interference with analysis for theophylline by high-performance liquid chromatography. 


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Stability of Glucose in Urine

To the Editor:

Before commencing a study of urinary glucose output in diabetics, in which 24-h collections were to be taken at home and stored for periods of up to two weeks before delivery to the laboratory, we were unable to find definite information on the long-term stability of urinary glucose. We therefore examined the stability of glucose in urine over a period of six weeks.

To prevent bacterial growth, we ordinarily add 5 mL of "Hibitane" (chlorhexidine gluconate, 200 g/L solution; ICI Ltd., Macclesfield, Cheshire, U.K.) to each 24-h collection bottle, but for this investigation we added 50 μL of it to 10 mL of freshly voided urine. The urine was stored for six weeks at 4 °C or at room temperature (23–25 °C). Glucose in the urine was measured by the hexokinase method (Glucose Kit A-1100; Roche Products Ltd., Welwyn Garden City, U.K.). Chlorhexidine, at the concentration used, has no effect on the estimation of glucose by the hexokinase procedure (1). The results (urinary glucose, mmol/L, in five samples) are shown in the following tabulation:

<table>
<thead>
<tr>
<th>Fresh</th>
<th>After six weeks</th>
<th>After six weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>at 4 °C</td>
<td>23–25 °C</td>
<td>23–25 °C</td>
</tr>
<tr>
<td>136</td>
<td>140</td>
<td>134</td>
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<tr>
<td>125</td>
<td>125</td>
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<td>82</td>
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<tr>
<td>70</td>
<td>70</td>
<td>69</td>
</tr>
<tr>
<td>13</td>
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</tr>
</tbody>
</table>

Clearly, the glucose in urine so preserved is remarkably stable; thus collections made at home need not be stored in a refrigerator before transmission to the laboratory.

Reference


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Probable Deficiencies in the DuPont aca Method for CK-MB

To the Editor:

Our studies suggest probable deficiencies with the aca (E.I. Du Pont de Nemours & Co., Wilmington, DE 19898) pack method for the MB isoenzyme of creatine kinase (CK-MB; EC 2.7.3.2). The problem involves at least one pack lot, B9225A.

Eighteen patients’ samples run in a DuPont aca III and compared with results by agarose gel electrophoresis (Corning Scientific Instruments, Medfield, MA 02052) gave a correlation coefficient (r) of 0.70 for CK-MB. For total CK and aca CK-MB, r was 0.93. By comparison, the correlation coefficient between total CK and electrophotetic CK-MB was only 0.47, as one might expect. This suggests that the aca CK-MB determination is a reflection of total CK rather than CK-MB.

Additional findings with the DuPont aca CK-MB pack method indicate that the presence of CK-BB in patients’ samples causes false-positive results.

DuPont is aware of these problems and has notified users.

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Avoiding Xanthine Interference in Urate Determination

To the Editor:

Hande et al. (1) commented on xanthine interference in the uricase method for uric acid determination.

Apparent plasma urate will decrease in the presence of increased amounts of xanthine if insufficient uricase is present to compensate for the inhibitory effect of xanthine. We confirmed this with a uricase-based method ("Urica-quant," cat. no. 124753; Boehringer Mannheim GmbH, Mannheim, F.R.G.) in which urate may be estimated either by kinetic or endpoint means.

On analysis of a pooled plasma containing added xanthine, the apparent decrease in urate occurred only with the kinetic method of estimation and was overcome by adding twice the prescribed amount of uricase to the working solution.

Therefore, an alternative method for estimation of urate in individuals with suspected or known hyperxanthinemia is not necessary. However, manufacturers and users of uricase-based methods should ensure that sufficient enzyme is present to compensate for such circumstances.

Reference


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Persistent Increase of Serum Lactate Dehydrogenase Activity Related to Enzyme-IgG (Lambda) Immunoglobulin Complex

To the Editor:

Several papers have been published on the occurrence of complexes of lactate dehydrogenase (LD; L-lactate: NAD+ oxidoreductase, EC 1.1.1.27) and immunoglobulins in human serum (1). The complexes described so far involve LD and IgA or LD and IgG. Here I describe a patient with rheumatoid arthritis in whom an interaction between LD and circulating IgG (lambda) caused a persistent increase of serum LD activity and an atypical isoenzyme pattern.

Mrs. A.M., a 79-year-old woman with a long history of rheumatoid arthritis, was admitted for investigation of anemia. Laboratory investigations led to the diagnosis of iron-deficiency anemia. However, her serum LD activity was high (1.5- to threefold the upper limit of normal), and had persisted so for more than one year.

LD isoenzymes were separated by immunoelectrophoresis with a Corning ACI system (Corning Medical, Medfield, MA 02052) and stained for LD activity. Gel filtration was carried out on a Sephadex G200 column (42 × 2.5 cm) with 0.1 mol/L tris(hydroxymethyl)-methylamine·HCl buffer, pH 8.0, containing 8.5 g of sodium chloride and 2.0 g of sodium azide per liter, to determine the molecular size of the complex.

The LD isoenzyme pattern showed no