

## Laboratory Report

# Acid-Base Balance Nomogram—A Boston-Copenhagen Detente

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Quantitation of actual and expected complete compensation for chronic hypercapnia in man is facilitated by addition of data of Brackett *et al.* in the form of an arc to the Siggaard-Andersen alignment nomogram. Percentage of compensation is defined as 100 times the ratio of observed to predicted (fully compensated) base excess at the patient's  $P_{CO_2}$ . Extracellular fluid base excess, defined as  $BE_{EF}$ , is estimated from a 3-g hemoglobin line added to the nomogram. (Key words: Acid-base equilibrium; Measurement techniques, acid-base.)

COMPENSATION of pH, buffer base and  $HCO_3^-$  in chronic hypercapnia may be compared with the "complete" compensation found by Brackett and co-workers<sup>1</sup> in patients documented to be stably hypercapnic by addition of an arc to Siggaard-Andersen's nomogram<sup>2</sup> (fig. 1). A line drawn from the observed or assumed chronically maintained  $P_{CO_2}$  tangent to this arc intersects the pH,  $HCO_3^-$  and base excess lines to yield the predicted values of compensation. Incomplete compensation may be quantitated by the ratio of observed to predicted values. Acute changes of  $P_{CO_2}$  *in vivo* cause shifts of  $HCO_3^-$  between blood and the rest of the extracellular fluid (ECF). For this reason, base excess (BE), when read from the blood hemoglobin line on the grid, changes with  $P_{CO_2}$ . For example, after 15 minutes at  $P_{CO_2} = 80$  torr, with hemoglobin (Hb) = 15 g, blood will have changed from  $BE = 0$  to  $BE = 4$  mEq/l. However, the total ECF of the body

has not lost or gained base. One may estimate the ECF BE, or *in vivo* BE, by reading the nomogram at the approximate Hb concentration of ECF. Both theoretically and empirically, the ECF behaves as if it had about 2-5 g Hb distributed throughout ECF for a wide range of blood Hb concentrations and for both adults and the newborn.<sup>3</sup> For this reason, a 3-g line may be added to the nomogram for estimation of ECF BE, or  $BE_{EF}$ .

For those who wish to construct their own arcs, the arc is tangent to lines drawn through  $P_{CO_2}$  values of 40, 60, 80, 100, and 150 with corresponding values of pH of 7.4, 7.362, 7.323, 7.290, and 7.217, which suggests an arbitrary precision somewhat in excess of the clinical supporting data. For computer solution of Siggaard-Andersen's nomogram, the 3-g Hb line is solved by the expression:

$$BE_{EF} = 37(\exp[(pH - 7.4 + .345Y)] - 1) \quad (.55 - .09Y) - 1$$

When  $BE_{EF}$  and  $P_{CO_2}$  are known, pH may be computed from:

$$pH = 7.4 + (\ln[BE_{EF}/37 + 1])(.55 - .09Y) - .345Y$$

where  $Y = \ln(P_{CO_2}/40)$ .

Completely compensated  $BE_{EF}$ , ( $BE_{EF}$ ), from Brackett *et al.*, is approximated by:

$$BE_{EF} = 15 \cdot \ln(P_{CO_2}/40) + .095(P_{CO_2} - 40)$$

$BE_{EF}$  has the following characteristics as an index of the metabolic component of acid-base balance abnormality:

- 1) It is essentially unaffected by acute  $P_{CO_2}$  alterations.
- 2) It refers to the space into which  $NaHCO_3$  distributes when administered therapeutically.

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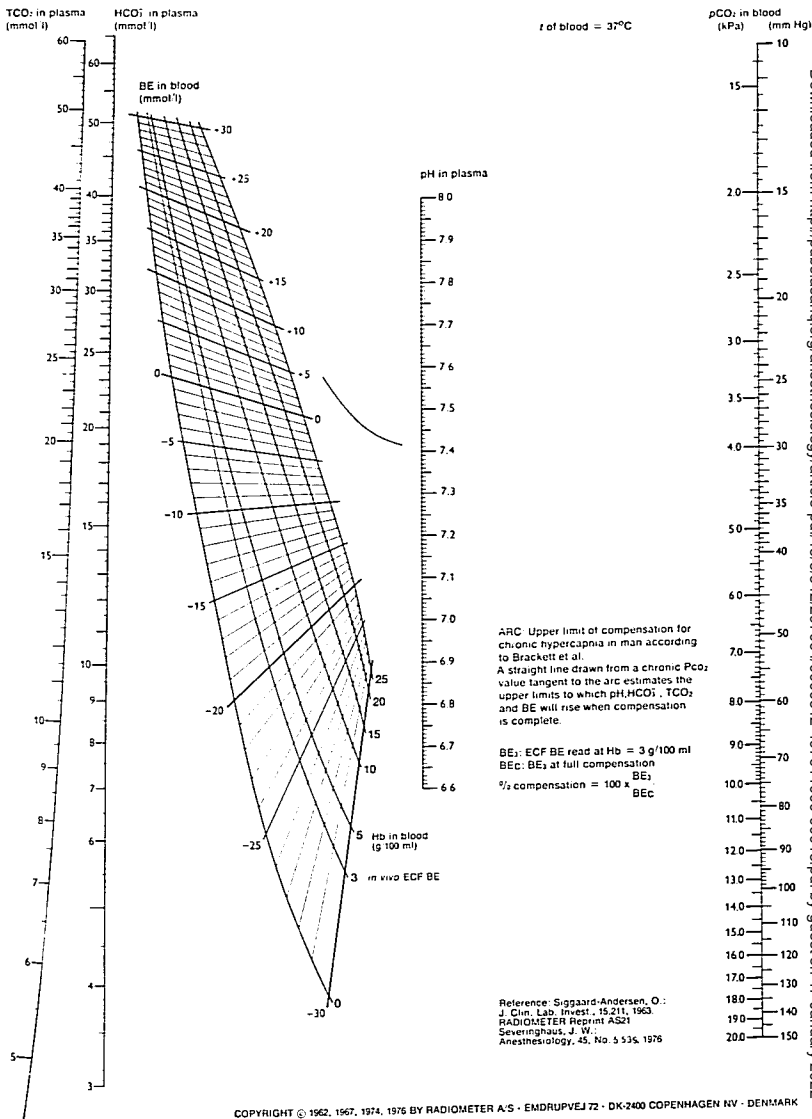


FIG. 1. A 3-g hemoglobin line and an arc have been added to this nomogram to permit estimation of the extent of completion of metabolic alkaline compensation for chronic respiratory acidosis. *Example:* At  $P_{iO_2} = 80$  torr (10.7 kPa), a line drawn through this  $P_{iO_2}$  and tangent to the arc suggests that a patient who maintained this  $P_{iO_2}$  over many weeks or months would be found to have  $pH = 7.323$ ,  $BE_e = BE_c = 14.2$  mmol/l,  $BE_e$  of blood at 15 g Hb = 10.2 mmol/l,  $HCO_3^- = 40.5$  mmol/l, and  $T_{iO_2} = 43.5$  mmol/l. If his  $pH$  were found to be 7.250 at  $P_{iO_2} = 80$  torr,  $BE_e$  reads +7.1 mmol/l and he would have achieved  $100 \times (7.1/14.2) = 50$  percent compensation. (Used with permission from Radiometer.)

- 3) It varies so little with blood Hb that determination of Hb is unnecessary for its quantitation.
- 4) Compensation may be expressed in mEq/l of ECF, independent of Hb, or as a percentage of complete compensation.

Siggaard-Andersen prefers 5 g Hb rather than 3 for the *in-vivo* line. I chose 3 to accommodate infants and anemic subjects; 5 is better at 14–18 g Hb. This nomogram is both more precise and more convenient than buffer diagrams, whether used to compute plasma  $HCO_3^-$ ,  $CO_2$ , or blood ECF  $BE$ . The clinically determined arc adds to the nomogram a missing relevance to patient condition, and permits computation of the compensation present in a patient at his observed  $P_{iO_2}$ , in terms of either  $HCO_3^-$  or  $BE$ . The total-body base excess is usually computed as  $BE_e \times .3 \times$  body weight (kg), the answer being milliequivalents.

The divergences between  $BE$ 's computed at blood Hb from this and prior nomograms, by adherents of the Copenhagen school, and clinical changes in  $HCO_3^-$  in chronic hypercapnia reported by the Boston school led to what Bunker called the "Great Trans-Atlantic Debate" more than a decade ago.<sup>4</sup> This nomographic "treaty" should facilitate detente for anyone still interested in the dispute.

### References

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