It is likely that the babies' symptoms were mainly due to the caustic action of cetrimide 1%^{2,3}; fortunately contact with the mixture was necessarily very brief as it is irritant with an unpleasant taste leading to immediate attempts to spit it out. Treatment should consist of avoiding gastric lavage; whole egg mixed in milk can be given to neutralise chlorhexidine, but there is no specific antidote to cetrimide. Four affected babies were given hydrocortisone and ampicillin sodium intramuscularly, and two who were irritable were given intramuscular phenobarbitone.

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Mildly anaemic toddlers respond to iron

Sir,

Some general readers might be tempted to conclude from the paper by Parks *et al* that, after oral iron treatment, a change in mean haemoglobin concentration from 108 g/l to 126 g/l, mean corpuscular volume from 64.8 fl to 69.3 fl, and mean serum ferritin from 8.3 µg/l to 14.8 µg/l constitutes 'iron deficiency, end of haematological story'.¹ This will of course be far from the case in some Asian toddlers.

Haemoglobin A2 and F determination will not exclude α thalassaemia, which occurs with high frequency in Asian populations. A mean corpuscular volume of 69.3 fl is below the 3rd centile quoted by Dallman and Siimes,² whose reference group 'may well include iron deficient children' as Parks *et al* point out themselves. From the data they present, some of the children could well have α thalassaemia, which can coexist with iron deficiency. At the very least their families could be investigated. What diagnoses did they make in those whose mean corpuscular volume remained substantially lower than the mean of 69.3 fl (the SD was 3.9 fl) after iron treatment?

References

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Effect of misoprostol on fat malabsorption in cystic fibrosis

Sir,

The improved fat malabsorption shown in the patients with cystic fibrosis reported by Robinson *et al* is interesting and significant but the data presented are inadequate to make a judgment as to the drug's place in patient management.¹ A study of this type should include the number of pancreatin (Pancrease, Ortho-Cilag) capsules taken by the patients. The fact that only three patients achieved better than 85% absorption and six suffered from 'chronic abdominal pain' suggests that their malabsorption was inadequately controlled due to inadequate dosing with Pancrease; perhaps an increase in Pancrease would have been more appropriate than the addition of misoprostol, yet another medication for their already overloaded treatment regime.

If the absolute values of intake, output, and enzyme dose are not included it is impossible to confirm that these patients were receiving too small a dose of enzymes to control their malabsorption at the start of the study as appears to be the case.

It would be unfortunate if physicians responsible for patients with cystic fibrosis were to add misoprostol rather than treating their patients' malabsorption correctly—that is, increasing the dose of modern pancreatic microsphere preparation until symptoms are controlled, then checking the faecal fat output and intake. If the fat absorption is less than 90% the dose of enzyme should be increased and the process repeated. The manufacturer's literature is misleading as many patients require 6-10 capsules per meal of the microsphere preparations available in the United Kingdom. It would then be of interest to know if the addition of misoprostol to the treatment of patients with cystic fibrosis having optimal control of their malabsorption by conventional preparations would further improve their fat absorption.

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Drs Robinson, Sly, and Smith comment:

We thank Drs Littlewood and Kelleher for their comments but it would appear that they have missed the main point of our paper, and as such our suggested role for misoprostol in the care of patients with cystic fibrosis. The idea that a patient with cystic fibrosis who does not have normal fat