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 ^{51}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 gastroduodenal 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[3]: (i) 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 (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 (25%) 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.

Histological findings in children studied

Histological findings	*No (%) of patients*
Normal duodenal mucosa	27 (48)
Mild duodenitis	29 (52)
Total	56
Normal antral mucosa	16 (30)
Superficial antral gastritis	38 (70)
Mild inflammation	24
Moderate inflammation	5
Active chronic gastritis	9
Total	54
Normal body mucosa	26 (47)
Superficial body gastritis	29 (53)
Mild inflammation	21
Moderate inflammation	3
Active chronic gastritis	5
Total	55
Superficial cardiac gastritis	14 (100)
Mild inflammation	14
Total	14
Normal oesophageal mucosa	32 (54)
Mild oesophagitis	27 (46)
Total	59

Sixty six of the children studied had an underlying gastroduodenal 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). *H pylori* colonisation was found in five of the 71 children studied, using both haematoxylin-eosin stained slides and cresyl fast violet staining.[4] 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,[2] raise the possibility that the same aetiological factors that cause gastritis may also be involved in the genesis of duodenitis.[2]

In a group of 51 children with non-specific abdominal pain, Oderda *et al* found oesophagitis in 11 cases, but the diagnosis was made endoscopically and not confirmed histologically.[5] 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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1 Van der Meer SB, Forgett PP, Arends JW. Abnormal small bowel permeability and duodenitis in recurrent abdominal pain. *Arch Dis Child* 1990;**65**:1311–9.
2 Beck IT. Duodenitis. In: Bockus HL, ed. *Gastroenterology*. Vol II. 4th Ed. Philadephia: Saunders, 1985:1403–21.
3 Apley J, Naish N. Recurrent abdominal pains: a field survey of 1000 school children. *Arch Dis Child* 1958;**33**:165–70.
4 Burnett RA, Brown IL, Findlay J. Cresyl fast violet staining method for campylobacter like organisms. *J Clin Pathol* 1987;**40**:353.
5 Oderda G, Vaira D, Holton J, Dowsett JF, Ansaldi N. Serum pepsinogen I and IgG antibody to Campylobacter pylori in non-specific abdominal pain in childhood. *Gut* 1989;**30**: 911–2.

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 cite 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 Berkey *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.[3] 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 they have really demonstrated is 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'.[4] 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 preventive activity may become a source of unjustified parental concern.

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1 Edwards AGK, Halse PC, Parkin JM, Waterston AJR. Recognising failure to thrive in early childhood. *Arch Dis Child* 1990;65:1263–5.
2 Smith DW, Truog W, Rogers JE, *et al.* Shifting linear growth during infancy: illustration of genetic factors in growth from fetal life through infancy. *J Pediatr* 1976;89:225–30.
3 Berkey CS, Reed RB, Valadian I. Longitudinal growth standards for preschool children. *Ann Hum Biol* 1983;10:57–67.
4 Hall DMB, ed. *Health for all children: a programme for child health surveillance.* Report of the Joint Working Party on Child Health Surveillance. Oxford: Oxford University Press, 1989.

Drs Edwards, Halse, and Waterston comment:
The three points made by Drs Porter and Skuse cover the genetic 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 "lagging 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 centile.

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 deviate: 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 psychosocial 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'.[1]

Previous 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.

1 Anonymous. Failure to thrive revisited. *Lancet* 1990;336:662–3.

Baby Check score card

SIR,—The series of articles by A J Thornton, C J Morley, and S J Green *et al* on the Baby Check score card make very interesting reading and represent a timely attempt to provide parents with a diagnostic tool to grade their infant's severity of illness.[1–4]

As a general practitioner I must admit to some reservations about many parents' capacity (particularly with first children) to document accurately their children's symptoms. I am frequently presented, as are my colleagues, with children whose parents cheerfully report that they have vomited everything they have been fed for the last 48 hours or longer, haven't passed urine for the last 24 hours, and are drowsy all the time ('He definitely isn't himself, doctor'!?). A set of symptoms belied by the fully hydrated and cheerful infant sitting in a wet nappy in the surgery ('He must have just done it').

This lack of reliability in parents' impressions of their children's symptoms is supported by a letter by Francy Pillo-Blocka *et al* from the Hospital of Sick Children in Toronto.[5] They reported on the subject of mothers of infants with gastro-oesophogeal reflux and their estimates of the quantity of fluid spilled on a baby's wash cloth. A spillage of 5 ml in volume produced a mean estimate of spillage of 35 ml (range 3–120 ml) and a 10 ml volume produced a mean estimate of spillage of 77 ml (range 7–240 ml). Of the 58 mothers tested, only one accurately assessed both volumes and the result was independent of education status or age of the mother. The authors advised caution in accepting parental impression of vomiting as a result.

I accept this finding only affects a small subsection of the Baby Check, but I would be very concerned about the widespread application of the score card before more work is done on the accuracy of parental assessment of individual signs and symptoms included in the score card.

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1 Morley CJ, Thornton AJ, Cole TJ, Hewson PH, Fowler MA. Baby Check: a scoring system to grade the severity of acute systemic illness in babies under 6 months old. *Arch Dis Child* 1991;66:100–5.
2 Thornton AJ, Morley CJ, Green SJ, Cole TJ, Walker KA, Bonnett JM. Field trials of the Baby Check score card: mothers scoring their babies at home. *Arch Dis Child* 1991;66: 106–10.
3 Morley CJ, Thornton AJ, Green SJ, Cole TJ. Field trials of the Baby Check score card in general practice. *Arch Dis Child* 1991;66: 111–4.
4 Thornton AJ, Morley CJ, Cole TJ, Green SJ, Walker KA, Rennie JM. Field trials of the Baby Check score card in hospital. *Arch Dis Child* 1991;66:115–20.
5 Pillo-Blocka F, Jurimae K, Khoshoo V, Zlotkin S. How much is 'a lot' of emesis? *Lancet* 1991; 311:12.

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 incapable 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.

When assessing a baby's illness 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, the information obtained is 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 least half the 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 a baby's 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 our 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 his patients. I think he might be pleasantly surprised 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

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 insult 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 or ischaemic episode days before the mother's confinement. Cavitation, as opposed to echogenicity, of the infant brain at two hours postnatal age would be convincing in that respect. The reader is not informed.