Progressive increase in the functional G cell mass with age in atrophic gastritis

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SUMMARY Patients with atrophic gastritis but normal antral mucosa and achlorhydria were divided into three groups according to age—under 40, 40 to 70, and over 70 years. Serum gastrin, both basal and following a standard protein meal, was estimated in all patients by radioimmunoassay. There was a significant correlation between the magnitude of the gastrin response and age, the older the patient the greater the response. These results suggest that with increasing duration of gastritis and continued stimulation of a normal antrum in the absence of inhibition by acid, the functional G (gastrin) cell mass increases. However the possibility exists that each cell may secrete more gastrin in response to the same stimulus with age. This may be resolved by counting the number of G cells in the stomachs of subjects with atrophic gastritis and different ages.

Patients with pernicious anaemia or atrophic gastritis sero-positive for the parietal cell antibody in general show antral sparing from the gastritic process, basal hypergastrinaemia, and an increased gastrin-secreting (G) cell mass (Korman, Strickland, and Hansky, 1971; Strickland, Bhathal, Korman, and Hansky, 1971; Korman, Strickland, and Hansky, 1972). The degree of gastrin elevation is quite variable and there is a significant correlation between basal serum gastrin levels and age in these patients (Strickland, Korman, and Hansky, 1973). It was suggested that G cell numbers in the spared antrum increase with age and increasing duration of gastritis.

In the present study, the functional G cell mass has been measured in patients with atrophic gastritis and antral sparing and correlated with age.

Material and Methods

Fourteen patients with atrophic gastritis and a histologically normal antral mucosa were studied. All had histamine-fast achlorhydria which refers to failure to change pH by more than 1 unit after stimulation, and a positive test for parietal cell antibody. There were seven females and seven males, aged between 24 and 85 years.

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After an overnight fast, a 19-gauge needle was inserted into a forearm vein, kept patent by frequent flushing with a solution of heparin, 1000 units in 20 ml of 0.9% sodium chloride, and blood was collected 30 minutes before, at the time of, and at 15-minute intervals for two hours after a standard protein meal (Korman, Soveny, and Hansky, 1971).

Serum gastrin was estimated in duplicate by radioimmunoassay (Hansky and Cain, 1969; Hansky, Soveny, and Korman, 1971). The comparison of differences between group means was by use of Student's t test; the relation between magnitude of gastrin response to protein and age was assessed by regression analysis (Snedecor and Cochran, 1968).

Results

The patients were grouped according to their ages: four patients less than 40 years, six patients between 40 and 70 years, and four patients over 70 years.

Figure 1 compares the serum gastrin responses to protein in these three patient groups. In patients under 40 years, serum gastrin (mean ± SEM) rose significantly from a basal level of 157 ± 19 pg/ml to a peak of 330 ± 31 pg/ml, 60 minutes after protein (p < 0.001). In patients between 40 and 70 years, serum gastrin rose significantly from a basal level of 640 ± 79 pg/ml to a peak of 1345 ± 130 pg/ml, 75 minutes after protein (p < 0.001). In patients over 70 years, serum gastrin rose sig-
patients
is
103
line
is
550
Fig 1 Serum gastrin in patients with atrophic gastritis plotted against age. Scale of vertical axis is 10^3 pg/ml. All values are mean ± SEM.

Fig 2 Absolute rise in serum gastrin in patients with atrophic gastritis plotted against age. Correlation line is significant (r = 0.8684, p < 0.001).
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