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-gastroenteritis 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Again, we cannot comment on their unpublished series, 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 
ilness 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 intol-
erance. 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. 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 gastro-
enteritis 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 re-
petuted convulsions for which a period of ventilation was 
necessary, and also had a hypoxic cardiomyopathy 
leading to cardiac failure. Immediately before digitalisa-
tion 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 de-
tected 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 gall-
bladder distension was a consequence of cystic duct 
obstruction by hepatic venous engorgement.

Reference

1 Peevy K J, Wiseman H J. Gallbladder distension in sep-

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