Effects of histamine acid phosphate and pentagastrin on gastric secretion in normal human subjects

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SUMMARY Six normal subjects were tested with various doses of histamine acid phosphate (1, 10, 40, and 80 μg/kg/hr) and pentagastrin (0.15, 1.5, 6.0, and 12.0 μg/kg/hr), the different doses of the stimulants being administered by separate continuous intravenous infusions. In each sample of gastric juice, which was collected at 15-minute intervals, we estimated the concentrations of the H⁺, Na⁺, K⁺, and Cl⁻ and the concentration of pepsin.

The drugs elicited equal maximal outputs of acid and pepsin. Pentagastrin was more potent than histamine in stimulating acid and pepsin secretion, and the rate of the responses was faster with pentagastrin than with histamine. Apart from this, however, the patterns of secretion of the various constituents of the gastric juice and the interrelationships between the concentrations of the electrolytes were identical with the two drugs. We therefore concluded that the actions of histamine acid phosphate and pentagastrin on human gastric secretion were identical.

Several studies in man have shown that the synthetic pentapeptide pentagastrin (I.C.I. 50, 123), is as effective a stimulant of acid gastric secretion as is histamine (Aagaard and Schmidt, 1967; Konturek, 1967; Konturek and Lankosz, 1967; Multicentre Pilot Study, 1967; Wormsley, Mahoney, and Key, 1967). Makhlof, McManus, and Card (1964a and b) found that gastrin II was more effective than histamine in stimulating human gastric acid secretion, but Johnston, Jepson, Lari, Gumpert, Fawcett, Duthie, and Wormsley (1968) found that gastrin, pentagastrin, and histamine resulted in equal maximal outputs of acid.

Although not free of side effects, pentagastrin is less unpleasant than histamine when administered to human subjects, and hence, since the two stimuli elicit equal maximal outputs of acid, pentagastrin may replace histamine in clinical practice.

In the study now reported, various doses of histamine acid phosphate and pentagastrin were given to a small group of normal subjects, the aim of the investigation being to define in detail the effects of these drugs on human gastric secretion.

Methods

TESTS OF SECRETION
The study was performed on six normal volunteers, five male and one female. Their ages ranged from 21 to 54 years, and the mean age of the group was 33.5 years. All were normal healthy subjects and none had a history of dyspepsia or of other abdominal diseases.

Histamine acid phosphate and pentagastrin were each administered, by continuous intravenous infusion, on four occasions, to each subject. The doses were 1, 10, 40, and 80 μg/kg/hr of histamine and 0.15, 1.5, 6.0 and 12.0 μg/kg/hr of pentagastrin. In the tests using histamine, a prior injection of mepyramine maleate was given to counteract side effects. The dose of
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![Histograms showing concentrations of components in gastric juice](image)

**Fig. 1a.** The mean concentrations of the components in gastric juice, per 15 minutes, in six normal subjects, before and during stimulation by histamine acid phosphate. In all figures the infusions of histamine or pentagastrin were commenced after the second 15-minute period.

Mepyramine was 25 mg for the lower two doses of histamine and 50 and 100 mg for the larger two doses respectively.

The stimulants were diluted in a sterile container with 0.9% saline. The concentration of the stimulant in solution was varied according to the body-weight dose required. The rates of infusion varied from 1.34 to 10.74 ml per 60 minutes, depending on the dose of the stimulant administered. A Palmer constant infusion pump was used to deliver the solution through a scalp vein needle, inserted into a dorsal hand vein.

Each test was preceded by a fast of at least 12 hours, and at least three days elapsed between tests on the same person. The sequence of administration of the stimulants was quite random.

The secretory tests were performed by the technique previously described from this laboratory (Lawrie, Smith, and Forrest, 1964). The throat was sprayed with 4% lignocaine and a 14 F gastric tube was swallowed for a distance of approximately 57 cm. The subject then lay supine, half turned to the left on the examination couch. The position of the tube was adjusted until hand syringe aspiration freely withdrew the gastric contents. The stomach was emptied in this manner, and thereafter continuous low pressure (3-4 mm Hg) pump suction was applied to the gastric tube. To ensure uninterrupted flow of gastric juice a small air vent was cut in the gastric tube and every two to three minutes a few millilitres of air were injected down it. The infusion was delivered into a large vein on the dorsum of the right wrist and the right arm was supported throughout the test by a pillow along the left side.

Two 15-minute samples of basal (unstimulated) gastric juice were collected (and pooled) and then the infusion was started. Thereafter, throughout each test, gastric juice was continuously collected in 15-minute samples. Each test was continued until four successive 15-minute collections of gastric juice of approximately equal volume and acidity were obtained, this 60 minutes being regarded as the steady state, or plateau hour, of acid secretion.

The gastric juice was collected into a flask surrounded by ice, and, following collection, each specimen was filtered through muslin.

**Estimations carried out on each sample of gastric juice**

The volume of each 15-minute sample was recorded in millilitres. The hydrogen ion (H⁺) concentration was determined using a Radiometer automatic titrator (type PPIC). Aliquots of 10 ml of gastric juice were used for titration with 0.1 N NaOH to a pH of 7.0.

Estimations of the concentrations of the sodium (Na⁺) and potassium (K⁺) ions were performed on 0.1 ml aliquots of gastric juice appropriately diluted with freshly deionized water using a flame photometer (EEL standard type). For each batch of specimens, the photometer was standardized over a range of 0 to 100 m-equiv/l for Na⁺ and 0 to 7.5 m-equiv/l for K⁺. This range of concentration was covered by six standard sodium-containing solutions and by
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![Graph showing the effects of different concentrations of pentagastrin on gastric secretion.](image)

Fig. 1b  The mean concentrations of the components in gastric juice before and during stimulation by pentagastrin.

The mean 15-minute concentrations and outputs of each constituent of the gastric juice, in the group of six subjects, are shown in Figure 1. The general pattern of response to stimulation by histamine or pentagastrin was the same. The outputs of acid, chloride, and potassium increased until plateau values were reached. The output of sodium fell, but became approximately constant when the steady state of acid secretion was achieved. The pepsin response to both drugs was characterized by an initial transient peak of output followed by a more or less constant output at lower levels. This output was still in excess of that found in basal secretions.

The volumes of the parietal and non-parietal secretions, calculated according to Hunt’s formula (1951), are shown in Figure 2. Those of the parietal secretion increased to a plateau while those of the non-parietal component fell when steady-state conditions of acid secretion were achieved.

It was noted that, with the larger doses of both stimulants, there was an initial transient increase in the volume of the non-parietal component, and corresponding with this, there was a transient increase in the output of sodium.

Minor differences in the rates of the responses to the drugs were evident. At maximal levels of acid secretion, pentagastrin resulted in an earlier appearance of the steady state and similarly pentagastrin consistently activated the peptic response faster than did histamine.

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SECRETORY OUTPUTS

The mean outputs of the various constituents of the gastric juice, during the plateau hours of acid secretion, in the six subjects, are shown in Table I. Those of acid and pepsin are plotted against the logarithms of the millimolar doses of the stimulants in Figure 3. Clearly, at all levels of secretory activity studied, the potency
pentagastrin is greater than that of histamine acid phosphate, on a molar basis, for both acid and pepsin secretion. At maximal levels of acid and pepsin secretion, the comparative potency of the drugs appears similar and is \( \times 80 \) and \( \times 60 \) respectively.

The mean maximal outputs of acid were elicited by 40 \( \mu \text{g/kg/hr} \) of histamine acid phosphate and by 1-5 \( \mu \text{g/kg/hr} \) of pentagastrin, and there was no significant difference between these outputs, and those in response to 80 \( \mu \text{g/kg/hr} \) of histamine acid phosphate and 6 and 12 \( \mu \text{g/kg/hr} \) of pentagastrin respectively. The mean maximal outputs of pepsin were achieved with 10 \( \mu \text{g/kg/hr} \) of histamine acid phosphate and 0-15 \( \mu \text{g/kg/hr} \) of pentagastrin, and the larger doses of either drug did not result in inhibition of pepsin secretion.

In the group of subjects, the maximal plateau hour outputs of acid, elicited by 80 \( \mu \text{g/kg/hr} \) of histamine acid phosphate and by 6 \( \mu \text{g/kg/hr} \) of pentagastrin, were compared by a paired \( t \) test. They were not significantly different. In the same way, we compared the mean maximal outputs of pepsin, in response to 80 \( \mu \text{g/kg/hr} \) of histamine acid phosphate and 1-5 \( \mu \text{g/kg/hr} \) of pentagastrin, and again we found that they were not significantly different.

**REGRESSION ANALYSIS OF OUTPUTS DURING THE STEADY STATES OF ACID SECRETION**

The relationships between the outputs of the various constituents of the gastric juice, during the steady state hours of acid secretion, following stimulation by the four doses of histamine or pentagastrin, were determined by regression analyses, in individual subjects and in the group of subjects (Table II).

There were highly significant positive correlations between the outputs of acid and those of chloride, potassium, and pepsin. The outputs of acid and sodium were not correlated.

**REGRESSION ANALYSIS OF CONCENTRATIONS DURING THE STEADY STATES OF ACID SECRETION**

In each subject, the relationships between the concentrations of the electrolytic constituents to the gastric juice were determined by regression analyses (Table III). In these analyses, we used...
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### Table II Correlations between outputs of constituents of gastric juice during the steady state hours of acid secretion stimulated by continuous intravenous infusions of histamine acid phosphate and pentagastrin in normal subjects

<table>
<thead>
<tr>
<th></th>
<th>Correlations in the Group</th>
<th>Range of Correlations in Individual Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Histamine</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acid × chloride</td>
<td>0.96&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.91 to 0.99</td>
</tr>
<tr>
<td>Acid × potassium</td>
<td>0.89&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.83 to 0.99</td>
</tr>
<tr>
<td>Acid × sodium</td>
<td>0.18&lt;sup&gt;1&lt;/sup&gt;</td>
<td>−0.11 to −0.79</td>
</tr>
<tr>
<td>Acid × pepsin</td>
<td>0.76&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.54 to 0.97</td>
</tr>
<tr>
<td><strong>Pentagastrin</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acid × chloride</td>
<td>0.96&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.77 to 0.97</td>
</tr>
<tr>
<td>Acid × potassium</td>
<td>0.71&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.08 to 0.92</td>
</tr>
<tr>
<td>Acid × sodium</td>
<td>0.23&lt;sup&gt;1&lt;/sup&gt;</td>
<td>−0.06 to 0.66</td>
</tr>
<tr>
<td>Acid × pepsin</td>
<td>0.49&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.48 to 0.81</td>
</tr>
</tbody>
</table>

<sup>1</sup>Indicates that P < 0.001
<sup>2</sup>Indicates that P > 0.05

The relationships between the concentrations of the H⁺ and Na⁺ in all but one subject (subject 4, after stimulation by pentagastrin). In four subjects with histamine and in all subjects with pentagastrin, there were highly significant positive correlations between the concentrations of the H⁺ and Cl⁻. With the exception of subject 4, highly significant negative correlations were demonstrated between the concentrations of the H⁺ and (Na⁺ + K⁺), in all subjects, after the administration of both stimulants. With the exception of subject 1 with pentagastrin, consistent highly significant, positive correlations related the concentrations of the (Cl⁻ - H⁺) and (Na⁺ + K⁺). The relationship between the concentrations of the H⁺ and K⁺ was either not significant or variable.

The most useful correlations recorded were between the concentrations of the H⁺ × Na⁺ and between those of the (Cl⁻ - H⁺) × (Na⁺ + K⁺). The secretory data were pooled from all the subjects, for stimulation by histamine or pentagastrin. Except in one subject, the concentrations of the H⁺ and K⁺ were not significantly correlated. There was an inconstant relationship between the concentrations of H⁺ and the concentration of pepsin.

**REGRESSION ANALYSIS OF CONCENTRATIONS THROUGHOUT THE INFUSIONS**

The relationships between the concentrations of the electrolytes in the gastric juice following the administration of the four doses of histamine or pentagastrin were also determined in each subject, taking into account all of the 15-minute estimations throughout the whole time of the infusions (Table IV).

The 4 × 15-minute estimations during each of the plateau hours, following stimulation by the four doses of histamine acid phosphate or pentagastrin. In four subjects with histamine and in five subjects with pentagastrin there were highly significant negative correlations between the concentrations of the H⁺ and Na⁺. The concentrations of the H⁺ and Cl⁻ were highly significantly correlated in three subjects with histamine and in five subjects with pentagastrin.
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**Fig. 2**  The mean secretion of the parietal and non-parietal components in gastric juice, per 15 minutes, in six normal subjects, before and during stimulation by histamine acid phosphate (a) and pentagastrin (b). As in Figure 1, the infusions of histamine and pentagastrin were commenced after the second 15 minute period.
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![Graphs showing effects on acid output and pepsin output](http://gut.bmj.com/)

Fig. 3a. The relationships between the mean outputs of acid (a) and pepsin (b) in the gastric juice and the molar doses of histamine acid phosphate and pentagastrin, in six normal subjects.

### Table III  Correlations between the concentrations of the electrolytes and the concentration of pepsin, during steady state conditions in each of six normal subjects.

<table>
<thead>
<tr>
<th>Subject</th>
<th>$H^+ \times Na^+$</th>
<th>$H^+ \times Cl^-$</th>
<th>$H^+ \times K^+$</th>
<th>$H^+ \times Pepsin$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-0.74</td>
<td>0.54</td>
<td>0.071</td>
<td>0.68</td>
</tr>
<tr>
<td>2</td>
<td>-0.33</td>
<td>-0.48</td>
<td>-0.311</td>
<td>-0.80</td>
</tr>
<tr>
<td>3</td>
<td>-0.93</td>
<td>0.82</td>
<td>-0.391</td>
<td>-0.66</td>
</tr>
<tr>
<td>4</td>
<td>-0.18</td>
<td>0.97</td>
<td>0.78</td>
<td>-0.66</td>
</tr>
<tr>
<td>5</td>
<td>-0.93</td>
<td>0.341</td>
<td>0.341</td>
<td>0.351</td>
</tr>
<tr>
<td>6</td>
<td>-0.91</td>
<td>-0.051</td>
<td>0.031</td>
<td>-0.371</td>
</tr>
</tbody>
</table>

1 Indicates that $P > 0.05$

### Table IV  Correlations between the concentrations of the electrolytes throughout the infusions in each of six normal subjects.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Number of Observations</th>
<th>$H^+ \times Na^+$</th>
<th>$H^+ \times Cl^-$</th>
<th>$H^+ \times K^+$</th>
<th>$(Cl^- - H^+)$</th>
<th>$H^+$</th>
<th>Histamine</th>
<th>Pentagastrin</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>20</td>
<td>-0.74</td>
<td>0.050</td>
<td>0.26</td>
<td>0.68</td>
<td>0.62</td>
<td>-0.69</td>
<td>-0.71</td>
</tr>
<tr>
<td>2</td>
<td>21</td>
<td>-0.70</td>
<td>-0.02</td>
<td>0.07</td>
<td>0.62</td>
<td>0.62</td>
<td>-0.77</td>
<td>-0.66</td>
</tr>
<tr>
<td>3</td>
<td>25</td>
<td>-0.89</td>
<td>0.87</td>
<td>-0.46</td>
<td>0.81</td>
<td>0.81</td>
<td>-0.87</td>
<td>-0.27</td>
</tr>
<tr>
<td>4</td>
<td>24</td>
<td>-0.56</td>
<td>0.92</td>
<td>0.49</td>
<td>0.66</td>
<td>0.66</td>
<td>-0.27</td>
<td>-0.89</td>
</tr>
<tr>
<td>5</td>
<td>25</td>
<td>-0.95</td>
<td>0.57</td>
<td>0.66</td>
<td>0.85</td>
<td>0.85</td>
<td>-0.95</td>
<td>-0.71</td>
</tr>
<tr>
<td>6</td>
<td>23</td>
<td>-0.90</td>
<td>0.121</td>
<td>0.091</td>
<td>0.90</td>
<td>0.90</td>
<td>-0.89</td>
<td>-0.27</td>
</tr>
</tbody>
</table>

1 Indicates that $P > 0.05$
pentagastrin, and the correlations between these parameters were again determined by regression analyses. The slopes of the regression lines relating to stimulation by histamine and pentagastrin were then compared by analyses of covariance. For each correlation, the slopes of the regression lines were not significantly different with the two drugs (Figure 4 and Table V).

**STATISTICAL ANALYSIS OF THE SODIUM RESPONSES**

*Sodium output at different steady states of acid secretion*

The outputs of sodium, at different steady states of acid secretion, were compared, in the group of six subjects, by analyses of variance. The 15-minute outputs of sodium during the steady state hours of acid secretion were used in these analyses. There was no significant difference (at the 0.05 probability point) in the output of sodium: (1) in response to the four doses of histamine (variance ratio 0.295); (2) in response to the four doses of pentagastrin (variance ratio 0.068); and (3) in response to the eight doses of histamine and pentagastrin (variance ratio 0.162).

*The relationship between volume of non-parietal component and output of sodium before the establishment of the steady state of acid secretion*

The volume of the non-parietal component, in each 15-minute specimen of gastric juice before the start of the steady state of acid secretion, was calculated in each individual test (Hunt, 1951). The results were pooled from all the subjects for stimulation by histamine or pentagastrin, and the relationship between the volume of the non-parietal component and the output of sodium was established by regression analysis. There were highly significant positive correlations between these parameters, whether histamine or pentagastrin was used to stimulate gastric secretion, and furthermore, the slopes of the regression lines were not significantly different (Fig. 5) with the two drugs ($F = 1.051$).

**Discussion**

Our results clearly show that, on a molar basis, pentagastrin is a more potent stimulant than

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**Table V**  Analysis of pooled data in six normal subjects

<table>
<thead>
<tr>
<th>Parameters (mmol/L)</th>
<th>Histamine</th>
<th>Pentagastrin</th>
<th>Difference between the Drugs $F$ (Slope)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$H^+ \times Na^+$</td>
<td>$r = -0.77$</td>
<td>$r = -0.81$</td>
<td>$0.196^{*}$</td>
</tr>
<tr>
<td>$Cl^- - H^+$</td>
<td>$y = 51.1 - 0.35 x$</td>
<td>$y = 46.5 - 0.33 x$</td>
<td></td>
</tr>
<tr>
<td>$(Na^+ + K^+)$</td>
<td>$r = 0.81$</td>
<td>$r = 0.73$</td>
<td>$0.054^{*}$</td>
</tr>
<tr>
<td></td>
<td>$y = 15.2 + 0.55 x$</td>
<td>$y = 9.2 + 0.54 x$</td>
<td></td>
</tr>
</tbody>
</table>

1Indicates that $p < 0.001$.

*Indicates that the slopes of the regression lines were not significantly different at, least, the conventional 5% level.
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![Graph showing the regression of the output of sodium on the volume of the non-parietal secretion during rising secretory levels (before the steady states of acid secretion), in six normal subjects, following stimulation by histamine acid phosphate and pentagastrin.](image)

Histamine acid phosphate for both acid and pepsin secretion.

As was expected, the mean maximal output of acid was elicited by 40 μg/kg/hr of histamine acid phosphate. With pentagastrin, no single dose consistently evoked maximal acid secretion in all individuals as was reported by Wormsley and Mahoney (1967). The mean maximal output of acid was achieved with a dose of 1.5 μg/kg/hr. This finding agrees with those of Konturek (1967) and Mason and his colleagues (1969), who found that a dose of 1.2 μg/kg/hr of pentagastrin was sufficient to elicit maximal gastric acid secretion; it does not agree with those of the Multicentre Pilot Study (1967) and Aagaard and Schmidt (1967), who concluded that 6 μg/kg/hr of pentagastrin was necessary to evoke maximal gastric acid secretion. It is interesting that with both drugs maximal outputs of pepsin were elicited by doses which were submaximal for stimulation of acid secretion.

Supramaximal doses of either drug did not result in inhibition of acid or pepsin secretion. These findings are consistent with those of Lawrie et al (1964) and Wormsley and Mahoney (1967), and Wormsley et al (1967) but they do not agree with those of Aagaard and Schmidt (1967), who found that 12 μg/kg/hr of pentagastrin resulted in a significantly lower output of acid than 6 μg/kg/hr.

Our findings, that equal maximal outputs of acid and pepsin were elicited by the two stimulants, are in keeping with those of several previous workers (Aagaard and Schmidt, 1967; Konturek, 1967; Konturek and Lankosz, 1967; Multicentre Pilot Study, 1967; Wormsley et al, 1967; Makhlouf, McManus, and Card, 1967).

In the gastric juice, the interrelationships between the concentrations of the Na⁺, K⁺, Cl⁻, and H⁺ were the same with the two drugs—both at steady state conditions and during the whole periods of the tests. Furthermore, analyses of covariances showed that, in the group of subjects, the relationships between the concentrations of H⁺ × Na⁺ and between those of (Cl⁻ − H⁺) × (Na⁺ + K⁺) were not significantly different, whether histamine acid phosphate or pentagastrin was the secretory stimulant. In fact, over a wide range of acid secretion, the actions of the two drugs on electrolyte secretion appeared identical.

It is noteworthy that we found no correlation between the concentrations of the H⁺ and K⁺. This observation agrees with those reported by Linde and Öbrink (1950), Bernstein (1952), Nordgren (1958), Clocher and Hollander (1959), Hollander and Colcher (1960), Hollander (1961), and Öbrink and Waller (1967) but not with that of Makhlouf, McManus, and Card (1966) who found that, at steady-state conditions, there was a highly significant correlation between the concentrations of these ions, whether histamine or gastrin II was the stimulant of secretion. Our data therefore support the view that potassium is not secreted by the parietal cells (Cicardo, 1941; Macmillan and Vane, 1956; Colcher and Hollander, 1959; Hollander and Colcher, 1961; Hollander, 1961; Öbrinsk and Waller 1967).

Like Makhlouf and his colleagues (1966), who used histamine and gastrin II, we found that the output of sodium was constant at different steady states of acid secretion, stimulated by histamine or pentagastrin. This output was well below that found in spontaneous (unstimulated) secretions. This finding clearly supports Makhlouf’s suggestion that the secretion of sodium is determined by non-specific factors, which probably operate at capillary rather than cellular levels. We also confirmed that, during the initial periods of stimulation, when the secretory rate was increasing, there was a strict relationship between the output of sodium and the volume of the non-parietal component (Makhlouf et al, 1966). This relationship also was identical with the two drugs.

We should like to thank Dr T. Khosla, Lecturer in Statistics, Welsh National School of Medicine, for help with the statistics; Mr H. Kincaid and his staff for technical assistance; Mr N. Pearce for the illustrations; and Mrs O. Palmer for her secretarial work.

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