Hiatus hernia and heartburn

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SUMMARY The symptoms in a group of 80 patients with a pure sliding hiatus hernia were investigated using the pyloric regulation test (Capper, Airth, and Kilby, 1966). It was found that there was a high correlation between the symptoms of heartburn and the reflux of duodenal barium into the stomach.

Heartburn is one of the most distressing of presenting symptoms in patients with hiatus hernia. The definition followed here resembles that of Tuttle, Rufin, and Bettarello (1961); it is described as 'a warm discomfort situated in the epigastrium or retrosternal region, usually radiating up to the throat or more rarely across the chest, and is usually associated with intake of food or change in posture'.

The evidence for the pain of heartburn originating from the lower oesophagus is strong and investigations have been along two lines: (1) the upward reflux of a noxious fluid from the stomach; and (2) response of the lower oesophagus in the form of inflammatory changes, motility changes, or both. We decided to investigate the former aspect, ie, the possible nature of the fluid. So far, the evidence has been in favour of gastric rather than duodenal juice being responsible for heartburn.

Evidence for gastric juice has been gathered in several ways. Aylywin (1953) and Flood, Wells, and Baker (1955) have used aspiration techniques, and found a fluid of low pH containing pepsin in symptomatic patients. Tuttle, Bettarello, and Grossman (1960) and Tuttle et al (1961) measured the pH at the lower end of the oesophagus and found the level to fall to pH 4 coinciding with the onset of heartburn, and a rise to a more neutral value coinciding with relief. Bernstein and Baker (1958) perfused the lower end of the oesophagus with N/10 hydrochloric acid in order to differentiate chest pain due to oesophagitis from chest pain of cardiac origin. They found that a high proportion of patients in the former group gave a positive response.

Interest in the evidence for duodenal juice is increasing today and has been prompted by the fact that heartburn is known to occur after total gastrectomy, after acid-reducing operations (Brain, 1966), and in the presence of achlorhydria (Palmer, 1960).

Excellent experimental work by Levrat, Lambert, and Kirshbaum (1962), using a delicate surgical technique in rats, showed that diverting the pancreatic juice and also bile onto the mucosa of the oesophagus caused a much more severe oesophagitis than would pure gastric juice. Though the oesophagus of the rat is histologically different from that of man, this work adds interest to comparative pathology.

Davenport (1967) by means of experimental work in dogs showed that duodenal juice caused structural and functional damage to the gastric mucosa as a result of the lytic action of duodenal juice on the protective mucous layer of the stomach. It therefore seems reasonable to us that when the cardia is incompetent this same juice can equally affect the oesophageal mucosa. The observation volunteered by several of our patients that they spat 'green bile into their handkerchiefs' prompted us to investigate this problem more fully.

MATERIAL

The assessment was made by separate clinical and radiological teams, the latter team not aware of the clinical findings, its sole duty being to report on the behaviour of the pyloric sphincter.

Eighty patients attending general surgical, general medical, or thoracic surgical departments, either as inpatients but mostly as outpatients, were selected. All had a full barium meal examination and were found to have no other radiological lesion than a hiatus hernia.

All but two of these 80 patients were interviewed by one of us (E.W.G.) and their histories were based on the following questionnaire:

1 Heartburn The patient's description had to be in his or her own words, but to correspond more or less to our definition. He or she had to have experienced heartburn on the day of the test or the day before.

2 Vomiting More than twice in the previous six months.
FIG. 1. Group A: the normal competent pylorus.

FIG. 2. Group B: incompetent pylorus, erect position.

FIG. 3. Group B: incompetent pylorus, supine position.
3 Regurgitation This meant the awareness of unpleasant fluid in amounts which could be swallowed or spat out.

4 Abdominal pain Recorded when it occurred separately from heartburn.

5 Dysphagia A sensation of food sticking in a retrosternal position on swallowing. This had to be more than twice in the previous six months.

6 Bleeding This had to be frank haematemesis. A story of melaena alone was not accepted.

METHOD

The 80 patients were submitted to the pyloric regurgitation test as described by Capper et al (1966) and were examined by either of the two radiologists (G.R.A. or M.J.G.). The purpose of this radiological examination was to group patients according to their pyloric competence: group A, competent (Fig. 1) and group B, incompetent (Figs 2 and 3).

The duodenal intubations were always done by the same person (I.B.) in the ward or department, according to whether they were in-patients or out-patients. About three or four hours later they were taken to the X-ray Department for screening. When possible, patients in group B were tipped into a mild Trendelenburg position (about 20°) to see if a hernial sac at the hiatus could be demonstrated (Fig. 4).

RESULTS

According to the test, 24 patients fell into group A and 56 into group B. The ratio of males to females was 13:11 and 25:31, respectively. In the latter group it was not always possible to outline the hernial sac itself, but in the patient of Fig. 4 a few seconds after demonstrating the sac an oesophageal stricture was also seen (Fig. 5). Every drop of barium outlining that stricture had originally been instilled into the second part of the duodenum, and the tube had been removed before the patient was tilted into the Trendelenburg position.

The distribution of symptoms attributable to the hiatus hernia was tabulated according to the two groups (Table I). For ease of comparison, a histogram was constructed to show the percentages of each symptom (Fig. 6).

The striking difference that can be seen in the two groups is that heartburn in group B is so much commoner. While dysphagia and bleeding have not

TABLE I

THE ASSOCIATION OF HEARTBURN AND AN INCOMPETENT PYLORIS

<table>
<thead>
<tr>
<th>With Heartburn (159 cases)</th>
<th>Without Heartburn (21 cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Competent</td>
<td>Incompetent</td>
</tr>
<tr>
<td>7</td>
<td>52</td>
</tr>
</tbody>
</table>

Yates correction: \( p = 0.001 \)
yet been shown to be significantly more common, the association of heartburn and an incompetent pylorus was highly significant (p = 0.001, Table II).

**TABLE II**

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Pylorus Competent (Group A) (%)</th>
<th>Pylorus Incompetent (Group B) (%)</th>
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</thead>
<tbody>
<tr>
<td>Heartburn</td>
<td>7 (29%)</td>
<td>51 (93%)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>13 (56%)</td>
<td>30 (54%)</td>
</tr>
<tr>
<td>Regurgitation</td>
<td>17 (71%)</td>
<td>35 (65%)</td>
</tr>
<tr>
<td>Belching</td>
<td>17 (71%)</td>
<td>35 (65%)</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>17 (71%)</td>
<td>41 (71%)</td>
</tr>
<tr>
<td>Dysphagia</td>
<td>6 (25%)</td>
<td>29 (52%)</td>
</tr>
<tr>
<td>Bleeding</td>
<td>4 (17%)</td>
<td>20 (36%)</td>
</tr>
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</table>

**DISCUSSION**

The actual mechanism which leads to heartburn is still not clearly understood. Abnormal motility findings have been clearly shown in conjunction with heartburn by Jones (1938) and Texter (1967). Also, oesophagitis and heartburn definitely coexist. Siegel and Hendrix (1963) in a series of 25 patients with heartburn found oesophagitis in 17. However, eight asymptomatic patients had biopsy evidence of chronic oesophagitis. Palmer (1960) found that some patients with heartburn had normal looking mucosa but biopsy revealed signs of submucosal inflammation.

In our own experience of 24 endoscopies, 19 patients suffered from heartburn, and 15 of those had definite oesophagitis to the naked eye. On the other hand, two out of the five without heartburn showed mild inflammation.

It is tempting, but too soon, to draw clinical applications from this work and that of others. There has been a movement to perform a pyloroplasty instead of repairing the hernia. Experimental work by Herron, Thomas, and Merendino (1957) showed a decrease from 72 to 9% in the incidence of oesophagitis after Heller myotomy in dogs. Girvin and Merendino (1958) found that pyloroplasty lessened the incidence of oesophagitis after oesophagogastronomy. The suggestion was that drainage of the retained gastric juice was the important factor. We are not convinced yet that this alone is adequate in man. Franklin at Hammersmith Hospital, London, has been doing a Nissen fundoplication combined with pyloroplasty for the last two years, and is satisfied with the results so far. Wells (1967) described how heartburn was cured by high gastrectomy and Roux-en-Y jejuno-jejunoanastomosis, but he abandoned this procedure because of the high incidence of postprandial dumping. Holt and Large (1961) have applied the same principle in achalasia, both after myotomy and oesophagogastronomy.

We need more knowledge of the physiology of the antroduodenal region and the cardia before deciding on the full comprehensive treatment of hiatus hernia.

Our work has been done on the intact upper gastrointestinal tract using the pyloric regurgitation test which infers that if barium, which is introduced into the duodenum, pours back into the stomach, then duodenal juice should do the same. The evidence that regurgitation does not occur in the normal person is supported by Kilby (1967) who found that in 27 non-dyspeptic controls, 26 had a competent pylorus, as
in our group A. In time, we hope that this test will lead us to the ultimate goal of being able to predict those patients at risk of developing the complications of hiatus hernia and therefore requiring more aggressive treatment at their initial presentation.

We have found in this study that the concurrence of heartburn with an incompetent pyloric sphincter was highly significant, favouring the concept that the presence of duodenal juice in the stomach of such patients is the decisive factor in heartburn.

We would like to thank Mr D. Mearns Milne, of the Department of Thoracic Surgery, Frenchay Hospital, in particular, and the general surgeons in Bristol for additional clinical material. Also we should like to thank Dr J. M. Naish and Mr L. R. Celestin, both of the Gastroenterological Unit of Frenchay Hospital, for their helpful criticism and advice during this study.

REFERENCES


