No hyperglycemia after pyridostigmine administration

To the Editor: Francesconi and his colleagues (1) reported hyperglycemia in rats following administration of pyridostigmine. We have examined this issue in human volunteers. Nine volunteers received seven doses of pyridostigmine bromide tablets, 30 mg every 8 h.

Blood samples were drawn for glucose and cholinesterase analyses before and 2 h after the first, fourth, and seventh dose of pyridostigmine (3 sample pairs at hours 0 and 2, 24 and 26, 48 and 50). Glucose was measured by the O₂ electrode method (Beckman glucose analyzer), and cholinesterase activity was measured in whole blood using the radiometric methods as described by Johnson and Ressel (2). All subjects fasted for 8 h before the first sample until the second sample for each pair of samples. Results of glucose levels and cholinesterase inhibition are given in Table 1. No hyperglycemic effect was found after the first or repeated doses of pyridostigmine, and no correlation was found between cholinesterase inhibition and blood glucose levels.

Although cholinesterase inhibition levels were lower than those reported by Francesconi and his colleagues in rats, we were unable to demonstrate any significant pyridostigmine-induced hyperglycemia in humans receiving pyridostigmine bromide, 30 mg in single or repeated doses.

TABLE 1. Blood glucose level and cholinesterase inhibition before and after oral pyridostigmine

<table>
<thead>
<tr>
<th>Dose</th>
<th>Before Pyridostigmine</th>
<th>After Pyridostigmine</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Glucose, mg/100 ml</td>
<td>CHE inhibition, %</td>
</tr>
<tr>
<td>1st</td>
<td>84.1±5.4</td>
<td>0</td>
</tr>
<tr>
<td>4th</td>
<td>86.7±3.4</td>
<td>18.53±4.37</td>
</tr>
<tr>
<td>7th</td>
<td>80.8±7.9</td>
<td>16.9±3.65</td>
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</tbody>
</table>

Values are means ± SD obtained before and 2 h after oral administration of pyridostigmine bromide, 30 mg. CHE, cholinesterase.

REFERENCES


REPLY

To the Editor: Whereas we administered a dosage of nearly 2 mg/kg of pyridostigmine bromide by intraperitoneal administration to rats over 1.5 h, Glickson et al. administered ~3 mg/kg (assuming 70 kg body wt) orally to human volunteers over 48 h. We believe that the relative chronicity of their dosage, the routes of administration, the species difference, and the variation in cholinesterase inhibition could easily account for the difference in results.

In fact, we have recently completed experiments in rats wherein pyridostigmine (dissolved in the drinking water) was administered over 7 or 14 days and have also found no hyperglycemic responses when cholinesterase levels were more moderately inhibited than in our first experiments.

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