REFERENCES

2. Schwartz WB, Relman AS: A critique of the parameters used in the evaluation of acid–base disorders. New Engl

To the Editor:—Dr. Levesque’s letter, and subsequent correspondence between us, have sharpened my focus on the differences between the Boston and Copenhagen approaches to acid–base imbalance analysis. Dr. Levesque asks why I shun the more direct approach of "in-vivo titration." Is it more direct? Schwartz’ critique, which occupied six pages of the New England Journal of Medicine in 1963, did not describe the “in-vivo titration” procedure, since he wrote “It is beyond the scope of this paper to consider in detail how this ‘physiologic approach,’ which

utilizes clinical data and the classic parameters of pH, PaCO₂, and plasma bicarbonate, is applied to the analysis of complex acid–base disturbances.” The relative directness or simplicity of the two methods may best be illustrated in parallel columns, as shown in table 1. Dr. Levesque graciously consented to write the Boston bedside logic.

The difference in interpretation between “Boston” and “Copenhagen” is this:

The metabolic compensation for chronic respiratory acidosis is reported as a base excess by “Copen-

<table>
<thead>
<tr>
<th>Table 1. Two Methods for Quantifying the Non-respiratory Component of Acid–Base Balance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boston</td>
</tr>
<tr>
<td><strong>Index</strong></td>
</tr>
<tr>
<td>Sample</td>
</tr>
<tr>
<td>Measure</td>
</tr>
<tr>
<td>Calculate</td>
</tr>
<tr>
<td>Methods</td>
</tr>
<tr>
<td>Slide rule</td>
</tr>
<tr>
<td>Equation</td>
</tr>
<tr>
<td>or</td>
</tr>
</tbody>
</table>

**Laboratory Logic**

BEₐ estimates the patient's metabolic acidosis or alkalosis, in millimoles per liter of ECF. It represents the amount of H⁺ or OH⁻ needed to titrate each liter of the patient's ECF back to a normal pH of 7.4 at PaCO₂ = 40 torr.

BEₐ estimates BE₂ to be expected with full (renal) compensation (after weeks to months) at this PaCO₂.

Notes:

a) BEₐ is independent of the patient's PaCO₂ at the time of sampling.

b) BEₐ includes both compensatory and primary metabolic acidosis and alkalosis.

c) BE₂ is higher in venous blood by 0.3 mEq/l per g desaturated Hb due to the Haldane effect.

From the patient's history, measured PaCO₂ is substituted in the appropriate equation and [H⁺] obtained is compared with expected [H⁺]. Actual and expected [HCO₃⁻] are calculated using Henderson's relationship. The difference between expected and actual [HCO₃⁻] is a quantification of the true metabolic change. Renal compensation is not of metabolic origin and should not be lumped with HCO₃⁻ deficit or excess. Memorization of equations (a), (b) and (c) and Henderson's relation suffices to solve the problem in the majority of cases. No nomogram or slide rule is necessary.
hagen," but as “pure” chronic respiratory acidosis, and thus no metabolic alkalosis, by “Boston.” For example, when P_{CO_2} is stable at 80 torr, and HCO_3^- is 41.5 mEq/l, this is reported as BE_3 = 14.5 mEq/l in Copenhagen parlance and a zero metabolic alkalosis in Boston.

It seems to me that both are right, and both answers are needed. I believe the laboratory report cannot make clinical judgments, but should provide chemical characterizations. The report should include P_{CO_2}, P_{O_2}, pH, BE_3, and HCO_3^- . Clinical interpretation may then proceed from these data to reason as follows:

1) BE_3 measures total non-respiratory or “metabolic” deviation from normal, whether renal, compensatory or metabolic in origin.

2) ΔHCO_3^- calculated as described by Dr. Levesque herein measures the deviation of this patient’s state from that found in average patients with similar steady states.

3) 100 BE_3/BE_0 estimates percentage compensation to an observed hypercapnia.

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A Safer IV Catheter

To the Editor:—A recent article by Drabinsky¹ describing two cases of operative intervention for removal of an intracardiac “cath-embolus” prompts me to call attention to recent improvements in catheter technology that should make obsolete the problem of cut catheters and resultant catheter emboli. This accident usually occurs during catheterization of the subclavian or internal jugular vein when a catheter is inserted and manipulated through a cutting metal needle. The introduction of a closed catheter system enables the removal and discarding of the cutting metal needle after percutaneous venipuncture and prior to insertion of the preattached long catheter into the vascular system.*

In this design (fig. 1), a metal needle protrudes through a flexible self-sealing rubber tube, which is attached to a short “around-needle” introduction catheter. A long placement catheter is encased in a malleable plastic cover connected to the distal end of the rubber tube. Upon venipuncture, the needle


![Design of the system](image1)

![Insertion of the placement catheter](image2)