Table 1. Effect of Heparin Dilution on Arterial Blood Samples

<table>
<thead>
<tr>
<th></th>
<th>Sample 1</th>
<th>Sample 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Venous</td>
<td>Arterial</td>
</tr>
<tr>
<td>Hematocrit, %</td>
<td>41</td>
<td>27</td>
</tr>
<tr>
<td>Cell vol</td>
<td>41</td>
<td>41</td>
</tr>
<tr>
<td>Plasma vol</td>
<td>59</td>
<td>59</td>
</tr>
<tr>
<td>Heparin vol</td>
<td>52</td>
<td>27</td>
</tr>
<tr>
<td>Total vol</td>
<td>152</td>
<td>127</td>
</tr>
</tbody>
</table>

Dilution factor (F)    | 1.52     | 1.27     |
\[HCO_3^-\], mmol/L    | 24.3     | 17       |
\[HCO_3^-\]^\text{\textsuperscript{1}}\textsuperscript{\text{*}}, mmol/L (F) | 24.3 | 21.6     |
\[TCO_2\] \text{, mmol/L} | 25       | 24       |
PCO\(_2\), mmHg         | 22       | 24       |
Pco\(_2\), mmHg (F)     | 33       | 30       |
Pco\(_2\) × 0.031, mmol/L | 1.0 | 0.9      |
\[TCO_2\] \text{– dissolved CO}_2, mmol/L | 24.0   | 23.1     |
True \[HCO_3^-\], mmol/L | 24.0 | 23.1     | 21.6     |

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The authors of the Letter respond:

We thank Mr. Turton for his Letter and his agreement with our observations. In the interests of brevity we omitted from our original Letter the detailed exposition of the basic clinical chemistry involved, believing it to be common knowledge. The well-known small difference between TCO2 and calculated bicarbonate seemed to us insignificant in relation to the demonstrated large discrepancy between the ASTRA TCO2 value and the calculated bicarbonate in the heparin-diluted blood samples.

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Table 1. Plasma Ammonia Values (\(\mu g/dL\)) at Various Postnatal Ages

<table>
<thead>
<tr>
<th>Day</th>
<th>3</th>
<th>28</th>
<th>56</th>
<th>84</th>
<th>111</th>
<th>158</th>
<th>Adults</th>
</tr>
</thead>
<tbody>
<tr>
<td>±1</td>
<td>±1</td>
<td>±1</td>
<td>±1</td>
<td>±2</td>
<td>±2</td>
<td>±2</td>
<td>±2</td>
</tr>
<tr>
<td>n</td>
<td>28</td>
<td>20</td>
<td>20</td>
<td>21</td>
<td>20</td>
<td>28</td>
<td></td>
</tr>
</tbody>
</table>
x    | 84    | 61    | 58    | 58    | 56    | 63    |
| SD  | 21    | 14    | 17    | 17    | 10    | 18    |

Table 2. Effect of Feeding On Plasma Ammonia Concentrations

Hours after feeding | <1 | >1 <2 | >2 <3 | >3 <4 |
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>31</td>
<td>30</td>
<td>20</td>
<td>15</td>
</tr>
</tbody>
</table>
x \(\mu g/dL\)      | 55 | 57    | 63    | 67    |
| SD, \(\mu g/dL\)   | 18 | 15    | 15    |

through adapter (Model 95-10; Orion Research, Inc., Cambridge, MA 02139). We measured plasma ammonia as previously described (I) in a group of normal-term, non-fasted infants who were exclusively breast- or bottle-fed. Table 1 summarizes the results and compares them with those obtained for a group of adults who had fasted overnight.

Plasma ammonia concentration is significantly greater (\(p < 0.05\)) at two to four days than in the older infant, suggesting rapid maturation of hepatic and urea-cycle activity. Because plasma ammonia values did not change significantly after 28 days of age, we pooled these results. They were normally distributed (as analyzed with the goodness-of-fit test and chi-square distribution (2)); 70% of the values were within one standard deviation of the mean and 97% within 2 SD. Mean plasma ammonia was 580 (SD 140) \(\mu g/L\), the range being 280-980 \(\mu g/L\).

We also evaluated the effect of feeding on plasma ammonia. The infants studied were 28 to 196 days old, and almost all were receiving formula or human milk. Table 2 summarizes the results. We saw no significant change in plasma ammonia within 3 h of feeding. Thereafter, it increased (\(p < 0.05\)), probably reflecting protein absorption from the gastrointestinal tract. Whether this increase is clinically significant in the evaluation of hepatic function is not clear, particularly since these values do not differ significantly from those obtained for fasting adults. We conclude that plasma ammonia can be validly determined in the non-fasting infant if the sample is collected within 3 h of feeding.

References

2. Remington RD, Schork MA. Statistics with Applications to the Biological and

Reference


To the Editor:

We wish to report reference values for ammonia in the plasma of infants, as measured with an ammonium ion-selective electrode probe with flow-
Lidocaine Is Stable In Serum and Blood

To the Editor:

Lidocaine is a frequently administered anti-arrhythmic drug, particularly useful in emergency situations because of its rapid onset of action (1). Therapeutic drug monitoring of lidocaine is recommended because of its potent central nervous system toxicity. Moreover, lidocaine has been implicated in numerous accidental and homicidal deaths (2). Thus its chemical stability in patients' specimens and in quality-control specimens needs to be known. Two metabolites of lidocaine, monoethylglycinexylidide and glycineylidide, are unstable in plasma and urine (3), but no work has been published on the stability of lidocaine itself. Therefore, we have assessed the stability of lidocaine in serum and blood.

Sera from patients being treated with lidocaine were pooled and stored at -20 °C until used. Cadaver blood containing lidocaine was also collected and pooled. After an initial quantification by gas chromatography (4), the serum and blood were each divided into two portions. One was stored at 25 °C, the other at 4 °C. At various time intervals, aliquots of fluid stored at each temperature were removed and re-assayed. After 70 days, at least 80% of the lidocaine was detected in serum at either temperature. Furthermore, no changes in blood lidocaine concentration were detected over a similar time period at either temperature. Any changes in lidocaine concentration were within the out-of-run CV of the method.

We conclude that lidocaine is quite stable in serum and blood over a two-to-three-month period.

References


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Evaluation of the Bromcresol Purple Method for Albumin as Used with the acá

To the Editor:

Bromcresol green (BCG) dye-binding methods are widely used to quantify albumin, even though numerous reports indicate that BCG lacks specificity (1, 2). Bromcresol green reacts within seconds with albumin, but within minutes it also reacts with acute-phase reactants; gamma-globulins do not interfere (3). Results of BCG methods in which this phenomenon is taken into account (4) correlate better with those by immunochemical and electrophoresis methods. At higher temperatures the slower reaction makes the assay even less specific for albumin (1), and this has caused additional problems for its use in the Du Pont acá discrete analyzer, which operates at 37 °C with a reaction time of 261.5 s.

In an attempt to eliminate this problem, Du Pont recently adapted the bromcresol purple (BCP) dye-binding method for albumin to the acá. This method, first described by Louderback et al. (5), reportedly is more specific for albumin (6, 7).

Using specimens from 31 normal laboratory personnel, we compared results by these two acá procedures and also the BCG procedure as used in the Technicon SMA-II continuous-flow analyzer. We also compared 10 additional specimens with above-normal alpha-globulins as shown by electrophoresis on cellulose acetate. Total protein was determined with the biuret reaction, in the SMA-II. Both the acá and the SMA-II were standardized according to the manufacturers' recommendations. Our results for the 31 normal persons were:

<table>
<thead>
<tr>
<th>Method</th>
<th>Mean albumin, g/L</th>
<th>Normal range, g/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>acá-BCG</td>
<td>48 (3)</td>
<td>42–54</td>
</tr>
<tr>
<td>acá-BCP</td>
<td>43 (4)</td>
<td>35–61</td>
</tr>
<tr>
<td>SMA-BCG</td>
<td>41 (3)</td>
<td>35–47</td>
</tr>
</tbody>
</table>

The acá-BCG reaction gave consistently higher values, and our normal range was higher than the 38–48 g/L range given by the manufacturer. The means and normal ranges for the acá-BCP and SMA-II-BCG methods were more similar. The main differences between the SMA-II and acá-BCG methods are that in the SMA-II the reaction is at room temperature and a succinate buffer system as recommended by Dumas and Biggs (8) is used rather than the acetate buffer used in the acá. Both endpoints are read after about 4.5 min, while in the new BCP method the reaction time is 29 s, similar to that in rapid BCG methods (4). (The BCP dye is added in Breaker/Mixer II on the acá, while BCG was added at Breaker/Mixer I.)

For the group of 10 patients the acá-BCG method again consistently gave the highest values for albumin, with a mean of 38 (SD 6) g/L. The mean by the BCP method and electrophoresis was 30 g/L; the SMA-II-BCG method gave intermediate results, with a mean of 33 g/L. However, there was considerable intersubject variation in results by these methods. In six of these patients, the BCP results averaged 4 g/L higher than the electrophoresis results; for the other four the BCP method gave a lower value than electrophoresis (mean difference, 4 g/L). For these same four patients, the SMA-II-BCG method agreed well with electrophoresis in three (mean difference, 1 g/L), who also had the highest albumin/globulin ratios (as determined by electrophoresis) in the abnormal group. In the remaining patients, all with albumin/globulin ratios &lt;0.8, the SMA-BCG method consistently yielded higher albumin values than did electrophoresis (mean difference, 6 g/L).

We conclude that the acá-BCP method is a significant improvement over the acá-BCG method. Both normal and abnormal populations give lower albumin values with the BCP method, probably because the dye is most specific for albumin, although the shorter reaction time cannot be excluded. Of the three methods, results by the BCP method agreed best with electrophoresis in patients with acute-phase reactants. However, even these few data