fore, we studied the effect of captopril on prolidase activity in plasma.

Using a previously described method (2), we showed that plasma prolidase activity in vitro was slightly decreased (residual activity: 85%) in the presence of 3 mmol of captopril per liter, a concentration about 500-fold larger than the C_{max} determined in pharmacokinetic studies (4). In patients receiving a single high oral dose of captopril (1 mg/kg body wt), prolidase activity was not significantly different before and 2 h after drug administration (n = 31, paired t-test). Similarly, prolonged treatment with captopril (50–100 mg/day, in two equal daily doses) for six months or more did not change the activity of this enzyme in plasma (controls: n = 46, mean 770, SD 195 U/L; patients: n = 13, mean 720, SD 155 U/L).

We also studied the in vitro effect of other angiotensin-converting enzyme inhibitors. The following tabulation shows that these drugs were not or less inhibitory than captopril:

<table>
<thead>
<tr>
<th>Inhibitor, 3 mmol/L</th>
<th>Manufacturer</th>
<th>Residual prolidase acty (% in plasma)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>Squibb</td>
<td>85</td>
</tr>
<tr>
<td>Enalapril</td>
<td>Merck, Sharp &amp; Dothe−</td>
<td>99</td>
</tr>
<tr>
<td>Perindopril</td>
<td>Servier</td>
<td>98</td>
</tr>
<tr>
<td>Ramipril*</td>
<td>Hoechst</td>
<td>93</td>
</tr>
<tr>
<td>Ramiprilate*</td>
<td>Hoechst</td>
<td>99</td>
</tr>
</tbody>
</table>

*Active metabolite.
Each value represents the median of three determinations.

We conclude that plasma prolidase activity is not altered in patients treated with captopril and other angiotensin-converting enzyme inhibitors.

References

Errors in Blood Glucose Determinations in Hemolysates by the Glucose Dehydrogenase Method on the Cobas Mira Analyzer, E. Roaabo (Dept. of Clin. Chem., Central Hospital, Holbeek, 4300 Denmark)

Measuring blood glucose in hemolysates by using glucose dehydrogenase is quite popular, owing to its rapidity and ease of automation (1). This method is easily adapted to the Cobas Mira analyzer (Hoffmann-La Roche, Switzerland). However, we encountered erroneous results for five specimens from patients with high leukocyte counts.

We used a kit from E. Marck, Darmstadt, F.R.G. (cat. no. 13886; Gluc-DH*), based on the method of Banauch et al. (2).

Procedure: To sample capillary blood, we used 20-μL heparinized micro pipets (Hounischen Laboratoriedutyr, Risikov, Denmark). The samples were put into polypropylene cups (Sarstedt, no. 72.690 FF), containing 1000 μL hemolyzing solution and analyzed according to the Cobas Mira program.

For the five specimens with leukocyte counts ranging from 67 to 270 × 10^9/L, the double determinations differed considerably and deviated highly from the values for plasma. Re-sampling gave still other results, as did re-assaying the same hemolysate.

When we examined the hemolysates, we found them to be unusually viscous, apparently due to gel formation, and in the cuvettes there was insufficient mixing of the hemolysate with the Gluc-DH reagent. Various hemolyzing solutions, included one from Roche, gave variable results, displaying atypical reaction curves.

With regard to the hemolyzing solution from Merck (1, 3), this phenomenon seems to be caused by the high concentration of lyzed leukocytes in the hemolysates, in the presence of a high concentration of sodium chloride (1.0 mol/L). Thus, by decreasing the concentration of sodium chloride in the hemolyzing solution to 0.5 mol/L, but keeping the concentrations of phosphate at 0.05 mol/L, EDTA at 2.8 mmol/L, and, as detergent, Triton X-100 at 2.0 g/L, expected values are obtained for glucose, with at least one-week stability, stored at 4 °C.

Although errors in glucose determinations are seldom encountered until cell counts exceed about 50 × 10^9/L, the chance for misleading results is present with the commercial reagents mentioned above.

References

Stimulating Effects of Calcium and Magnesium on Serum Pseudocholinesterase Activity, Kunio Kobayashi, Takafumi Sakoguchi, and Akira Matsuoka (Dept. of Clin. Pathol. and Clin. Labs., Hyogo College of Medicine, 1-1, Mukogawa-cho, Nishinomiya 663, Japan)

Serum pseudocholinesterase (pchE, EC 3.1.1.8) activity is important in the diagnosis of hepatic diseases, although the biological role of the enzyme is not understood. The activity can be reversibly or irreversibly inhibited by eserine, histidine, quaternary ammonium bases, organic phosphorus compounds, and fatty acids.

Recently, we reported on the effects of heavy metals and alkaline earths on the pchE activity (Jpn J Clin Chem 1986;15:304–9). It was strongly inhibited by Cu^{2+}, Ca^{2+}, and Sr^{2+}, which are physiologically distributed in low concentrations (10^{-8}–10^{-9} mmol/L) in the body, but was stimulated by Ca and Mg, which are present in a relatively high concentration (10^{-3} mmol/L) in body fluids and tissues. The pchE activity in clinical serum samples containing