Vagal influence in gastric acid secretion in normals
and in duodenal ulcer patients

HARMIT SINGH, R. K. GOYAL,1 D. S. AHLUWALIA, AND H. K. CHUTTANI2

From the Gastroenterology Unit of the Department of Medicine, Maulana Azad Medical College and
Associate Hospitals, New Delhi, India

The nature of hypersecretion of gastric acid in duodenal ulcer subjects remains controversial. The
duodenal ulcer patients, on an average, secrete more than twice the amount of acid secreted by normal
persons. It is now known that the duodenal ulcer patients have an increased parietal cell population in
the stomach. Whether this increased acid output is solely due to the increased parietal cell mass is
uncertain. Dragstedt and his group has been actively propagating their view of 'vagal hypertonicity' in
these patients for more than 20 years (Dragstedt, Harper, Tovee, and Woodward, 1947; Dragstedt, 1967).
Grossman has, on the other hand, remained unconvinced of Dragstedt's arguments (Grossman,
against the existence of a supranormal drive on the parietal cells in duodenal ulcer patients, whereas
Baron (1963) has suggested that about half the duodenal ulcer patients have a supranormal drive to
explain their basal hypersecretion.

If duodenal ulcer patients do have vagal hypertonicity, blocking the vagal influence should reduce
the gastric acid output to a greater degree in ulcer patients than in the normal person. There are many
studies on the influence of vagal transection (Waddell, 1957; Gillespie, Clark, Kay, and Tankel, 1960;
Bachrach, 1962; Bell, 1964; Gelb, Barofsky, and Janowitz, 1961; Barabas, Payne, Johnstone, and
Burns, 1966; Bank, Marks, and Louw, 1967) and vagal block with drugs, 'medical vagotomy',
upon gastric acid secretion in duodenal ulcer patients. Such data for normal healthy persons are
not available.

This study compares the results of the response of gastric acid secretion to vagal block with a combina-
tion of hexamethonium bromide and atropine sulphate in normal persons and in patients with
duodenal ulcer.

1Present address: Hospital of St Raphael and the Yale University
School of Medicine, New Haven, Connecticut.

2Requests for reprints should be addressed to Dr. H. K. Chuttani.

MATERIAL AND METHOD

Twenty-five healthy volunteers and 24 duodenal ulcer patients were investigated. All these were adult males of
comparable age, weight, and physique. None of them showed evidence of any parasites in the stools. All the
normal subjects were free of abdominal symptoms and had a normal haemoglobin. The normalcy of the upper
gastrointestinal tract was confirmed in each instance by barium meal examination. The duodenal ulcer patients
were radiologically proved cases of the disease. Five of them were operated upon and ulcer was demonstrated in
all of them.

The initial test was performed according to the tech-
nique described in detail elsewhere (Goyal, Gupta, and
Chuttani, 1966).

At another sitting, always within one week of the
initial test, a repeat test was performed following vagal
block with a combination of hexamethonium bromide and
atropine sulphate (McArthur et al, 1960) as follows.
After an overnight fast, a Levine tube with wide holes
near the tip was passed through the nose and the position of
the tip was adjusted to be in the most dependent part of
the stomach under fluoroscopic control. Mechanical
suction was then applied which was assisted by inter-
mittent manual suction and periodic injection of a few
millilitres of air to clear away any mucus obstructing the
flow of gastric juice. The subject was then given a single
intramuscular injection of a combination of hexa-
methonium bromide (50 mg) and atropine sulphate
(0:325 mg). The gastric juice collected over the following
one hour was designated as 'post-vagal block-basal acid
output'. In the middle of this one-hour period, 50 mg of
mepyramine maleate (Anthisan) was injected intra-
muscularly and at the end of the hour, histamine acid
phosphate in a dose of 0:04 mg/kg body weight was given
subcutaneously. In the following hour, four 15-minute
samples of gastric juice were collected. This constituted the
post-vagal block maximal acid output.

The titrations were done immediately afterwards
against freshly prepared 0:1N sodium hydroxide, using
phenolphthalein as indicator. The post-vagal block BAO
and post-vagal block MAO were calculated in m-
equivalents per hour. The repeatability of the test was
tested. The coefficient of variation was 9:16% for MAO
and 16:6% for BAO.

OBSERVATIONS

NORMAL SUBJECTS

The average age of these persons

Gut, 1968, 9, 604-608

604
was 26.84 (SD 5.77) years and their mean weight was 49.72 (SD 4.90) kg.

The values for the volume and acid content of the basal and maximal histamine-stimulated secretion before and after vagal block have been summarized in Table I. The percentage reduction in the volume of basal secretion was 63.0% and it was 78.49% for the basal acid output. The mean reduction was 52.74% for the volume and 66.73% for the acid content after histamine stimulation.

There was good correlation between the basal and the maximal acid output in normal persons (Fig. 1). The coefficient of correlation 'r' was 0.7471 (p < 0.01) before vagal block. A significant correlation between the BAO and MAO continued to exist even after vagal block. Coefficient of correlation 'r' was 0.633 (p < 0.01).

The mean value for the BAO : MAO ratio expressed as a percentage was 21.45 before and 17.42 after vagal block. The difference in these two values was not statistically significant (t = 1.71, p > 0.05).

### DUODENAL ULCER PATIENTS

The mean values, range, and standard deviation of volume and acid content of spontaneous acid secretion and maximal acid output before and after vagal block have been summarized in Table II.

After vagal block, the mean percentage reduction

---

**TABLE I**

<table>
<thead>
<tr>
<th>TABLE I</th>
<th>VOLUME AND ACID CONTENT OF SPONTANEOUS AND 'MAXIMAL' HISTAMINE-STIMULATED SECRETION IN 25 NORMAL MEN BEFORE AND AFTER VAGAL BLOCK</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>Mean</td>
<td>65.60 ± 22.88</td>
</tr>
<tr>
<td>Range</td>
<td>25-120 ± 5.40</td>
</tr>
<tr>
<td>Acid (m-equiv/hr) Mean</td>
<td>3.22 ± 0.71</td>
</tr>
<tr>
<td>Range</td>
<td>0-6-4.7 ± 0.05-2.0</td>
</tr>
<tr>
<td>'Maximal' Acid Output</td>
<td>151.00 ± 70.68</td>
</tr>
<tr>
<td>Mean</td>
<td>90-220 ± 28.62</td>
</tr>
<tr>
<td>Acid (m-equiv/hr) Mean</td>
<td>14.73 ± 4.78</td>
</tr>
<tr>
<td>Range</td>
<td>5.0-19.0 ± 0.6-8.8</td>
</tr>
</tbody>
</table>

**TABLE II**

<table>
<thead>
<tr>
<th>TABLE II</th>
<th>VOLUME AND ACID CONTENT OF SPONTANEOUS AND MAXIMAL HISTAMINE-STIMULATED SECRETION IN 24 DUODENAL ULCER PATIENTS BEFORE AND AFTER VAGAL BLOCK</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>Mean</td>
<td>91.12 ± 35.94</td>
</tr>
<tr>
<td>Range</td>
<td>45.235 ± 25-80</td>
</tr>
<tr>
<td>Acid (m-equiv/hr) Mean</td>
<td>5.32 ± 1.70</td>
</tr>
<tr>
<td>Range</td>
<td>1-8-15.3 ± 0-1-5</td>
</tr>
<tr>
<td>'Maximal' Acid Output</td>
<td>260.71 ± 100.54</td>
</tr>
<tr>
<td>Mean</td>
<td>190-385 ± 42.04</td>
</tr>
<tr>
<td>Acid (m-equiv/hr) Mean</td>
<td>28.67 ± 9.89</td>
</tr>
<tr>
<td>Range</td>
<td>20-8-46.97 ± 2.0-24.12</td>
</tr>
</tbody>
</table>
was 56.78% and 67.63% for volume and acid content of basal secretion, respectively, and 60-31% for volume and 64.45% for acid content of the maximal histamine-stimulated secretion.

There was no correlation between the basal and the maximal acid output in these patients before and after vagal block. The coefficient of correlation was 0.2496 (p > 0.05) before and 0.3345 (p > 0.05) after vagal block.

The BAO:MAO ratio was 19.04% before and 18.67 after vagal block. There was no significant difference in these two ratios (t = 1.10, p > 0.05).

**COMPARISON OF VALUES OF NORMALS AND DUODENAL ULCER PATIENTS** The normal persons and duodenal ulcer patients did not differ significantly in their age distribution (t = 1.77; p > 0.05) and their weight (t = 1.27, p > 0.05).

Values for all the components of gastric acid were significantly more in duodenal ulcer patients both before and after vagal block (Table III). The absolute reduction in maximal acid output was also greater in ulcer patients. However, the reduction expressed as the percentage of the initial value was similar in the two groups of cases. This was true for both basal as well as the maximal acid output (Table IV).

**COMMENTS**

These observations indicate that the percentage reduction in acid output, after vagal block, is not different in duodenal ulcer and normal persons and, therefore, negates the hypothesis of a supranormal vagal influence in duodenal ulcer patients to account for their hypersecretion.

Hunt (1950), analysing Ahrer's data (1938), pointed out that duodenal ulcer patients secreted significantly more gastric acid than normals in response to insulin as well as to histamine stimulation. However, the

**TABLE III**

<table>
<thead>
<tr>
<th>Before Vagal Block</th>
<th>Normal</th>
<th>Duodenal Ulcer</th>
<th>Value of t</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal acid output</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Volume (ml/hr)</td>
<td>65.60</td>
<td>91.12</td>
<td>2.27</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Acid (m-equiv/hr)</td>
<td>3.22</td>
<td>5.32</td>
<td>3.07</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Maximal acid output</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Volume (ml/hr)</td>
<td>151.00</td>
<td>260.71</td>
<td>8.78</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Acid (m-equiv/hr)</td>
<td>14.73</td>
<td>28.67</td>
<td>8.82</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>BAO/MAO x 100</td>
<td>21.45</td>
<td>19.94</td>
<td>0.86</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>After Vagal Block</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basal acid output</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Volume (ml/hr)</td>
<td>22.88</td>
<td>35.94</td>
<td>2.32</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Acid (m-equiv/hr)</td>
<td>0.71</td>
<td>1.70</td>
<td>3.62</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Maximal acid output</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Volume (ml/hr)</td>
<td>70.68</td>
<td>100.54</td>
<td>2.92</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Acid (m-equiv/hr)</td>
<td>4.78</td>
<td>9.89</td>
<td>4.31</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>BAO/MAO x 100</td>
<td>17.42</td>
<td>18.67</td>
<td>0.36</td>
<td>&gt; 0.05</td>
</tr>
</tbody>
</table>

**TABLE IV**

<table>
<thead>
<tr>
<th>Reduction in Acid Secretion Expressed as a Percentage of the Initial Value Upon Vagal Block in Normals and Duodenal Ulcer Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal Acid Output</td>
</tr>
<tr>
<td>Volume (ml/hr)</td>
</tr>
<tr>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Acid (m-equiv/hr) ± SD</td>
</tr>
<tr>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Maximal Acid Output</td>
</tr>
<tr>
<td>Volume (ml/hr)</td>
</tr>
<tr>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Acid (m-equiv/hr) ± SD</td>
</tr>
<tr>
<td>Mean ± SD</td>
</tr>
</tbody>
</table>
Vagal influence in gastric acid secretion in normals and in duodenal ulcer patients

Gastric acid secretory response was investigated before and after vagal block with a combination of hexamethonium bromide and atropine sulphate in 24 duodenal ulcer patients and 25 healthy subjects.

The mean values for acid secretion, volume as well as acid output in milliequivalents per hour were significantly higher in duodenal ulcer patients than in normal persons, both before and after vagal block. However, the percentage reduction on vagal block was 78.49% in normals and 67.63% in ulcer patients for basal acid output and 66.73% in normals and 64.45% in duodenal ulcer for maximal acid output. These differences in the percentage reduction in normal and duodenal ulcer patients were not statistically significant.

The basal to maximal acid output ratio was 21.45% and 19.04% before vagal block and 17.42% and 18.67% after vagal block in normals and duodenal ulcer patients respectively. All these values are statistically insignificant.

These findings negate the thesis of vagal hypersecretion to explain hypersecretion in duodenal ulcer patients.

We are grateful to Dr O. P. Bhardwaj, Professor of Radiology for his help, and Dr P. C. Dhanda, Principal-Director Maulana Azad Medical College and Associate Hospitals to publish this material.

REFERENCES


—— (1967). Dragstedt's editorial on gastric secretion tests. (Comments.) Ibid., 53, 681.


