Abnormal mucosa in gastroenteritis

Sir,

We thank Drs. Walker-Smith and Rossiter (1973) for their comments regarding abnormal mucosa in gastroenteritis, but feel constrained to reply to one or two points at this late stage.

They reject the statement that single biopsy specimens provide reliable information about the degree of damage to duodenal mucosa on the basis that 3 cases biopsied serially showing 'a consistent trend towards normality' is insufficient evidence. This may be a valid point and we did point out in the article (Barnes and Townley, 1973) that 'multiple biopsies ... would be required to exclude the possibility of patchy damage in the duodenum'. Walker-Smith's (1972) postmortem studies of small bowel from children with abnormal mucosa refer only to gross morphology. The grading of mucosal damage in our study included degree of infiltration of the lamina propria and epithelial damage, which is probably more relevant when disaccharidase depression is being considered.

Lack of correlation between severity of biopsy abnormality and clinical status may not be related only to incorrect histological sampling, as Walker-Smith and Rossiter suggest. Surely biochemical changes are likely to be more relevant in a situation where fluid and electrolyte loss is the prime mechanism causing symptoms?

If the patchy nature of the lesion is the cause of lack of correlation of persisting sugar intolerance with disaccharidase depression, why is the sugar intolerance almost completely confined to the infants under 6 months of age? Histological damage was no more severe in this group. One would not expect biopsies to sample areas of lesser damage in the group under 6 months and areas of greater damage in the 6- to 12-month group.

It should be mentioned that approximately 75% of patients had Clinitest-positive stools (>1%?) in the first 48 hours of the illness. The article was referring, however, to persisting sugar intolerance lasting more than 1 week.

While accepting some of the points made by your correspondents as worthy of consideration, we feel that their own explanations do not satisfactorily answer the questions posed by our study.

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REFERENCES


Dr. Walker-Smith replies as follows.

I am most grateful for the opportunity to comment upon Drs. Barnes and Townley's reply to our letter concerning their most important study of the duodenal mucosa in infants with gastroenteritis (Barnes and Townley, 1973).

In our letter (Walker-Smith and Rossiter, 1973) we hoped to emphasize that it is not just the patchy nature of the abnormal mucosa in the duodenum which may account for the lack of correlation between the biopsy findings and persisting sugar intolerance in children with gastroenteritis. Rather, we also referred to evidence that it is the extent of the abnormal mucosa along the small intestine which is likely to be a critical factor in determining the severity and persistence of clinical sugar intolerance, in a manner analogous to coeliac disease where the extent of the mucosal abnormality appears to determine clinical severity. This fact was indeed commented upon by Barnes and Townley in their original paper.

It is possible that infants under 6 months with gastroenteritis often may have more extensive mucosal damage along the length of the small intestine than older children, and in support of this are observations made in a necropsy study of 10 children dying from enteritis or enterocolitis (Walker-Smith, 1972). 2 out of 4 children under the age of 6 months had a very lengthy mucosal abnormality, whereas all 6 children aged 6 months to 11 years had far less extensive mucosal damage along the length of the small intestine.

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REFERENCE

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