variety of chronic diarrhoeas, particularly coeliac disease.

(2) We agree that a 0.5 g/kg dose of lactose may be inappropriately low for the diagnosis of lactose intolerance. As we explained in our paper, we chose this dose to avoid precipitating marked fluid loss in patients who might be very sensitive to a lactose load. When this proved not to be the case we increased the load to 2 g/kg.

(3) The 40% of our patients who showed a positive breath hydrogen test after a 2 g/kg lactose load were children with acute gastroenteritis, and cannot be compared directly with their quoted figure of 38% in post-gastroenteritic children.

(4) They suggest that all of our patients with high breath hydrogen levels should have been treated with a lactose-free diet. We preferred to treat the patient rather than the test results, and found that all our patients rapidly recovered despite the reintroduction of milk into the diet. Again, we cannot comment on their unpublished series, but would suggest that in the context of a short-lived illness like gastroenteritis clinical response coincident with the use of a lactose-free diet could not be taken as evidence that the response was due to the treatment. We feel it is essential to draw the distinction between biochemical evidence of lactose malabsorption, as shown by the hydrogen breath test, and clinical lactose intolerance. As we pointed out, only one of our patients had clinical evidence of lactose intolerance, and his breath test was normal.

(5) We agree that children may be less tolerant of lactose in water than in milk, and that false-negative responses may be associated with treatment with antibiotics. Neither of these comments appears relevant to our paper. The children who developed positive breath hydrogen tests after a load were asymptomatic and therefore not intolerant of lactose despite their positive test, and the single false-negative result we had was in a patient who had not recently been given antibiotics.

(6) The work of Solomons *et al.*⁵ suggesting reduced breath hydrogen responses in children with active diarrhoea had not been published when our study was performed. However it does not detract from our findings, but possibly enhances them, suggesting that the hydrogen breath test is not appropriate in acute gastroenteritis in young children. Fasting young infants with acute gastroenteritis overnight, as suggested by Robb and Davidson, is not a practical or appropriate form of clinical manage-

ment, and could not be justified even if it were likely to improve the accuracy of this test.

Neonatal gallbladder distension

Sir,

The recent description of 8 cases of neonatal gallbladder distension¹ and the comment on the rarity of reported cases prompts us to report our own recent experience of 2 cases in which gallbladder distension occurred in circumstances not previously described.

In the first, gallbladder distension was detected clinically in the third week of life and was confirmed by ultrasound examination. This infant was greatly growth retarded, had suffered severe perinatal asphyxia, developed repeated convulsions for which a period of ventilation was necessary, and also had a hypoxic cardiomyopathy leading to cardiac failure. Immediately before digitalisation the liver was enlarged to 6 cm. After a satisfactory clinical response with reduction in hepatomegaly the gallbladder was found to be palpable. At this point the baby was off ventilator support, had no evidence of infection, and was clinically in a recovery phase. The gallbladder diminished in size during a period of a week.

In the second case an enlarged gallbladder was detected on palpation at the end of the third week. No ultrasound confirmation was obtained. This baby had evidence suggesting a patent ductus arteriosus and was bordering on cardiac failure with tachypnoea and a liver enlarged to 4 cm. No treatment was necessary and by the next fortnight the gallbladder was impalpable and the liver reduced to normal. Neither infant was jaundiced.

It is interesting to postulate that in each case gallbladder distension was a consequence of cystic duct obstruction by hepatic venous engorgement.

Reference

- ¹ Peevy K J, Wiseman H J. Gallbladder distension in septic neonates. *Arch Dis Child* 1982; **57**: 75-6.

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