concentrations of approximately 1 g/L of urine, which are commonly achieved 2 to 3 h after intravenous infusion, a false-positive result for Bradshaw's test will occur.

We wish to alert clinical biochemists and physicians to this source of interference, and stress the necessity of confirming the validity of all screening tests for Bence Jones protein by measuring total urinary protein and using specific electrophoretic and immunochromatographic tests to detect light chains in fresh concentrated urine.

References

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Questioning the Presence of Pregnancy-Specific β1-Glycoprotein (or Immunologically Similar Material) in Cerebrospinal Fluid

To the Editor:

Pregnancy-specific β1-glycoprotein (PSG1), a high-molecular-mass glycoprotein that normally is synthesized by the syncytiotrophoblasts during pregnancy, is increased in serum not only in pregnancy but also in patients with trophoblastic and, less frequently, with non-trophoblastic malignancies (1, 2). If it is absent (<0.3 μg/L) from the sera of non-pregnant, healthy female and male subjects (2), but recently the presence of PSG1 in cerebrospinal fluids of men and women was reported (2). The range of concentrations, as determined by radioimmunoassay (detection limit 1 μg/L), was 1.9 to 3.6 μg/L (3). A correlation with other factors or with any specific cerebral diseases could not be established.

We studied PSG1 in 15 cerebrospinal fluid specimens, from children and adult patients with cerebral tumors and inflammatory and ischemic cerebral diseases. We used a solid-phase enzyme-linked immunoassay (Enzymnost-SP1; Behringwerke AG, Marburg, F.R.G.), for which the detection limit is 0.6 μg/L (2). In no case did we detect immunoreactive material in undiluted specimens. The absorbances at 492 nm ranged from 0.020 to 0.040, no different from that of blank incubations (0.020 to 0.035). Similar negative results were also obtained for cerebrospinal fluids concentrated fourfold by membrane filtration. We conclude that, in contradiction to the previous report (3), immunoreactive PSG1 is either absent from cerebrospinal fluid or is present in concentrations definitely below 0.15 μg/L. Because the sensitivity of the enzyme immunoassay (0.6 μg/L) is at least equal to or greater than that reported for the radioimmunoassay (1 μg/L), insensitivity of the applied method cannot be the reason for our failure to confirm the presence of immunoreactive PSG1 in human cerebrospinal fluid.

References

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Sodium Drift in the Beckman Astra: A Response

To the Editor:

The Letter of Seddon et al. (Clin. Chem. 29: 212, 1983) deserves comment in the interest of all Astra users. We agree with the authors that an "upward drift of 10 to 20 mmol/L in results for sodium" is not acceptable. We disagree, however, that an ionic wash solution provides an adequate fix for the problem. The fact that a sodium hypochlorite rinse (not a recommended procedure) will "temporarily solve this drift" suggests some type of contamination problem in the sample-handling system of the particular instrument involved.

Ongoing proficiency-survey programs, such as the College of American Pathologists' Comprehensive Chemistry Survey, exhibit typical standard deviations of inter-laboratory Astra sodium results in the area of 1.5 mmol/L. A typical drift of 10 to 20 mmol/L would surely be reflected in such proficiency testing.

The introduction into the sample-handling system of sodium, potassium, chloride, and calcium for an instrument designed to analyze these constituents may be somewhat risky. Sodium drift of the magnitude reported is not normal, and it is suggested that the instrument in question be given a closer look. Illegal grounding, salt bridges, and static charges are possible offenders in a situation like this.

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Urine Urea Nitrogen as Measured in the Beckman Astra-8

To the Editor:

In the Beckman Astra discrete analyzer, ion-selective electrode technology is used. The urea nitrogen method in this system was designed for use at conductivities normally found for plasma. When plasma samples contain high, out-of-range concentrations of urea nitrogen, the samples routinely are diluted with physiological saline before assay.

Because the salt concentration of urea varies widely, the question of diluting urines with either physiological saline or water before assay for urea nitrogen becomes an important issue with the Astra.

Blumenfeld and Griffith (1) examined extensively the Astra's performance with urine Na⁺, K⁺, Cl⁻, urea nitrogen, glucose, and creatinine, but they did not comment on the preferred diluent to use with a particular category of assay.

We have compared urine urea nitrogen as measured with the Astra-8 and the American Monitor KDA over a wide range of concentrations with isotonic saline as the diluent. In the KDA, the enzymatic urease procedure is used for urea nitrogen analyses. Thirty patients' samples, analyzed by both methods, gave mean values that compared favorably (KDA = 87.39 mmol/L vs Astra = 83.92 mmol/L).

In our laboratory, we routinely dilute urine urea nitrogen samples with physiological saline to minimize the inter-patient variability in urinary salt concentrations. With this approach, we have not experienced the analytical
fluctuations in urine urea nitrogen reported by other Astra users with this method.

Reference


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Evaluation of a Kit to Measure HDL Cholesterol (HDL-C) in Serum

To the Editor:

A clinic laboratory got low values for serum HDL-C on using a kit supplied by Electro-Nucleonics, Inc. In the kit, the precipitating reagents (phosphotungstate and MgSO4) are supplied as solids in a tube, to which 0.5 mL of serum is added according to the manufacturer's package insert.

We analyzed split human serum samples in parallel with such kits and using our in-house phosphotungstate method, a procedure like that of Lopes-Virella et al. (1), except that we adjust the pH of the phosphotungstate reagent to 7.4 or slightly less (2), and add only 80 mL of phosphotungstate plus 20 mL of MgCl2 reagents to 1.0 mL of serum. Values obtained with the kit were from 30 to 170 mg/dL lower than results with the in-house method, differences averaging 18.2% (SD 5.4%, n = 16) for values ranging from 280 to 1120 mg/dL. Such differences would cause many samples with total cholesterol/HDL-C ratios near 5.3, based on the in-house method, to have ratios near 6.5 with the kit. These higher ratios would imply an importantly greater risk of coronary artery disease (3).

We further evaluated the kit by titrating (4) three human serum samples with increasing ratios of precipitant to serum. To a series of kit tubes (each contained the prepackaged pre-

<table>
<thead>
<tr>
<th>Tube no.</th>
<th>Serum vol, mL</th>
<th>Apparent HDL-C, mg/L</th>
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<tbody>
<tr>
<td>1</td>
<td>1.8</td>
<td>470</td>
</tr>
<tr>
<td>2</td>
<td>1.5</td>
<td>480</td>
</tr>
<tr>
<td>3</td>
<td>1.3</td>
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</tr>
<tr>
<td>4</td>
<td>1.2</td>
<td>380</td>
</tr>
<tr>
<td>5</td>
<td>1.1</td>
<td>400</td>
</tr>
<tr>
<td>6</td>
<td>1.0</td>
<td>390</td>
</tr>
<tr>
<td>7</td>
<td>0.9</td>
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</tr>
<tr>
<td>8</td>
<td>0.8</td>
<td>380</td>
</tr>
<tr>
<td>9</td>
<td>0.7</td>
<td>380</td>
</tr>
<tr>
<td>10</td>
<td>0.5</td>
<td>350</td>
</tr>
</tbody>
</table>

Because agarose gel electrophoresis showed no LDL or VLDL in the supernates of tubes 3–10, the "true" HDL-C concentration appears to be 390 mg/dL (tubes 3–6). The HDL-C concentration measured by our in-house phosphotungstate method was 390 mg/dL, while the concentration measured with 0.5 mL of serum added to the kit tube was 350 mg/dL. Similarly, in the other two titrations also, the HDL-C values were low when 0.5 mL of serum was added to the kit tube. With 1.0 mL per tube, however, agreement was satisfactory.

The clinic laboratory then split a group of serum samples, measured the HDL-C in one aliquot with use of 1.0 mL of serum per kit tube, and sent us the other aliquot for analysis by our in-house method. All values measured at the clinic with the kit were 10 to 40 mg/dL lower than our values, which ranged from 260 to 630 mg/dL. The differences between the methods averaged 5.8% (SD 3.2%, n = 8). Although these differences were statistically significant, they were deemed acceptable because they were small and consistent. Compared with the earlier differences when 0.5 mL per tube was used, the improvement is obvious. Data from the other titrations suggest that a serum volume slightly less than 1.0 mL (e.g., 0.85 mL) would be optimal, but the gain in accuracy is apt to be offset by the difficulty in measuring such a volume.

The above findings were communicated to the kit manufacturer, Electro-Nucleonics, Inc. Their response pointed out that the package insert in the kit makes no claims about the total cholesterol/HDL-C ratio. Further, they pointed out the recognized facts that the results of HDL-C assays depend on conditions and reagents and that there were differences in both of these when our phosphotungstate reagents and method were compared with their kit and the instructions in the package insert.

References


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Cefoxitin Interference with Urinary 17-Hydroxycorticosteroid Determination

To the Editor:

Because of the marked minute-to-minute variation in plasma cortisol concentrations, either free cortisol in urine or total 17-hydroxycorticosteroids (17-OHCS) excretion is the best index to steroid output. The normal response to dexamethasone has been better established for urinary 17-OHCS than for free cortisol (1); thus, accurate determination of urinary 17-OHCS is essential.

With the large increase in the number of pharmacological agents in recent

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1 Use of products from named companies does not imply that the products are superior to similar products of comparable grade from other companies, nor does it constitute endorsement of the products by the United States Air Force.