GASTRIC ACID SECRETION IN HYPERTHYROIDISM AMONG CHINESE PATIENTS

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SYNOPSIS

The effect of hyperthyroidism on gastric secretion in Chinese subjects was studied. Basal and pentagastrin-stimulated peak acid outputs were measured before commencing anti-thyroid therapy and repeated when euthyroid state was achieved. Each patient therefore acted as its own control. The results were also compared to Chinese control subjects who had no gastrointestinal or thyroid diseases. There was a significant increase in both basal and peak acid outputs with the control of hyperthyroidism. However the gastric secretory capability in both the toxic and euthyroid phases were significantly greater than the controls. This is contrary to previous reports of normal or hyposecretion in thyrotoxic patients. Increased acid secretion in patients with hyperthyroidism may be peculiar to the Chinese.

INTRODUCTION

It had long been shown that hyperthyroidism was associated with decreased gastric acid outputs and a significant number of patients with hyperthyroidism had achlorhydria. Patients with pernicious anemia had been observed to have previously suffered from hyperthyroidism. Two probably causes for hyposecretion were suggested, namely that of associated presence of autoimmune chronic gastritis in the gastric mucosa (Doniach et al 1963, William et al 1966) and that of hypertonia of the sympathetic pathways (Mlodski et al 1970). However the effect of hyperthyroidism as far as we know, in the Chinese ethnic group had never been investigated.

MATERIALS AND METHODS

Basal and pentagastrin-stimulated peak acid outputs were determined in 10 patients with hyperthyroidism and 20 control subjects. All the subjects were Chinese. The ten patients with
hyperthyroidism had no gastrointestinal complaints while the control subjects had neither thyroid nor gastrointestinal disorders. The diagnosis of hyperthyroidism was made on clinical findings and laboratory data. Taking a score of 20 and above to indicate hyperthyroidism on the Wayne's index, the mean score (± S.D.) for the 10 patients was 32.3 ± 4.4

Basal and pentagastrin-stimulated gastric acid outputs in untreated newly diagnosed hyperthyroid patients were determined. The procedure for the gastric secretory study had been previously described (Fung et al 1972). Pentagastrin (Peptavlon I.C.I.) given intramuscularly at a dose of 6 ug/kg body weight was used. The tests were repeated in the euthyroid phase. The mean (± S.D.) interval between the tests were 113.2 (± 34.2) days.

The results were subjected to statistical analysis taking P O.05 to be the limits of significance of difference.

RESULTS

The results of basal and pentagastrin-stimulated peak acid outputs in the group of 10 patients are shown in Fig 1. and Fig 2. The mean (± S.D.) B.A.O in the euthyroid is 3.54 (± 3.06) which is significantly greater than in the thyrotoxic, 1.60 (± 1.34) with P < 0.02. The mean (± S.D.) PAO in the euthyroid is 16.49 (± 7.66) which is like-wise significantly greater than in the thyrotoxic, 11.08 (± 7.17) with P < 0.01. Both the BAO and PAO in the thyrotoxic and in the euthyroid patients are significantly greater than in the control, P < 0.0005. Table I and Table II.

There was no patient with achlorhydria.

DISCUSSION

As early as 1904 Miesowiscz noted impaired gastric acid secretion in patients with hyperthyroidism. The gastric mucosa and the thyroid gland are both derived from the primitive foregut. Because of their common analage the gastric mucosa, like the thyroid follicles, are capable of concentrating iodide from the blood stream (Brown-Grant, 1961).

Early reports showed significant incidence of achlorhydria, and chronic gastritis was blamed as the cause of hyposecretion. A possible autoimmune process was suggested to be the basis of chronic gastritis. A group of patients with pernicious anaemia was noted to have suffered from previous hyperthyroidism. Autoimmune reaction can be demonstrated in the majority of patient with thyroid disease and pernicious anaemia, and in a smaller proportion of cases cross-reaction occurred. (Bock et al, 1963;
TABLE I: Result of basal and pentagastrin-stimulated peak acid output before and after treatment for thyrotoxicosis.

<table>
<thead>
<tr>
<th></th>
<th>B.A.O.</th>
<th>Euthyroid vs Thyrotoxic</th>
<th>P.A.O.</th>
<th>Euthyroid vs Thyrotoxic</th>
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<tr>
<td></td>
<td>n</td>
<td>Mean</td>
<td>SD</td>
<td>t</td>
</tr>
<tr>
<td>Thyrotoxic</td>
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<td>1.60</td>
<td>1.34</td>
<td>-</td>
</tr>
<tr>
<td>Euthyroid</td>
<td>10</td>
<td>3.54</td>
<td>3.06</td>
<td>3.087</td>
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</table>

TABLE II: Result of basal and pentagastrin-stimulated acid output — control vs patients.

<table>
<thead>
<tr>
<th></th>
<th>B.A.O.</th>
<th>Thyroid Group vs Control</th>
<th>P.A.O.</th>
<th>Thyroid Group vs Control</th>
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<tr>
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<td>3.54</td>
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William et al. (1966).

The reason for a high incidence of achlorhydria in the early reports was due to the fact that the stimulus used were either some form of test meals and or conventional doses of histamine, all of which provided inadequate stimulus for the parietal cells (Card et al., 1955). Using augmented histamine test some authors (Kay 1953; Card and Sircus 1958) reported no alteration in gastric secretion in hyperthyroid patient while others (William et al. 1964) reported basal and peak acid outputs were considerably reduced as compared to normal subjects. However gastric acid secretion returned to normal when euthyroidism was obtained (Młodski et al., 1970). It would appear that chronic gastritis in the gastric mucosa was incidental and had little or no direct relation to decreased acid outputs, unless it was suggested that chronic gastritis improved with euthyroidism.

Our findings not only showed that gastric secretion increased with treatment of hyperthyroidism but also the increased gastric outputs in patients against normal control subjects in either hyperthyroid or euthyroid states. This finding may be intrinsically unique in the ethnic Chinese where achlorhydria and pernicious anaemia are rarely diagnosed.

REFERENCES