Correspondence

Hydrogen breath test in gastroenteritis

Sir,

Having been concerned in breath hydrogen testing during a 3-year period (950 patients), we were surprised at the findings of Gardiner et al.1 and in particular at such statements as ‘the hydrogen breath test was not an appropriate technique for detecting it’ (lactose intolerance in gastroenteritis). Their assessment of the usefulness of this test differs considerably from our own, and others,2-4 and the reasons for this need to be determined to restore clinicians’ confidence in the hydrogen breath test.

The test dose of lactose we use, and the one most commonly used by laboratories, is 2 g/kg, and closely approximates physiological conditions. Therefore test doses of 0.5 g/kg may be inappropriately low when attempting to assess concordance of the hydrogen breath test and clinical results. Using Gardiner’s criteria for an abnormal breath hydrogen rise, 8 (40%) of 20 post-gastroenteritis patients given 2.0 g/kg lactose had an abnormal breath hydrogen rise in his series. This figure is similar (38%) to our findings soon to be published.

Differing conclusions from the same data seem to be related to the interpretation of clinical intolerance. We often do not find symptoms of intolerance in the 24 hours after the test dose of lactose, but in every case in our series in which lactose malabsorption was demonstrated by hydrogen breath tests (38 patients), the patients responded positively to lactose withdrawal. A lack of symptoms during and immediately after breath hydrogen testing, but response to a lactose-free diet, has been reported elsewhere.3 It appears that none of the 8 patients with abnormal breath tests in Gardiner’s series was given a trial of a lactose-free diet in order properly to assess the clinical response.

There is the potential for a false-positive breath hydrogen test as outlined by Solomons et al.,5 whereby lactose in whole milk may be better tolerated than lactose in water, presumably due to the effects of differing gastric emptying rates, but we have not encountered this problem. We agree that false-negative results can be obtained, but in our experience this is almost always due to either recent antimicrobial therapy, the effects of which can persist for at least a month, or to some mechanical failure of the breath test—for example vomiting of the test dose or premature termination of sampling.

It is worth remembering the importance of intraluminal pH and its effect on hydrogen production as outlined by Pernan et al.6 This may be part of the mechanism responsible for the reportedly lowered hydrogen responses in children with active diarrhoea,7 although with our methodology8 and by fasting the patients overnight, we did not find this effect.9

We believe that the hydrogen breath test in the assessment of carbohydrate malabsorption is valuable provided the potential for false-negative and false-positive results is appreciated, and care is taken to ensure that the methodology is designed to circumvent these pitfalls. We have found excellent correlation between breath hydrogen tests and the effects of dietary intervention, and as a consequence, have had to cope with an ever increasing demand on this service by informed clinicians.

References


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Dr Tarlow and co-workers comment:

Dr Robb and Dr Davidson make many points and it seems reasonable to deal with each separately:

1. Our assessment of the value of the breath hydrogen test differs from theirs and that of others. They have not published their own results and it is therefore not possible for us to comment on them, but none of the three references they quote deals with the role of breath hydrogen testing in the course of acute gastroenteritis. Two use it in the diagnosis of hyposucrasia (one in adults and one in children) and the third discusses its role in a
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. 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 management, 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 distension\(^1\) 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


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