n = 20) was 4.4% (0.79 ± 0.035 μmol/L) and 4.7% (1.1 ± 0.051 μmol/L); the between-day CV (n = 18) was 6.7% (0.87 ± 0.058 μmol/L) and 7.5% (1.25 ± 0.093 μmol/L). Linearity was apparent between 0.15 and 10 μmol/L (r = 0.99). The recovery was 101% ± 10%.

Because serum ubiquinone-10 values in our sample pediatric population did not follow a gaussian distribution (assessed by the Kolmogorov–Smirnov test), we calculated the median and the 2.5 and 97.5 percentiles for reference values. The ubiquinone concentration was independent of sex (P > 0.05, Mann–Whitney test) but decreased significantly with age (r = –0.383, n = 102, P < 0.0001 for ubiquinone in μmol/L; r = –0.384, n = 102, P < 0.00005 for ubiquinone in μmol/mol cholesterol), whereas cholesterol values did not change with age. Ubiquinone concentrations correlated with cholesterol concentrations (r = 0.485, P < 0.00001, Spearman test). After applying statistical analysis to all age groups (Kruskal–Wallis), we established two groups whose ubiquinone concentrations (in μmol/L and μmol/mol cholesterol) were the most significantly different from one another (Mann–Whitney, P < 0.005): 1 month–7 years [n = 62; median, 0.8 μmol/L (interval, 0.46–1.38 μmol/L) and 203 μmol/mol cholesterol (interval, 137–341 μmol/mol cholesterol)], and 8–18 years [n = 40; median, 0.57 μmol/L (interval, 0.34–1.03 μmol/L) and 169 μmol/mol cholesterol (interval, 111–248 μmol/mol cholesterol)].

HPLC with UV detection is a rapid and useful procedure for the quantification of total ubiquinone and correlates well with other procedures (5). We established reference intervals for serum ubiquinone in a Mediterranean pediatric population, showing a decrease of ubiquinone related to age, as was also reported by other authors in several tissues (1). Dietetic habits may influence serum ubiquinone-10 concentrations (6). Therefore, little difference in serum ubiquinone-10 values would be expected between several populations. In our experience, both ubiquinone-10 and ubiquinone-9 are present in substantial amounts in serum (data not shown). Ubiquinone-9 is not synthesized by humans; however, small amounts have been found in serum by some authors (7) but not by others (2). Interestingly, vegetable oils are the richest sources of ubiquinone-9 (6), an important food in Mediterranean area. Consequently, ubiquinone-9 is not suitable for use as internal calibrator to assay serum ubiquinone-10, at least in our population; we, therefore, chose ubiquinone-7.

Serum ubiquinone results are usually reported as molar concentrations but rarely are related to cholesterol (8). The reference intervals thus obtained are different, and the ubiquinone-to-cholesterol ratio yields a more adjusted reference interval. Moreover, ubiquinone-10 in serum protects lipoproteins from peroxidation. Therefore, the relationship between ubiquinone and cholesterol concentrations seems the best means of assessing its biological function in blood.

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References

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Positive Interference of Icodextrin Metabolites in Some Enzymatic Glucose Methods

To the Editor:

The glucose polymer icodextrin has become widely used in continuous ambulatory peritoneal dialysis (CAPD) (1). Icodextrin is hydrolyzed in the systemic circulation to oligosaccharides such as maltose, maltotriose, and maltotetraose. The use of icodextrin leads to substantial concentrations of these icodextrin metabolites in the blood, where they are not normally found (2). The presence of these metabolites could have an effect on enzymatic glucose measurement. A common complication in diabetes mellitus patients is renal insufficiency, which may lead to dialysis. Patients with diabetes mellitus treated by icodextrin-CAPD are at risk for having erroneous blood glucose measurements.

To evaluate the possibility of interference of the icodextrin metabolites in various glucose assays, glucose was measured in 2–11 sets of four heparinized blood samples (800 μL); each set was obtained from a single specimen. These samples were supplemented with 10 μL of 9 g/L NaCl solution as a blank sample or 10 μL of a solution of maltose, maltotriose, or maltotetraose (all sugars from Sigma Chemical Co.), achieving final concentrations for the icodextrin me-
tabolites of 1.2, 1.2, and 0.6 g/L, respectively. These icodextrin metabolite concentrations were reported to be the maximum reachable concentrations in vivo (2). From each sample, 100 μL was centrifuged and analyzed on a Hitachi 911 with a plasma glucose dehydrogenase (GDH) method (Boehringer Mannheim). The whole blood samples were analyzed using an EML 105 with a glucose oxidase (GOD) membrane method (Radiometer), a Chiron 865 with a GOD membrane method accompanied by a glucose reference electrode, and bedside glucose analyzers: Accutrend Sensor with a bioamperometric GDH method (Boehringer Mannheim), Glucocard Memory with a bioamperometric GOD method (Menarini Diagnostics), Glucotouch with a colorimetric GOD method (Lifescan, Johnson & Johnson), One Touch Profile with a colorimetric GOD method (Lifescan), One Touch II with a colorimetric GOD method (Lifescan), and Precision with a GOD method (Medsense). The results of the samples containing icodextrin metabolites were compared with the blank sample and expressed as a mean difference. Interference was defined as a mean difference >0.5 mmol/L (Table 1).

<table>
<thead>
<tr>
<th>Analyzer</th>
<th>n</th>
<th>Mean</th>
<th>Range</th>
<th>Maltose, 1.2 g/L</th>
<th>Maltotriose, 1.2 g/L</th>
<th>Maltotetraose, 0.6 g/L</th>
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<tr>
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<td>EML 105</td>
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<td>Hitachi 911</td>
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<td>3.2–13.0</td>
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<tr>
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<td>3.3–10.3</td>
<td>3.4b</td>
<td>2.0b</td>
<td>0.7b</td>
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<tr>
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<td>0.2</td>
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<td>2.7–9.5</td>
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<td>One Touch II</td>
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<td>0.5</td>
<td>0.5</td>
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</tr>
</tbody>
</table>

*a Number of samples tested.

*b Interference was defined as a mean difference >0.5 mmol/L.

c n = 1; second sample read "LO".

In conclusion, the icodextrin metabolites may cause erroneously high glucose results, depending on the analysis system used. The clinically significant effect of interference by icodextrin metabolites is the potential risk of missing the diagnosis of hypoglycemia. Moreover, in diabetic patients undergoing icodextrin-CAPD therapy, the observed icodextrin metabolite interference may lead to unexplainable fluctuations in measured glucose.

References

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Influence of Uremic Toxins and Nonesterified Fatty Acids on Drug and Thyroid Hormone Binding in Serum

To the Editor:

The findings of Takamura et al. (1) suggest that the furan fatty acid 3-carboxy-4-methyl-5-propyl-2-furan propionate (CMPF) is the major uremic toxin that inhibits albumin binding of the drug furosemide in chronic renal failure. These authors also invoke a potentially important cascade mechanism that can increase the unbound concentration of furosemide as a result of increased occupancy of