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Erroneous Electrolyte Results Caused by Catheters

To the Editor:

We have discovered serious analytical errors in sodium and potassium measurements, of a magnitude that could jeopardize patient care, when specimens collected through certain catheters are analyzed with ion-selective electrodes.

In 1985, Eastman Kodak Company (Rochester, NY 14650) issued a bulletin to its customers, providing notice that cationic surfactants, such as benzalkonium salts, cause a false elevation in sodium measurement with the Ektachem systems. These same compounds also falsely increase potassium measurements with electrodes in which vanilminoc is used as an ion exchanger. Since virtually all potassium electrodes are of this type, most ion-selective analytical systems will exhibit this error. Even microgram per liter concentrations of benzalkonium salts cause clinically significant errors. The Ektachem vanilminoc potassium electrode, however, does not yield erroneous results; perhaps this is related to the dry, multilayered reagent construction of the Ektachem system.

Benzalkonium salts are generally known to be used as topical antiseptics. However, despite the absence of such topical preparations in our hospital, we have observed this interference repeatedly over the past year. We have now discovered a major new source for this contaminant.

Investigating these errors, we found that specimens drawn from newly inserted indwelling catheter lines (American Edwards Laboratories, Anasco, PR 00610) showed grossly erroneous results for sodium when tested with an Ektachem system, and for potassium when tested with an Astra-8 Analyzer (Beckman Instruments, Inc., Brea, CA 92621). For one patient, results immediately and 2 h after insertion of a catheter were: Astra-8: Na = 142, 141; K = 5.8, 4.4; Ektachem: Na = 186, 156; K = 4.0, 3.6 mmol/L.

We demonstrated these interferences in vitro by analyzing 5-mL portions of a control serum drawn through a new catheter. We found (Table 1) that 15-20 mL of serum was required to clear the interference from the line.

We have been informed by the manufacturer that American Edwards Laboratories treats all catheter products with benzalkonium heparin. We do not know whether other catheter manufacturers follow a similar practice.

Those using ion-selective electrodes for sodium and potassium measurement should investigate the catheters used in their institutions. Flushing catheters containing benzalkonium heparin must greatly exceed the usual nursing practice, which in our institution constitutes a 5-mL flush. Alternatively, laboratories could use flame photometry to validate all above-normal results for sodium and potassium before reporting results to medical personnel.

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Table 1. Effect of Catheter on Electrolyte Results

<table>
<thead>
<tr>
<th>5-mL portion no.</th>
<th>Astro-8</th>
<th>Ektachem</th>
<th>Flame</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Na</td>
<td>K</td>
<td>Na</td>
</tr>
<tr>
<td></td>
<td>Measured concn, mmol/L</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>136</td>
<td>7.2</td>
<td>165</td>
</tr>
<tr>
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<td>143</td>
</tr>
<tr>
<td>5</td>
<td>137</td>
<td>4.8</td>
<td>139</td>
</tr>
<tr>
<td>6</td>
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Citrice Acid and Calcium Nephrolithiasis

To the Editor:

In their recent article, Cowley et al. (1) suggest a mechanism that might explain hyperoxaluria in kidney-stone patients on the basis of malabsorption of citrate, ascorbate, and possibly other hydroxycarboxylic acids. In particular, they found evidence of depressed intestinal citrate absorption in these patients after an oral loading test when results for six recurrent stone formers and five healthy subjects were compared. However, in our opinion, some points deserve further comments.

Firstly, on the basis of the results in their Figure 1, it seems more appropriate to conclude that, in formers of calcium stones, only the initial rate of citrate absorption is lower than that observed in control subjects. No statistically significant differences are in fact reported as far as the other time points are concerned, particularly at 30 min, the time at which maximal citrate absorption is generally expected (2).

Secondly, we have some reservations about the preliminary conclusions reached by the authors. In fact, if malabsorption of citrate contributes to the pathogenesis of stone formation, we would expect the mean concentrations of citrate in serum from kidney-stone patients to be significantly lower than that of control subjects. It would be therefore interesting to know the concentrations of citrate in the serum of patients studied by the authors. However, from published results (2,3) these mean citrate concentrations in kidney-stone patients appear to be within the normal range or even higher than those of control subjects.

We have recently studied (ms. in preparation) citrate metabolism in 45 stone formers and 39 control subjects. All patients had normal radio-opaque stones demonstrable on abdominal roentgenograms. None of them had ingested any drugs for at least a month before the investigation, none had active urinary tract infection, and none showed evidence of renal tubular acidosis or primary hyperparathyroidism. Interestingly, we found significantly decreased mean (± SD) excretion of citrate in urine of kidney-stone patients (2.5 ± 1.5 vs 3.5 ± 1.0 mmol/24 h, P <0.001), whose mean concentrations of citrate in serum were the same as those of the control subjects (0.10 ± 0.03 mmol/L vs 0.11 ± 0.03, difference not significant). Furthermore, mean (± SD) tubular reabsorption of citrate in these patients (86.2% ± 7.7%) was sig-