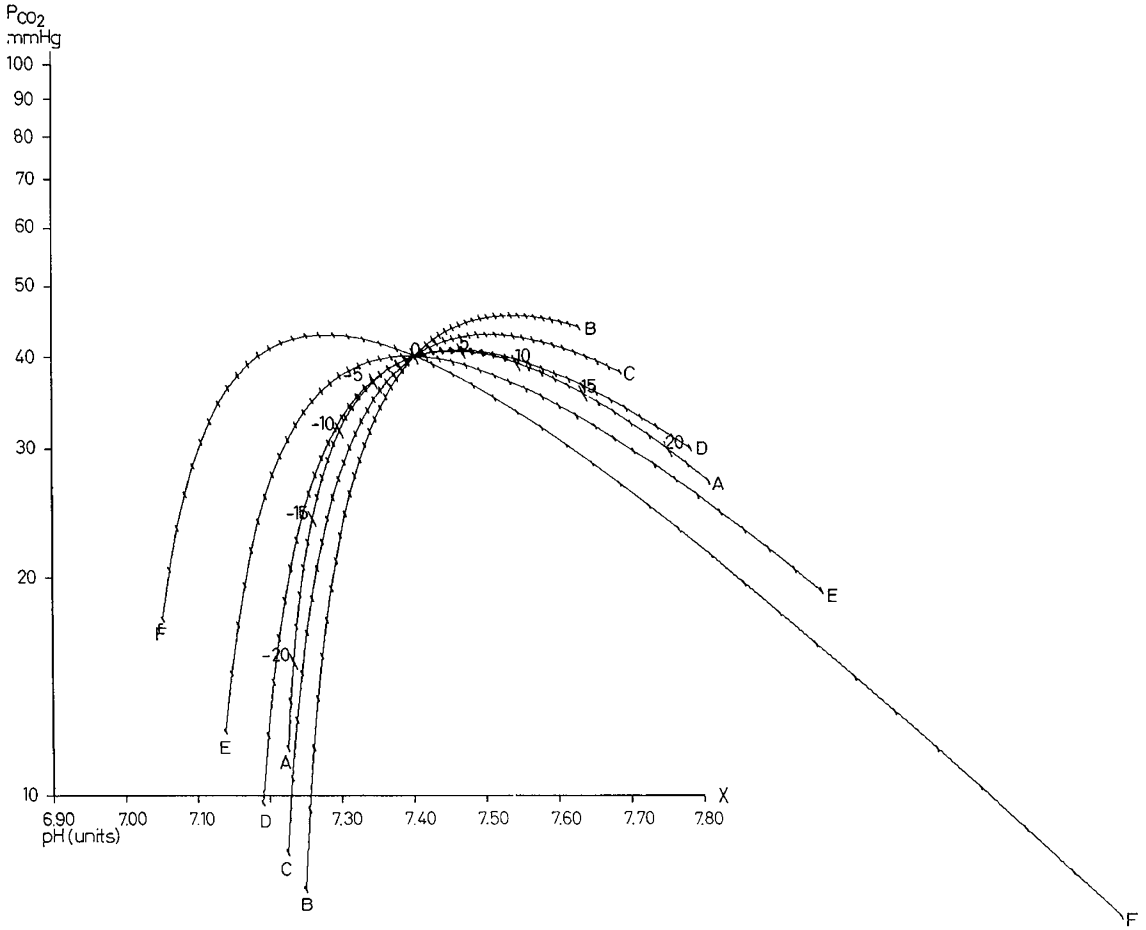
Figure 3 shows SIGGAARD-ANDERSEN's curve superimposed on a series of theoretical Base Excess curves in which the MCHC was allowed to vary from 20 to 40 in increments of 5. The serum protein concentration was held constant at 7.2 g/100 ml at an albumin/globulin ratio of 1.6 to 1.

The Effect of Tris-(hydroxymethyl)-aminomethane

Figure 4 shows SIGGAARD-ANDERSEN's curve superimposed on a theoretical Base Excess curve based on the intracellular-extracellular hydrogen ion distributions which occur when blood is collected in ACD solution and buffered to physiologic pH with THAM. The MCHC was held constant at 33.4 and the serum protein concentration at 7.2 g/100 ml at an albumin/globulin ratio of 1.6 to 1.

Table I gives examples of Base Excess values which might be obtained by using SIGGAARD-ANDERSON's [7] curve with abnormal blood samples.

Fig. 3. Theoretical Base Excess curves for blood samples of differing MCHC. Curve A (SIGGAARD-ANDERSEN's [7]), uses an MCHC of 33.4. Curves B, C, D, E and F assume the MCHC to be 40, 35, 30, 25 and 20, respectively.



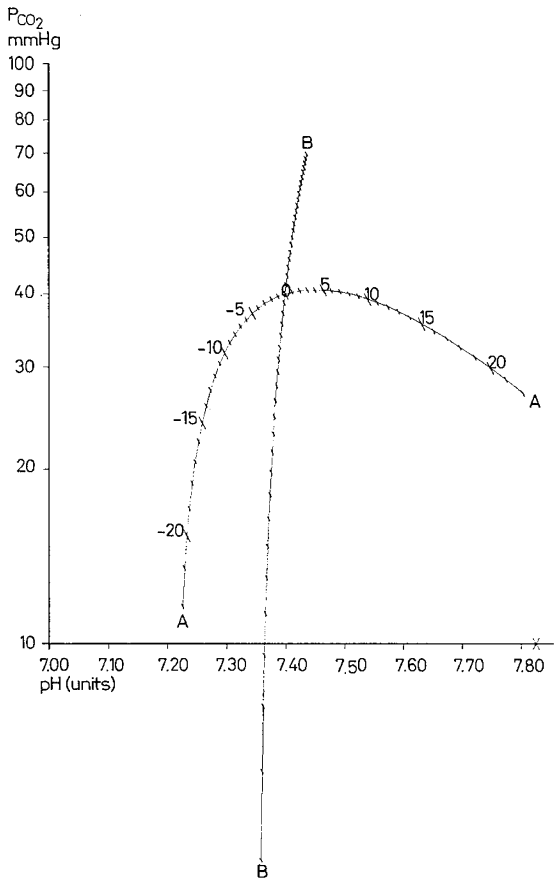


Fig. 4. Theoretical Base Excess curve for a blood sample collected in ACD solution and buffered to physiologic pH with THAM (curve B). Curve A is SIGGAARD-ANDERSEN's [7] curve for normal blood.

Discussion

Nomogram-determined Base Excess values provide a highly satisfactory estimate of titratable acid or base in most circumstances. Hypoproteinemic or hypochromic samples do not occur uncommonly, however, so the physician should be aware that conventional Base Excess determinations may not be as reliable in the presence of such abnormalities.

The purpose of this study was not to detract from the validity of the Base Excess concept but to illustrate that at least three clinical situations may exist in which the Base Excess and the titratable acid or base of a blood sample may not be the same. It should be noted that deviant Base Excess curves become important only at their extreme 'ends'. As long as agreement exists in the current definition of Base Excess, all Base Excess curves must pass through the same point (pH, 7.40; P_{CO_2}, 40 mm Hg, at 38°). Near this point, error is negligible.

Base Excess is usually determined by plotting straight line buffer regression curves from pH determinations at two known P_{CO_2}'s and noting where the line intersects the Base Excess curve. Despite wide dissimilarities

Table I. Base Excess values from SIGGAARD ANDERSEN's [7] curve compared to values from the present model. Straight line CO_2 titration curves (calculated from the $\frac{dHCO_{3p}^-}{dpH_p}$ of equations 26 and 29, Appendix) were first passed through points on appropriate model curves where TB = –20, –15, –10, –5, 0, +5, +10, +15 and +20 mEq/l, and from thence through SIGGAARD ANDERSEN's [7] curve. Values obtained from the latter are given for comparison

Characteristics of model	M.C.H.C. (g/100 ml)c	Hemoglobin (g/100 ml)b	Total protein (g/100 ml)p	Titratable base (mEq/l)								
				–20.0	–15.0	–10.0	–5.0	0.0	5.0	10.0	15.0	20.0
Normal	33.4	15.0	7.2	–21.2	–15.2	–10.1	–5.0	0.0	5.1	10.2	15.3	20.4
Hypoproteinemia	33.4	15.0	1.0	–22.3[1]	–15.9	–10.6	–5.2	0.0	5.3	11.0	16.3	22.0
Hypoprot., anemic	33.4	5.0	1.0	–22.5[1]	–16.2	–10.9	–5.5	0.0	5.7	11.2	17.9	24.3[1]
Hyperproteinemic	33.4	15.0	13.0	–20.1	–14.7	– 9.7	–4.9	0.0	4.9	9.6	14.3	19.0
Hyperprot., anemic	33.4	5.0	13.0	–20.0	–14.5	– 9.7	–4.9	0.0	4.9	9.6	14.4	19.1
Hypochromic	20.0	15.0	7.2	–22.8[1]	–16.9	–11.3	–5.7	0.0	6.2	12.5	19.4	25.0[1]
Hypochromic, anemic	20.0	5.0	7.2	–21.5	–15.8	–10.5	–5.2	0.0	5.5	11.1	16.9	22.9[1]
Hyperchromic	40.0	15.0	7.2	–20.7	–14.8	– 9.8	–4.9	0.0	4.9	9.6	14.4	19.0
Hyperchromic, anemic	40.0	5.0	7.2	–21.1	–15.2	–10.0	–5.0	0.0	5.2	10.4	15.8	21.4
ACD, THAM	33.4	15.0	7.2	–18.3	–12.6	– 8.1	–3.8	0.0	3.7	7.2	10.8	14.4
ACD, THAM, anemic	33.4	5.0	7.2	–20.4	–14.4	– 9.4	–4.6	0.0	4.6	9.1	13.6	18.2

[1] Beyond the limits of SIGGAARD-ANDERSEN's [7] curve. Extrapolated.

between the traditional curve and the abnormal curves depicted here, plots of buffer regression lines may still give values for Base Excess which are reasonably similar, except at the extreme 'ends' of the curve. Regardless of the nomogram employed, the 'ends' of the Base Excess curve are somewhat inaccurate as long as straight line buffer regression curves are employed. The error introduced by using straight lines has been discussed by SIGGAARD-ANDERSEN [9].

Finally, it is important to emphasize that this study was entirely theoretical and was designed only to examine the advisability of using a single nomogram to compute Base Excess from all types of blood samples.

Summary

A mathematical model of the Base Excess curve has been derived and programmed into a digital computer-plotter. When normal values for serum protein concentrations, MCHC and intracellular-extracellular hydrogen ion distribution ratios are fed to the computer, the Base Excess curve produced is similar to the one proposed by SIGGAARD-ANDERSEN for normal blood. When abnormal values are used, uniquely different curves are generated suggesting that the Base Excess curve in common use may be an ill-defined curve for certain blood samples.

Appendix

Derivation of the Base Excess Curve

Titratable Base (TB) is here defined as the concentration (in mEq/l) of excess, strong, nonvolatile acid or base found on titration of a blood or plasma sample to a *plasma* pH of 7.40 at a P_{CO_2} of 40 mm Hg, at 38°, at which point TB is defined as 0. A negative value of TB implies an excess of strong acid; a positive value an excess of strong base. TB, so defined, is identical with SIGGAARD-ANDERSEN's Base Excess [7].

The Base Excess curve is defined as the locus of all points on the pH-bicarbonate diagram of plasma where $TB_c = TB_b = TB_p$ [7] (the subscripts c, b and p refer to red cell fluid, whole blood and plasma, respectively). The distribution of TB between red cell fluid and plasma depends upon the P_{CO_2} [9]. For example, at very high P_{CO_2}'s, a greater quantity of excess, strong, nonvolatile acid is buffered within cells such that $TB_c < TB_b < TB_p$. At very low P_{CO_2}'s the reverse is true so that $TB_c > TB_b > TB_p$. At an intermediate point, therefore, $TB_c = TB_b = TB_p$ and that point lies on the Base Excess curve. Accordingly, at every point on the Base Excess curve, TB is independent of the volume of red cells in the sample.

When mixtures of bicarbonate ion and nonbicarbonate buffer base are equilibrated with carbon dioxide, bicarbonate is generated or destroyed as indicated by equation (1).

$$H_2CO_3 + NBBB^- \leftrightarrows HCO_3^- + H\text{–}NBBB \qquad (1)$$

where $NBBB^-$ is the concentration of nonbicarbonate buffer base, and HCO_3^- the concentration of bicarbonate ion. In an open system for carbon dioxide, where H_2CO_3 is unaffected by $NBBB^-$, HCO_3^- depends upon $NBBB^-$. Owing to the high concentration of hemoglobin within red cells, $NBBB_c^-$ is much higher than $NBBB_p^-$. A rise in P_{CO_2}, therefore, generates more bicarbonate within cells much of which diffuses into plasma. If the P_{CO_2} falls, the reverse occurs and bicarbonate shifts in the opposite direction. Therefore, the CO_2 titration curve of plasma equilibrated in the presence of red cells is significantly steeper than the curve for plasma equilibrated alone. The point where the two curves intersect represents a point where pH_p, $HCO_{3_p}^-$, $NBBB_p^-$ and, therefore, TB_p are the same in each sample. If it can be shown that the cells involved contained a $TB_c = TB_p$, the point lies on the Base Excess curve. Derivation of the equations for the two CO_2 titration curves which meet these requirements forms the basis of the present model.

The following assumptions are made: 1. The $NBBB_p^-$ derives entirely from albumin and globulin; 2. the $NBBB_c^-$ derives entirely from hemoglobin; 3. all hemoglobin is fully oxygenated; and 4. the temperature is 38°.

The CO_2-Titration Equation of Plasma

Let:

alb = concentration of albumin (g/100 ml)
glob = concentration of globulin (g/100 ml)
ALB^- = concentration of albuminate buffer base (mEq/l)
$GLOB^-$ = concentration of globulinate buffer base (mEq/l)

By performing CO_2-titrations on pure albumin and globulin solutions, VAN SLYKE, HASTINGS, HILLER and SENDROY [10] showed that ALB^- and $GLOB^-$ could be calculated from alb and glob if the pH were known. These relationships are linear and are given as equations (2) and (3).

$$ALB^- = 1.25\ alb\ (pH^- 5.16) \qquad (2)$$
$$GLOB^- = 0.768\ glob\ (pH^- 4.89) \qquad (3)$$

where 5.16 and 4.89 are the isoelectric pH's of albumin and globulin.

It is apparent from equation (1) that a change in P_{CO_2} produces a $\Delta HCO_3^- = -\Delta NBBB^-$. As $NBBB_p^- = ALB_p + GLOB_p^-$ (by assumption), it holds that

$$\Delta HCO_{3_p}^- = -(\Delta ALB_p^- + \Delta GLOB_p^-) \qquad (4)$$

Equations (2) and(3) further indicate that the magnitudes of ΔALB^- and $\Delta GLOB^-$ are determined by the magnitude of the pH change which results from changing the P_{CO_2}. If $HCO_{3_p}^-$ at $pH_p = 7.40$ is known, then HCO_3^- at any other pH_p (designated by x) may be calculated.

$$HCO_{3_p}^-{}_{pH=x} = HCO_{3_p}^-{}_{pH=7.40} + \Delta HCO_{3_p}^-{}_{pH=x} \quad (5)$$

$$HCO_{3_p}^-{}_{pH=x} = (HCO_{3_p}^-{}_{pH=7.40} + 2.80\ alb_p + 1.92\ glob_p) - [1.25\ alb_p\ (pH-5.16) + 0.768\ glob_p\ (pH-4.89)]_{pH=x} \quad (6)$$

When TB = 0 and $pH_p = 7.40$, $P_{CO_2} = 40$ mm Hg; hence $HCO_{3_p}^- = 24.0$ mEq/l (by the Henderson-Hasselbalch equation). Accordingly, if strong nonvolatile acid or base is added to the plasma but the pH_p is held constant at 7.40 by appropriately varying the P_{CO_2}, ALB_p^- and $GLOB_p^-$ are prevented from changing, all of the added strong acid or base must be buffered by $HCO_{3_p}^-$, and $HCO_{3_p}^-$ may be expressed as follows:

$$HCO_{3_p}^-{}_{pH=7.40} = 24.0 + TB \quad (7)$$

Substituting equation (7) into (6) gives (8), the CO_2 titration curve of plasma expressed in terms of TB.

$$HCO_{3_p}^-{}_{pH=x} = 24.0 + TB + 2.80\ alb_p + 1.92\ glob_p - [1.25\ alb_p\ (pH-5.16) + 0.768\ glob_p\ (pH_p-4.89)]_{pH_p=x} \quad (8)$$

The CO_2-Titration Equation of Plasma Equilibrated in the Presence of Red Cells

The CO_2-titration equation for plasma equilibrated in the presence of cells is derived from the equation for plasma alone (given above) and the CO_2-titration equation of red cells. The latter is derived as follows:

Let:

- hgb = concentration of hemoglobin (g/100 ml)
- HGB^- = concentration of oxyhemoglobinate buffer base (mEq/l)
- V_c = volume of packed red cells in a unit volume of blood (l/l)
- V_p = volume of plasma in a unit volume of blood (l/l; $V_p = 1.0 - V_c$)
- $[H^+]$ = molal hydrogen ion activity (nanoEq/l)

DAVENPORT [2] has shown that the CO_2-titration curve of pure oxyhemoglobin is a straight line having a slope of 2.16 hgb. Using VAN SLYKE, WU and MCLEAN's [11] value of 6.6 for the isoelectric pH of oxyhemoglobin, the following equation for converting hgb to HGB^- can, therefore, be written:

$$HGB^- = 2.16\ hgb\ (pH-6.6) \quad (9)$$

In order to obtain the CO_2-titration equation for red cell fluid, hgb_b must be converted to hgb_c. This is accomplished by equation (10).

$$hgb_c = hgb_b/V_c \quad (10)$$

The pH referred to in (9) must be the pH_c and this must be calculated from pH_p.

$$\text{Let: } r = [H^+]_p/[H^+]_c \quad (11)$$

Taking logarithms of both sides of (11) gives (12).

$$\log r = \log [H^+]_p - \log [H^+]_c \quad (12)$$

Adding $\log [H^+]_c - \log r$ to both sides of (12) and multiplying both sides by -1 gives (13).

$$-\log [H^+]_c = -\log [H^+]_p + \log r \quad (13)$$

which can be rewritten as (14).

$$pH_c = pH_p + \log r \quad (14)$$

DILL, EDWARDS and CONSOLAZIO's [3] measurements of pH_p and pH_c allow calculation of values for log r which can then be plotted against pH_p. With negligible error this plot may be regarded as linear between pH_p 7.0 and 7.6 and its equation is given as (15).

$$\log r = 2.02 - 0.301\ pH_p \quad (15)$$

Substituting the righthand sides of equations (10), (14) and (15) into (9) gives (16), which allows calculation of HGB_c^-.

$$HGB_c^- = 2.16 \frac{hgb_b}{V_c} (0.699\ pH_p - 4.58) \quad (16)$$

It is apparent from equation (1) that a change in P_{CO_2} produces a $\Delta HCO_3^- = -\Delta NBBB^-$. As $NBBB_c^- = HGB_c^-$ (by assumption), it holds that

$$\Delta HCO_{3_c}^- = -\Delta HGB_c^-. \quad (17)$$

Equation (16) indicates that the magnitude of ΔHGB_c^- may be determined from the plasma pH change which results from a change in P_{CO_2}. If $HCO_{3_c}^-$ at $pH_p = 7.40$ is known, then $HCO_{3_c}^-$ at any other pH_p (designated by x) may be calculated.

$$HCO_{3_c}^-{}_{pH_p=x} = HCO_{3_c}^-{}_{pH_p=7.40} + \Delta HCO_{3_c}^-{}_{pH_p=x} \quad (18)$$

$$HCO_{3_c}^-{}_{pH_p=x} = \left(HCO_{3_c}^-{}_{pH_p=7.40} + 1.28 \frac{hgb_b}{V_c}\right) - \left[2.16 \frac{hgb_b}{V_c} (0.699\ pH_p - 4.58)\right]_{pH_p=x} \quad (19)$$

DILL, EDWARDS and CONSOLAZIO [3] have shown from paired measurements of $HCO_{3_p}^-$ and $HCO_{3_c}^-$, that $HCO_{3_c}^-$ was related to $HCO_{3_p}^-$ and pH_p. The following

linear equation describes their data with negligible error from pH_p 7.0–7.8

$$HCO^-_{3c} = (3.90–0.442\ pH_p)\ HCO^-_{3p} \quad (20)$$

When TB = 0, $pH_p = 7.40$, and $HCO^-_{3p} = 24$ mEq/l, therefore $HCO^-_{3c} = 15.1$ mEq/l. If strong, nonvolatile acid or base is added to red cells, but by appropriately varying the P_{CO_2}, the pH_c is held constant at the value it would be if the pH_p were 7.40, then HGB^-_c cannot change, all buffering must be done by HCO^-_{3c}, and the following equation may be written:

$$HCO^-_{3c} = 15.1+TB \quad (21)$$
$$pH_p=7.40$$

Substituting the righthand side of (21) into (19) gives (22), the CO_2-titration equation of intact red cells.

$$HCO^-_{3c} = 15.1+TB+1.28\frac{hgb_b}{V_c} - \quad (22)$$
$$pH=x$$

$$[2.16\frac{hgb_b}{V_c}(0.699\ pH_p–4.58)]$$
$$pH=x$$

It is apparent that

$$HCO^-_{3b} = V_pHCO^-_{3p}+V_cHCO^-_{3c} \quad (23)$$

Substituting the righthand sides of (8) and (22) for HCO^-_{3p} and HCO^-_{3c} in (23) gives (24).

$$HCO^-_{3b} = [V_p\ (2.80\ alb_p+1.92\ glob_p+24.0+TB)$$
$$+V_c\ (1.28\frac{hgb_b}{V_c}+15.1+TB)]$$
$$-\{V_p[1.25\,alb_p(pH_p–5.16)+0.768\,glob_p(pH_p–4.89)]+$$
$$V_c[2.16\frac{hgb_b}{V_c}(0.699\ pH_p–4.58)]\} \quad (24)$$

Substituting the righthand side of (20) into (23), factoring HCO^-_{3p} and dividing both sides by $[(3.90–0.442\ pH_p)V_c+V_p]$ gives (25).

$$HCO^-_{3p} = HCO^-_{3b}/[(3.90–0.442\ pH_p)V_c+V_p] \quad (25)$$

Substituting the righthand side of (24) into (25) gives (26), the CO_2-titration equation of plasma, equilibrated in the presence of red cells in which $TB_c = TB_p = TB$.

$$HCO^-_{3p} = [V_p(2.80\ alb_p+1.92\ glob_p+24.0+TB)$$
$$+\ V_c\ (1.28\frac{hgb_p}{V_c}+15.1+TB)]$$
$$-\{V_p[1.25\,alb_p(pH_p–5.16)+0.768\,glob_p(pH_p–4.89)]+$$
$$V_c\,[2.16\frac{hgb_b}{V_c}(0.699\ pH_p–4.58)]\}\ /$$
$$[(3.90–0.442\ pH_p)V_c+V_p] \quad (26)$$

Simultaneous solution of equations (8) and (26) for pH_p and HCO^-_{3p} gives a point lying on the Base Excess curve for any desired value of TB. Repetitive simultaneous solutions over a range of values for TB defines the Base Excess curve. Values for the pH_p –log P_{CO_2} diagram may be computed from the Henderson-Hasselbalch equation.

The Base Excess Curve for ACD, THAM-Treated Blood

BATTAGLIA and MESCHIA [1] collected 31 samples of whole human blood into ACD solution, buffered them to physiologic pH with THAM and measured the pH of intact blood and frozen-thawed red cell hemolysate. Each pair of data allow calculation of values for r and log r. When plotted as functions of pH_p, approximate straight line curves were obtained. The linear, least squares regression equations are as follows:

$$\log r = 0.108\ pH_p–0.869 \quad (27)$$

$$r = 0.212\ pH_p–0.710 \quad (28)$$

Equations (27) and (28) are substituted for (15) and into (20) and derivation of (29) is carried out exactly as for (26).

$$HCO^-_{3p} = [V_p\ (2.80\ alb_p+1.92\ glob_p+24.0+TB)$$
$$+V_c\ (1.58\frac{hgb_b}{V_c}+20.6+TB)]$$
$$-\{V_p\ [1.25\ alb_p(pH_p–5.16)+0.768\ glob_p\ (pH_p–4.89)]$$
$$+V_c\,[2.16\frac{hgb_b}{V_c}(1.11\,pH_p–7.47)]\}\ /$$
$$[(0.212\,pH_p–0.710)V_c+V_p] \quad (29)$$

Repetitive simultaneous solutions of (29) and (8) were performed (as above) to give points on the Base Excess curve for ACD, THAM-treated blood.

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