LETTERS TO THE EDITOR

Recurrent abdominal pain of gastrointestinal disorder

Sir,—We were interested to read the study of van der Meer et al, which demonstrated a high incidence of duodenitis associated with abnormal intestinal permeability to Campylobacter jejuni among children with recurrent abdominal pain.1 We fully agree with their conclusion that duodenitis may play an important part in the pathogenesis of recurrent abdominal pain, although duodenitis rarely occurs without inflammation of other parts of the gastrointestinal tract.2

In a prospective study, duodenal, gastric, and oesophageal biopsies were obtained from 71 children with recurrent abdominal pain, aged 3 to 14 years (mean 8·6) who were undergoing upper gastrointestinal endoscopy for investigation of abdominal pain. We wanted to find out if there were gastrointestinal disorders (for example, duodenitis, gastritis, peptic ulcer) and if there was a relationship between these disorders and Helicobacter pylori. Patients were diagnosed as having recurrent abdominal pain if their symptoms fulfilled the criteria of Apley and Naish: (i) if at least three episodes of pain, (ii) if there was severe pain affecting the child’s activities, (iii) if pain was occurring over a period of not less than three months, and (iv) if attacks continued in the year preceding the examination.

The duration of patients’ symptoms before endoscopy ranged from 3-84 months (mean 16). The pain was periumbilical in 27 patients (38%), epigastric in 26 (37%), hypogastric in five (7%), diffuse in nine (13%), and had other locations in four patients: left upper quadrant (n=2), right upper quadrant (n=1), and right lower quadrant (n=1). The duration of attacks ranged from 1 to less than 5 minutes in eight patients (13%), from 5 to 60 minutes in 36 (51%), and from more than 60 minutes to all day in 27 (38%). The frequency of attacks was daily in 34 patients (46%), more than once a week in 21 (30%), once in every 2-4 weeks in seven (10%), and less than once a month in nine (13%). Forty children (56%) had the attacks of pain in the morning before getting up, 20 (28%) after breakfast, 34 (48%) after all other meals, 20 (28%) before all other meals, 18 (25%) at school, two (3%) immediately after sports, 12 (17%) in the afternoon, at home during play, and 18 (23%) at night, disrupting sleep.

Of these 71 children, 56 had suitable biopsy specimens from the duodenal bulb, 54 from the antrum, 55 from the body, 14 from the cardia, and 59 from the oesophagus. The histological findings in children studied are summarised in the table.

Many of the biopsies studied had an underlying gastrointestinal cause for their complaints: duodenitis (n=7), duodenitis and gastritis (n=13), gastritis (n=19), one of these children had gastric ulcer as well), duodenitis and oesophagitis (n=3), duodenitis plus gastritis and oesophagitis (n=6), gastritis and oesophagitis (n=14), and oesophagitis (n=4). Helicobacter pylori colonisation was found in five of the 71 children studied, using both haematoxylin-eosin stained slides and cresyl fast violet staining.3 Of these five patients, one had H pylori associated antral and body gastritis, and four H pylori antral gastritis only, although both antral and body gastritis were present in three of these four patients.

In the present study, though the presence of H pylori and antral gastritis is recognised, its association with recurrent abdominal pain is not established. These results provide further evidence that H pylori is not a primary pathogen of gastritis and support the hypothesis that H pylori may occasionally colonise gastric tissue as a result of inflammation rather than as a cause of it.

Our histological data show that duodenitis rarely occurs without inflammation of other parts of the gastrointestinal tract and that this condition is frequently associated with gastritis and less frequently with oesophagitis. These results, consistent with those of others,4 raise the possibility that the same aetiological factors that cause gastritis may also be involved in the genesis of duodenitis.5 In a group of 51 children with non-specific abdominal pain, Odera et al found oesophagitis in 11 cases, but the diagnosis was made endoscopically and not confirmed histologically.6 Our data confirm that there is a high incidence of mild oesophagitis among the children studied and the diagnosis was made endoscopically and histologically. These data provide further evidence that there is a significant association of oesophagitis with gastritis or duodenitis. The significance of this association remains to be elucidated.

In conclusion, our data provide strong evidence that there is a gastrointestinal origin of these patients complaints.

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When does slow weight gain become failure to thrive?

Sir,—Edwards et al seek to derive, from the analysis of longitudinal growth data, a ‘logical and generally accepted definition of failure to thrive’ in order to ‘enable vulnerable children to be identified at an early age’.1 We contend that their diagnostic criteria are neither logical, nor are likely to gain general acceptance, for a number of reasons.

Firstly, the authors Smith et al as having demonstrated that the genetic contribution to a child’s weight is ‘greater by the age of 4 to 8 weeks’ (than at birth, presumably). But Smith et al reported nothing of the sort. The paper cited does not even discuss weight gain but, as the title suggests, is concerned with shifting patterns of growth in length. A more relevant reference to the points being made is by Berkley et al who demonstrated that influences upon centile shifting in the first year of life include whether length or weight is being measured, the sex of the child, and whether the shift is towards or away from the 50th centile.2 Edwards et al do not seem to distinguish between weight shifts upward and those shifts downward from the mean.

Diagnostic validity can be viewed as comprising face validity (that is, agreement by clinicians), descriptive validity (that is, a distinctive set of symptoms), and predictive validity (that is, a differentiation on one basis must predict differences in other areas). All three have really demonstrated that infants whose weight persistently deviates (downwards) two or more major centiles from that position reached between four and eight weeks after birth will be rather smaller and lighter in the second year of life than those whose weight has not so deviated. Not a very surprising conclusion. Perhaps a corollary of this finding, a matter not entirely clear from the data presented, is that having deviated downwards in the first year by at least two major centiles an infant is unlikely to deviate up again in the second year. That would be more interesting but it is still not a validation of the concept of failure to thrive.

We would tend to agree with the recommendation of the Joint Working Party on Child Health Surveillance who ‘were not convinced that the advantages conferred by regular weighing justify the resources required or the anxiety generated by inexpert interpretation of growth charts’.3 It is essential to link the identification of a pattern of growth

Histological findings in children studied

<table>
<thead>
<tr>
<th>No (%) of patients</th>
<th>histological finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal duodenal mucosa</td>
<td>27 (48)</td>
</tr>
<tr>
<td>Mild duodenitis</td>
<td>29 (52)</td>
</tr>
<tr>
<td>Normal antral mucosa</td>
<td>16 (30)</td>
</tr>
<tr>
<td>Superficial antral gastritis</td>
<td>38 (70)</td>
</tr>
<tr>
<td>Mild inflammation</td>
<td>24</td>
</tr>
<tr>
<td>Moderate inflammation</td>
<td>6</td>
</tr>
<tr>
<td>Active chronic gastritis</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>55</td>
</tr>
<tr>
<td>Normal body mucosa</td>
<td>26 (47)</td>
</tr>
<tr>
<td>Superficial body gastritis</td>
<td>29 (53)</td>
</tr>
<tr>
<td>Mild inflammation</td>
<td>21</td>
</tr>
<tr>
<td>Moderate inflammation</td>
<td>14</td>
</tr>
<tr>
<td>Active chronic gastritis</td>
<td>14</td>
</tr>
<tr>
<td>Total</td>
<td>64</td>
</tr>
<tr>
<td>Normal oesophageal mucosa</td>
<td>32 (54)</td>
</tr>
<tr>
<td>Mild oesophagitis</td>
<td>27 (46)</td>
</tr>
<tr>
<td>Total</td>
<td>59</td>
</tr>
</tbody>
</table>

Presented in part at the Second Pan Pacific Congress of Paediatric Gastroenterology, Apey et al found First Oceanic Symposium on Paediatric Liver Transplantation in Cairns, Queensland, Australia, August 1990.


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