ABSTAINING FROM CIGARETTE SMOKING HAS NO MAJOR EFFECT ON GASTRIC EMPTYING IN HABITUAL SMOKERS

O. U. PETRING, B. ADELHØJ, M. IBSEN, J. BRYNNUM AND H. E. POULSEN

The effect of cigarette smoking on the rate of gastric emptying is controversial. Grimes and Goddard (1978) found that cigarette smoking accelerated the rate at which the liquid component of a test meal left the stomach. In contrast, Harrison and Ippoliti (1979) showed that smoking cigarettes with a high nicotine concentration delayed the gastric emptying of solid food in some subjects, and Ohzeki (1981) also reported that smoking produced a marked delay in emptying. We have investigated the effect of abstaining from cigarettes on the rate of gastric emptying in habitual smokers, using the rate of paracetamol absorption as an indicator of the rate of gastric emptying (Clements et al., 1978; Heading et al., 1973).

SUBJECTS AND METHODS

Seven healthy habitual smokers, aged between 25 and 35 yr, with a daily consumption of 15–25 cigarettes, were studied on two occasions separated by at least 2 weeks. The study was approved by the local Ethics Committee. On the first occasion the subjects were investigated after a night during which they did not take any fluid, food or tobacco. On the second occasion they smoked two cigarettes with a nicotine content 2.0 mg per cigarette within 30 min of the beginning of the study. Each investigation was started by the ingestion of paracetamol 20 mg kg⁻¹ dissolved in 200 ml of water and fasting was continued until the end of the study. Samples of venous blood were obtained from an indwelling cannula before, and at 10, 20, 30, 40, 50, 60, 75, 90 and 120 min after, the administration of the paracetamol. Serum was separated and stored at −20 °C until the measurement of serum paracetamol concentration by high pressure liquid chromatography (Knox and Jurands, 1978).

Statistics

Data were compared using Student’s t test for paired data. P values less than 0.05 were considered statistically significant.

RESULTS

When the subjects did not smoke, the time to reach peak paracetamol concentrations was 27 ± 3.6 min (mean ± SEM of seven values) (table I). Peak serum paracetamol concentration was 20.1 ± 1.8 μg ml⁻¹ and the area under the plasma concentration–time curve from 0 to 120 min was 1465 ± 74.5 μg min⁻¹ ml⁻¹. After smoking, the time to reach peak paracetamol concentrations was

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TABLE I. Time to the maximum concentration (t_{max}), the maximum concentration (C_{max}) of paracetamol (20 mg kg/body weight) and the area under the curve from 0 to 120 min (AUC) in seven healthy volunteers abstaining from smoking, and smoking

<table>
<thead>
<tr>
<th>Subject</th>
<th>t_{max} (min)</th>
<th>C_{max} (µg ml^{-1})</th>
<th>AUC (µg min^{-1} ml^{-1})</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No smoking</td>
<td>Smoking</td>
<td>No smoking</td>
</tr>
<tr>
<td>1</td>
<td>40</td>
<td>20</td>
<td>14.89</td>
</tr>
<tr>
<td>2</td>
<td>40</td>
<td>20</td>
<td>16.04</td>
</tr>
<tr>
<td>3</td>
<td>30</td>
<td>10</td>
<td>22.49</td>
</tr>
<tr>
<td>4</td>
<td>20</td>
<td>75</td>
<td>21.07</td>
</tr>
<tr>
<td>5</td>
<td>20</td>
<td>60</td>
<td>16.53</td>
</tr>
<tr>
<td>6</td>
<td>20</td>
<td>50</td>
<td>20.54</td>
</tr>
<tr>
<td>7</td>
<td>20</td>
<td>40</td>
<td>29.01</td>
</tr>
<tr>
<td>Mean ± SEM</td>
<td>27 ± 3.6</td>
<td>39 ± 9.0</td>
<td>20.1 ± 1.8</td>
</tr>
<tr>
<td>P</td>
<td>= 0.35</td>
<td></td>
<td>= 0.46</td>
</tr>
</tbody>
</table>

39.9 ± 9.0 min, and the peak serum paracetamol concentration was 18.1 ± 6.5 µg ml^{-1}. The area under the plasma concentration–time curve was 1089 ± 186 µg min^{-1} ml^{-1}.

None of these values was significantly different (P > 0.05). The respective 95% confidence limits for the difference between the two occasions were: time to peak paracetamol concentration −66 to 91 min; peak paracetamol concentrations −17.7 to 13.9 µg ml^{-1}.

**DISCUSSION**

In the present study paracetamol absorption was used as an indicator of the rate of gastric emptying, a method which has been shown to correlate well with other methods used to estimate gastric emptying (Clements et al., 1978). The data indicate that the liquid phase gastric emptying was not significantly changed by abstaining from cigarette smoking, the 95% confidence limits indicating that, at most, cigarette smoking could delay or accelerate liquid phase gastric emptying by about 1 h.

However, gastric emptying was delayed in four, uninfluenced and one and accelerated in two subjects. These different results may reflect the individual variation of gastric emptying, produced by abstaining from smoking.

Cigarette smoking has been shown either to increase (Piper and Raine, 1959) or to have no effect on basal gastric secretion (Cooper and Knight, 1956), and stimulation by pentagastrin has shown similar divergent results (Debas et al., 1971; Wilkinson and Johnson, 1971). Cigarette smoking decreases the gastro–oesophageal pressure and may allow reflux with accompanying risk of pulmonary aspiration, but only within 8 min after the end of smoking (Stanciu and Bennett, 1972; Chattopadhyay, Greaney and Irvin, 1977). Harrison and Ippoliti (1979) showed that smoking cigarettes with a high nicotine content stimulates duodenal–gastric reflux and, therefore, increases gastric volume, but a recent study has shown no effect of smoking before premedication (Adelhøj et al., 1985). Consequently, abstaining from smoking 2 cigarettes during the fasting period does not decrease the risk of pulmonary aspiration during or after anaesthesia.

**REFERENCES**


Knox, J. H., and Jurand, J. (1978). Determination of


