LETTERS TO THE EDITOR

Recurrent abdominal pain of gastrointestinal disorder

Sir,—We were interested to read the study of van der Meel et al., which demonstrated a high incidence of duodenitis associated with abnormal intestinal permeability to Cr-EDTA 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:1 (i) if there are 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 (48%), 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 pyloric body, 14 from the cardia, and 59 from the oesophagus. The histological findings in children studied are summarised in the table.

Histological findings in children studied

<table>
<thead>
<tr>
<th>Histological findings</th>
<th>No. (%) of patients</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>Normal gastritis</td>
<td>18 (33)</td>
</tr>
<tr>
<td>Mild inflammation</td>
<td>24 (45)</td>
</tr>
<tr>
<td>Moderate inflammation</td>
<td>3 (5)</td>
</tr>
<tr>
<td>Active chronic gastritis</td>
<td>9 (15)</td>
</tr>
<tr>
<td>Normal body mucosa</td>
<td>56</td>
</tr>
<tr>
<td>Superficial body gastritis</td>
<td>26 (47)</td>
</tr>
<tr>
<td>Mild inflammation</td>
<td>25</td>
</tr>
<tr>
<td>Moderate inflammation</td>
<td>5</td>
</tr>
<tr>
<td>Active chronic gastritis</td>
<td>5</td>
</tr>
<tr>
<td>Normal oesophageal mucosa</td>
<td>14 (100)</td>
</tr>
<tr>
<td>Mild oesophagitis</td>
<td>14</td>
</tr>
<tr>
<td>Total</td>
<td>59</td>
</tr>
</tbody>
</table>

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


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 cited 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' (that is, 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 Berkely 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, intuitively understandable, predictive validity (that is, a differentiation on one basis must predict differences in other areas). All the criteria cited above are 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 upwards 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...
termed 'failure to thrive' to some external criterion of developmental disadvantage, be that psychosocial deprivation or organic disease. Otherwise a time honoured preventative activity may become a source of unjustified parental concern.


Dr Edwards, Halse, and Waterston comment:
The three points made by Drs Porter and Skuse cover the generic influence on growth, the diagnostic validity of persistent centile deviation, and the need for additional criteria for psychosocial disadvantage. Definitive answers are not yet available on any of these points. Smith's paper indeed discusses length rather than weight, but the key statement we extracted from this work is that 'Those infants "catching up" after birth usually do so in early infancy (0-3 months) whereas those "dropping down" tend to do so in mid-infancy (3-6 months)'. We have provided the evidence in our paper for the 4-8 week centile being a better predictor of future growth than the birth one.

Our findings show that babies whose weight deviates downwards according to our definition are distinctly different in the second year from babies whose weight does not drop, they are not only lighter, but also shorter and thinner. We therefore believe that we have identified a different population of babies and suggest that these are children who may be regarded as vulnerable and worthy of psycho-social assessment. We believe that these findings demonstrate the value of regular weighing of children. Concerning the meaning of the term 'failure to thrive', we agree with the Lancet that this term, with its connotations of emotional poverty, would be better abandoned and replaced by 'failure to gain weight appropriately'.

Public definitions of failure to thrive which relate to growth below the 3rd or 10th centile do not take into account the growth trajectory, and are clearly unsatisfactory. We think that ours is a considerable advance, if still in need of further refinement.


Baby Check score card

SIR.—The observation by Sinha et al, that nine out of 232 newborn babies showed periventricular echogenicity two hours after birth requires clarification if inferences are to be drawn with regard to the timing of the radiographic result which leads to periventricular leucomalacia.1 Confusion will exist as long as paediatricians continue to use the terms echogenicity, ischaemia, periventricular leucomalacia, and periventricular cysts as though the terms were synonymous.

Echogenicity from the authors' own observations is reversible, as is ischaemia, for at least some patients. Periventricular leucomalacia with or without cyst formation is as permanent as the disability which it may cause. Periventricular leucomalacia is a particular form of cerebral infarction which becomes cystic only after a few days when sufficient numbers of dead cells have been removed for a cavity to be detectable. Precisely how long this interval is before a cyst is seen is something of an imponderable but is probably of the order of 10 days.

It comes as no surprise that there were nine infants whose brain pathology may have been initiated in the intrapartum or immediate postnatal period. Changing the supply of oxygen from placenta to lungs is bound to be intrinsically hazardous. A more interesting question is how many babies sustained cerebral infarction from a hypoxic episode days before the mother's confinement. Cavitition, as opposed to echogenicity, of the infant brain at two hours postnatal age would be convincing in that respect. The reader is not informed.

Dr Morley comments:

I would like to thank Dr Sowden for his interest in Baby Check. I understand his reservations about parents' ability to report their children's symptoms accurately. However, when mothers exaggerate their baby's symptoms one should consider whether they are really capable of reporting the symptoms accurately or whether they perceive that the doctor is disinterested in their baby's illness unless presented with florid and overt symptoms.

Dr Sowden uses mothers' lack of reliability at assessing the amount of fluid on a wash cloth as an indication of their inability to assess their baby's symptoms. This is a notoriously difficult thing to assess and I would be surprised if doctors could do better. I wouldenton that the first thing all doctors should do is ask the mother about the baby's symptoms. It is very difficult to come to an accurate diagnosis without taking a history. However, what is obtained more useful if the mother is asked questions she is likely to be able to answer. For example in Baby Check the mother is not asked the volume of the vomit but, 'Has the baby vomited at all since the last feed after each of the last three feeds?'

The research project, from which Baby Check was developed, set out to find the symptoms (reported by mothers) and signs (seen by the assessors) which could be used to grade the severity of a baby's illness. Interestingly, out of all the possible factors which might be considered important and useful, seven of the 19 factors selected by the analyses were symptoms. Despite any inaccuracy in the way these might have been reported by the mothers they were found to contribute significantly to the assessment of illness. Although we were concerned that mothers might not be able to use Baby Check two field trials showed that they had few problems with the interpretation of the symptoms or signs. Most people who used Baby Check in field studies found it helpful, particularly if they were inexperienced at assessing babies' illnesses. I would like to suggest that Dr Sowden tries Baby Check with a group of mothers who may be pleased to find how well mothers can assess their babies when given a new tool for a difficult task.

Ischaemic brain lesions diagnosed at birth in preterm infants

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