Measurements of the metabolic component of the acid–base status in acute clinical situations

C. T. KAPPAGODA, R. J. LINDEN, H. M. SNOW AND J. B. STOKER, Cardiovascular Unit, Department of Physiology, The University, Leeds, 6 August 1973

In an attempt to decide on a clinically acceptable method of assessing the metabolic component of the acid–base status of blood, Lewis & Stoddart [Clinical Science (1973) 44, 297–300] compared two indices of the non-respiratory state of blood in vivo. These were the non-respiratory pH (Kappagoda, Linden & Snow, 1970; Stoker, Kappagoda, Grimshaw & Linden, 1972) and the ‘corrected’ base excess (Prys-Roberts, Kelman & Nunn, 1966). Lewis & Stoddart (1973) were able to demonstrate a correlation between these two indices and concluded that the corrected base excess was the method of choice ‘because it retained the familiar terminology of Astrup, Jorgensen, Siggaard-Andersen & Engel (1960)’.

However, any method which is recommended for clinical use in the assessment of non-respiratory acid–base disturbances must satisfy one important criterion, i.e. the original data upon which the method is based must refer to changes which occur in blood in vivo. This requirement is based upon the finding that the slope of the CO₂ titration curve of blood in vivo differs from that of the CO₂ titration curve in vitro. Thus, concepts such as base excess and buffer anion concentration (Singer & Hastings, 1948), which are dependent on data obtained from blood in vitro, cannot be used to diagnose disturbances which occur in the body. In an attempt to circumvent these objections, Prys-Roberts et al. (1966) attempted to ‘correct’ the base excess values obtained from the conventional Astrup et al. (1960) nomogram using the following expression:

\[
\text{corrected base excess} = \text{measured base excess} - 0.0975 (P_a\text{CO}_2 - 40).
\]

It was their intention to compensate for the difference between the slope of the CO₂ titration curve in vivo and that of the CO₂ titration curve in vitro. This equation, which is based upon results obtained from subjects without a non-respiratory disturbance, implies that the ‘correction factor’ is solely dependent on \(P_a\text{CO}_2\). It has, however, been shown that the slope of the CO₂ titration curve changes with the degree of non-respiratory acidaemia (Kappagoda et al., 1970; Stoker et al., 1972) and therefore the ‘corrections’ suggested by Prys-Roberts et al. (1966) would not be applicable in these situations.

Further, the existence of a correlation merely indicates a relationship between the two parameters considered and does not permit any comment to be made about the relative merits of any one index as suggested by Lewis & Stoddart (1973). For example, we have re-examined our acid–base data obtained from a series of children with congenital heart disease and have found equally significant correlations between the non-respiratory pH and a variety of other indices of the non-respiratory status of the blood, as shown below:

<table>
<thead>
<tr>
<th>Regression equation</th>
<th>( r )</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( y = 64.3x - 476.5 )</td>
<td>0.94</td>
<td>0.001</td>
</tr>
<tr>
<td>( y = 58.8x - 349.4 )</td>
<td>0.92</td>
<td>0.001</td>
</tr>
<tr>
<td>( y = 56.6x - 419.5 )</td>
<td>0.93</td>
<td>0.001</td>
</tr>
</tbody>
</table>

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However, the merits of the index of the non-respiratory status should only be assessed in terms of the fundamental basis of the method. For instance, one could speculate on the possible existence of an equally significant correlation between non-respiratory pH and the respiratory rate and depth and observe that it would be illogical to assess a non-respiratory acid–base disturbance from the tidal volume.

The use of the corrected base excess, while being conceptually erroneous, also seeks to perpetuate the idea that it is possible to predict net deficiencies of base in the whole body. Base excess refers to a concentration and the only way in which this concentration could be translated into a net amount is by determining the total buffer store. Such information is obviously not available in the usual clinical situation. On the other hand, the concept of a non-respiratory pH, which is defined as the pH that would occur in blood if the patient’s arterial $P_{CO_2}$ is altered *in vivo* to 40 mmHg, permits the titration of the patient with alkali to a predetermined non-respiratory pH value. Also implicit in its use is the concept that it is impossible to predict the base deficit in any one patient because acid–base disorders are clinical situations where precise information about buffer stores is not available.

Lastly, as teachers, it seems to us that to retain a system ‘because it retained the familiar terminology of Astrup et al. (1960)’ as suggested by Lewis & Stoddart (1973), even though it is based on erroneous concepts and is demonstrably inadequate, will only increase the difficulties of students in this difficult field, whether undergraduate or postgraduate.

References


Authors’ Reply

D. G. Lewis and J. C. Stoddart, Department of Anaesthesia, Royal Victoria Infirmary, Newcastle-upon-Tyne

We wish to thank Dr Kappagoda and his colleagues for their comments on our recent paper (Lewis & Stoddart, 1973).

We accept their view that the method of choice for the clinical assessment of non-respiratory acid–base status should refer to changes occurring in blood *in vivo*. Ideally this assessment should be based on original data obtained from *in vivo* $CO_2$ titration curves. Accurate data are available in patients with acute non-respiratory acidaemia (Stoker, Kappagoda, Grimshaw & Linden, 1972) and without a non-respiratory disturbance; however, no such data exist for patients with chronic hypercapnia (Flenley, Franklin & Miller, 1970; Goldstein, Gennari &