

symptoms continued after dilatation; 13 others had occasional symptoms only. Though health gradually deteriorates even if symptoms are controlled by dilatation and drugs, 41 patients had a satisfactory quality of life after surgery (groups S_A, B, and C).

There was no appreciable difference in either post-operative mortality or recurrence of symptoms in the two groups having conservative or radical surgery. The 30 day mortality was similar to that reported for younger patients^{3,5} and might be reduced with better selection of patients. The five year survival in our

patients was 68%. More elderly patients should be considered for surgery for oesophageal stricture.

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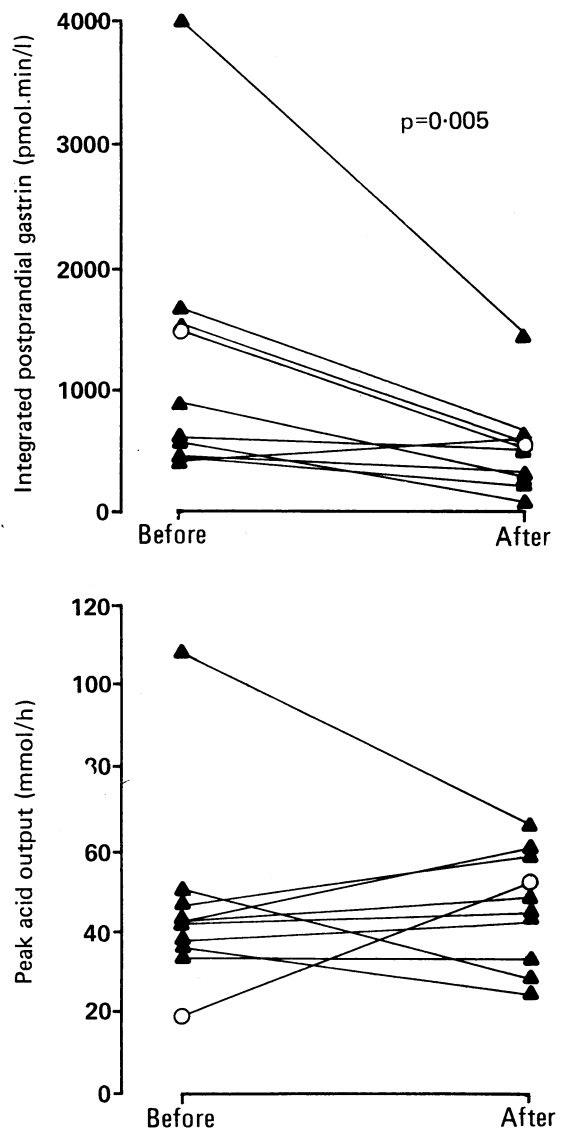
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Antral *Helicobacter pylori*, hypergastrinaemia, and duodenal ulcers: effect of eradicating the organism

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drop in the postprandial response of plasma gastrin concentration. This supports our contention that *H pylori* is responsible for hypergastrinaemia in patients with duodenal ulcers.²

Patients with duodenal ulcer disease tend to have higher rates of secretion of gastric acid and higher postprandial plasma gastrin concentrations than controls. Walsh *et al* showed that a low intragastric pH inhibits release of gastrin less effectively in patients with duodenal ulcer disease than in normal subjects.⁴ On the basis of our recent results² we proposed that *H pylori* in the gastric antrum increases the release of gastrin. *H pylori* produces a powerful urease that splits



Changes in integrated postprandial gastrin responses and peak acid output in 10 patients before and after treatment with colloidal bismuth subcitrate for four weeks and metronidazole for two weeks. ○—○=Patient in whom urease test after treatment yielded weakly positive result

There is strong epidemiological evidence to support a relation between colonisation of the gastric antrum with *Helicobacter pylori* (previously *Campylobacter pylori*) and duodenal ulcers,¹ though the basis of this association is speculative. We recently showed that in patients with duodenal ulcers secretion of gastric acid stimulated by pentagastrin and postprandial plasma gastrin concentrations are significantly higher in patients positive for *H pylori* than in those negative for the organism.² In the present study we examined whether the high postprandial gastrin concentrations respond to treatment that eradicates *H pylori*.

Patients, methods, and results

We studied 51 patients with active duodenal ulcers. Seven were negative for *H pylori* and 44 positive on urease testing of antral biopsy specimens.² Secretion of gastric acid was determined with pentagastrin (6 µg/kg/h). Venous blood was collected before and after a standard meal for radioimmunoassay of gastrin.²

Peak secretion of acid stimulated by pentagastrin was 45.2 (SEM 3.0) mmol/h in the patients who were positive for *H pylori* compared with 29.7 (4.0) mmol/h in those who were negative ($p < 0.05$). Basal gastrin concentrations were significantly higher in the patients who were positive for *H pylori* (13.1 (2.0) v 6.3 (2.6) pmol/l, $p < 0.05$). Integrated plasma gastrin responses to the standard meal were 1564 (267) pmol.min/l in the patients positive for *H pylori* and 965 (248) pmol.min/l in those negative for *H pylori* ($p < 0.05$).

Ten of the patients positive for *H pylori* were treated with metronidazole 400 mg thrice daily for two weeks and colloidal bismuth subcitrate 120 mg four times daily for four weeks.³ Two days after completing treatment nine patients were negative for the organism. Integrated postprandial gastrin responses fell from 1184 (350) to 498 (117) pmol.min/l ($p = 0.005$) while peak rates of secretion of acid remained unchanged (figure). Basal gastrin concentrations, however, were not significantly lower after treatment (7.5 (1.8) v 6.7 (1.9) pmol/l, respectively).

Comment

We showed that eradicating antral *H pylori* in patients with duodenal ulcers leads to a significant

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urea in the stomach to produce ammonia. We therefore postulate that ammonia increases the pH in the mucus layer that overlies the gastric antrum, resulting in impairment of the normal inhibition of gastrin release by intraluminal acid.

This is the first report of a fall in postprandial plasma gastrin responses after eradication of *H pylori* in patients with duodenal ulcers. This could explain the reduced likelihood of relapse of ulcers in patients treated with agents that suppress or eradicate *H pylori*.¹ The fall in postprandial plasma gastrin concentrations, which does not occur after treatment with H₂ antagonists⁵ and is therefore not simply a result of the ulcer healing, would be expected to result in a diminished postprandial secretory drive on the gastric parietal cells. Another probable long term result of the lower gastrin concentrations is a reduction in the number of parietal cells owing to a reduction in the trophic effect of gastrin on these cells. Peak acid output, which reflects the parietal cell mass, did not fall significantly in our patients after one month of

treatment to eradicate *H pylori* despite the lower gastrin concentrations. Thus a longer period of treatment may be needed before the number of parietal cells falls.

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Erythrocyte zinc concentrations in subacute (de Quervain's) thyroiditis

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Patients with subacute thyroiditis classically present with fever, neck pain, and a tender goitre. Functional changes in thyroid state include both hyperthyroidism and hypothyroidism. Constitutional symptoms may, however, be absent and patients may present with features of hyperthyroidism alone, thus making differentiation from other causes of hyperthyroidism difficult. Nevertheless, differentiation is important as the implications for treatment are different. In such cases measurement of the uptake of radioiodine by the thyroid gland is useful, but it may not be measured unless thyroiditis is suspected. Thus the disorder may be misdiagnosed and patients given inappropriate treatment.

As patients with subacute thyroiditis are hyperthyroid for a short time a marker that reflects the duration of their hyperthyroidism might be useful in differentiating their condition from established hyperthyroidism—for example, that due to Graves' disease. Erythrocyte zinc concentrations are decreased in patients with established hyperthyroidism.¹ Because, however, the circulating life span of erythrocytes is long, the erythrocyte zinc concentration reflects the integrated functional state of the thyroid over a period of months. Thus the erythrocyte zinc concentration may be used to differentiate between transient hyperthyroidism and established hyperthyroidism lasting more than two to three months. We previously reported that the erythrocyte zinc concentration differentiated between transient hyperthyroxinaemia associated with hyperemesis gravidarum and pre-existing thyrotoxicosis,² and we report the results in patients with transient hyperthyroidism associated with subacute thyroiditis.

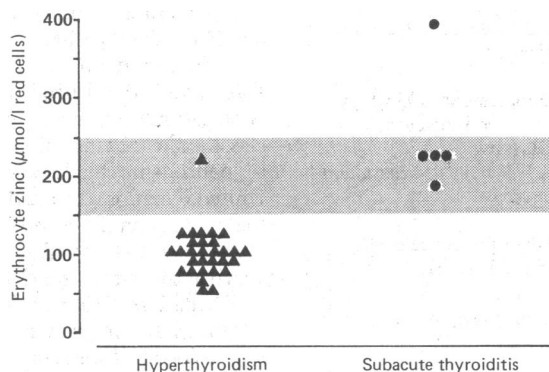
Patients, methods, and results

We studied five patients (four women and one man ranging in age from 35 to 42) with clinically inconvertible subacute thyroiditis. Hyperthyroidism was

suspected clinically in four of them and was confirmed in all five by measuring the concentrations of thyroid hormones. All five patients presented with fever, anterior neck pain, a tender goitre, and a raised erythrocyte sedimentation rate (mean 111 mm in the first hour, range 58-150); the uptake of radioactive iodine by the thyroid gland was subnormal in all five and none had thyroid antibodies.

Erythrocyte zinc concentrations were measured by atomic absorption spectrophotometry.¹ The coefficient of variation between days was 5%.

Erythrocyte zinc concentrations in the five patients were compared with those in 30 patients with established thyrotoxicosis and 110 control patients who were euthyroid (figure). The mean erythrocyte zinc



Erythrocyte zinc concentrations in 30 patients with established thyrotoxicosis (▲) and five patients with subacute thyroiditis (●). Hatched area shows 2 SDs of mean in 110 euthyroid control patients

concentration in the patients with thyrotoxicosis (105 (SD 64) µmol/l red cells) was significantly lower than that in the euthyroid control patients (196 (50) µmol/l red cells, $p < 0.001$). The mean erythrocyte zinc concentration in the patients with subacute thyroiditis was, however, 255 µmol/l red cells (range 185.6-394.7) and not significantly different from that in the control group. One patient had an erythrocyte zinc concentration of 394.7 µmol/l red cells but he also had a thalassaemia trait with microcytosis.

Comment

The normal erythrocyte zinc concentrations in our patients show that their hyperthyroidism was transient and therefore provides additional evidence to support