Gastritis which was associated with many long tightly coiled spiral organisms. The patient's symptoms resolved spontaneously over six weeks and she refused further endoscopy.

**Case 2**

In February 1989 a 40 year old man presented with a six year history of recurrent burning epigastric pain and nausea. There were no abnormal signs and routine blood tests provided normal results, but endoscopy showed a mild antral gastritis. Histological examination showed a mild chronic gastritis with many long coiled spiral organisms adjacent to the mucosa. His symptoms worsened and he was treated with bismuth subcitrate (Denol) 1 g only for four weeks. After this he improved and both repeat endoscopy and subsequent histology were normal, with no gastritic spiral organisms.

Both patients had a gastritis compatible with infection by *Gastrospirillum hominis* and bismuth subcitrate may possibly be an effective treatment. Clarification of this problem must await successful methods of culture.

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**Dr McNulty comments:**

I do not agree with the conclusions drawn by Dr Logan and his colleagues from their two case histories of *"Gastrospirillum hominis"* in patients attending endoscopy for the investigation of abdominal pain and nausea. They suggest that *Gastrospirillum hominis* was clinically important in these patients—by this I assume they meant that the organism was responsible for their presenting symptoms. The case histories do not bear this out. The symptoms of case 1 resolved spontaneously over six weeks: we do not know whether the organism was present when the woman was asymptomatic, but it is likely that they were as we and others (Heilmann KL, Borchard F. Gastric spiral bacteria. Second International symposium on Campylobacter pylori. Bad Nauheim, August 1989, to be published) have shown that the infection is chronic. The second patient's symptoms improved with bismuth subcitrate in parallel with his gastritis, suggesting that the organism, like C

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### MATTERS ARISING

**New spiral bacterium in the gastric mucosa: Gastrospirillum hominis**

We read with interest the paper by McNulty et al and note that they considered *Gastrospirillum hominis* to be responsible for the symptoms of only one of their five patients. We have seen two patients in whom we believe *Gastrospirillum* was a clinically important pathogen.

**Case 1**

A previously fit woman of 56 presented in March 1987 with a four month history of nausea, malaise, abdominal pain and weight loss. She did not smoke and drank 20 units of alcohol a week. Examination and routine blood tests gave normal results but endoscopy showed widespread submucosal haemorrhages and thickened yellow mucosal folds. Histological examination showed an active

**Results of patients and control studied**

<table>
<thead>
<tr>
<th>Mean (SD) values</th>
<th>Patient</th>
<th>Control</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgE (IU/ml)</td>
<td>132.36 (9.39)</td>
<td>42.94 (4.69)</td>
<td>p &lt; 0.01</td>
</tr>
<tr>
<td>Total eosinophil counts (mm³)</td>
<td>2.56 (0.2)</td>
<td>0.76 (0.11)</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>Eosinophils in peripheral blood film</td>
<td>8.77 (0.89)</td>
<td>6.97 (0.05)</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>Duration of the disease (years)</td>
<td>20.07 (2.1)</td>
<td>18.97 (0.9)</td>
<td>p &lt; 0.05</td>
</tr>
</tbody>
</table>

criteria of the disease were described in 1972 and have gained world wide acceptance. According to this classification when four major signs (oral and genital ulcers, non-ulcerative skin lesions, and ocular disease) are present it is accepted as complete Behcet's disease. The diagnosis of Behcet's disease was made according to the diagnostic criteria established by the Research Committee on Behcet's disease. The patients studied had no evidence of a personal or family history of atopy. The stools of the patients and healthy subjects were examined microscopically three times over six months. Patients receiving medication who had parasites in their stool were excluded from the study. All the patients were given a complete physical, ophthalmic, and dermatological examination in addition to the routine laboratory tests. Total number of eosinophils in the peripheral blood and peripheral blood film were counted. Serum IgE concentrations were measured with a radioimmunoassay method, using a slight modification of the Cska and Lunk method. The table shows the total number of eosinophils in the peripheral blood; the peripheral blood film didn't show any significant difference between patients with Behcet's disease and controls (p > 0.05). Serum IgE concentrations in Behcet's disease were higher than those of the controls (p < 0.01). Duration of the disease varied from four to 18 years and there was a positive correlation between serum IgE concentrations and the duration of the disease (r = 0.85 ± 0.06; p < 0.05).

It has been reported that thrombosis in the great veins and arteries can occur at any stage of Behcet's disease. IgE mediated antigenic response, probably by action on mast cells or basophils, can induce platelet activation and this activation results in platelet aggregates and perhaps arterial smooth muscle hyperplasia. Such evidence may suggest a reasonable biological pathway linking increased serum IgE concentrations and Behcet's disease.

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Serum IgE concentrations in complete Behçet's disease.

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