sorry that Dr Goel appeared to concur, albeit reluctantly.

Certainly the management of diarrhoea in developing countries will not be the same as in developed countries, but the central element is still fluid therapy. Dr Karan and Dr Limaye offer no evidence that Lomotil will reduce the morbidity of this severe disease, nor even that it will shorten its course. Fluid loss is of much greater importance than intestinal motility and the lack of efficacy of Lotomil in reducing frequency of bowel movements and the water content of the stools in acute childhood diarrhoea has been documented.³ The essential role of oral hydration in its management in all countries has been widely discussed.⁴⁻⁸ It is strange that Dr Karan asserts 'where children have no access . . . to fluid therapy, symptomatic control becomes important', when surely a home-based electrolyte mixture is more readily available, and is safer and more effective than Lomotil. A glucose electrolyte solution can be made up correctly by mothers at village level,⁹ and has a striking effect on mortality.¹⁰ In this climate of opinion propaganda in favour of symptomatic control of a killing disease seems at the least unwise.

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Dr Goel comments:

There is no disagreement that the mainstay of treatment of childhood diarrhoea in any part of the world is correction of dehydration and electrolyte acid-base status and not the use of an antidiarrhoeal drug such as Lomotil.

Of course, as indicated by Dr Waterston, any mother, whether from a developing or a developed country, can correctly make a glucose-electrolyte solution for oral therapy. However, one should remember that oral therapy

is effective only in a child with slight or moderate dehydration. In a severely dehydrated child intravenous fluid therapy is mandatory, especially if he is not retaining fluids orally, and such therapy may often be unavailable in more remote areas of the tropics.

As far as the use of Lomotil in tropical diarrhoea is concerned, I merely acquiesced in the statement of Dr Karan and Dr Limaye on their experience.

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Dr Karan comments:

In response to Dr Waterston's letter, I do not think that there is any argument about fluid therapy being an important element in managing diarrhoea, but I am equally disturbed at his suggestion that Lomotil has little place in managing tropical diarrhoea.

Lomotil is no substitute for oral fluids, but it is of symptomatic value in decreasing diarrhoea at least in the tropics, whether children have been given fluids or not. Field¹ (cited by Dr Waterston himself) stated 'Oral glucose-electrolyte therapy, however, does not decrease diarrhoea', and our own study² (cited in earlier correspondence) showed that in a control group given fluids alone the diarrhoea was not controlled as effectively as in the three groups given varying doses of Lomotil in conjunction with fluids. The Wilcoxon method of analysis and the permutation test showed that all three Lomotil groups in our study had diarrhoea for a significantly shorter time than the group given fluids alone ($P < 0.001$).

Other workers in India,³⁻⁶ and Africa⁷ have shown that Lomotil reduces the frequency of bowel movements (in many cases within 24 hours) in tropical diarrhoea and is safe if used as recommended, despite Portnoy's evidence to the contrary,⁸ cited by Dr Waterston. Incidentally, that study showed 12 of 39 cases to be of parasitic origin, and in 27 cases no stool culture results were available. Lomotil can hardly be of value in parasitic infections. Portnoy himself stated '... it is difficult to evaluate the meaning or usefulness of stool water content determinations in the study of diarrhoeal illness'.

Finally, I should like to stress that we do encourage the use of oral fluids, and are fully aware of their beneficial effect on mortality. However when oral fluids are made up in rural areas there is a great danger that the solution will be too concentrated or too diluted, as outlined by Sedgwick and Cutting.⁹

We recommend that under such conditions Lomotil should be given judiciously in conjunction with fluids. We know that the drug is no panacea but it plays a valuable role in acute nonspecific diarrhoea, at least in the tropics.

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Dominant Sotos's syndrome

Sir,

Since the first report of a syndrome of cerebral gigantism in childhood by Sotos *et al.*¹ many similar cases have been described. Sotos *et al.*² reviewed 105 other cases as well as 10 of their own. The condition was generally sporadic, but some reports indicated a genetic basis. Until now, there have been two reports of 4 families³⁻⁴ in which a dominant mode of inheritance was shown, considered most likely to be autosomal dominant, although dominant X-linked inheritance was not excluded. We report here the fifth example of dominant Sotos's syndrome.

We recently examined a 4-year-old girl because of speech delay and large body size—her height was over 97th centile, weight on 90th centile, and head circumference on 97th centile. Examination showed other signs of cerebral gigantism—such as a high arched palate, a high bossed forehead, hypertelorism, a flat nasal bridge, and prognathism. She had large hands and her shoes were size 3, the size worn by an average 10-year-old child. Bone age was advanced at 5.75 years. Banded karyotype was normal. She had slight mental retardation.

The mother was also examined as she, too, was large. Her height, weight, and head circumference were all above the 90th centiles, she had large hands and feet, pronounced prognathism, a very high palate, coarse facial features, and a deep hoarse voice. She had a full scale IQ of 85. Banded karyotype was normal. There was some enlargement of the lateral ventricles shown by computerised tomography. We consider that she has Sotos's syndrome. There was no evidence of gigantism in any male relative.

For both mother and daughter, the differential diagnosis was gigantism due to general genetic endowment, or gigantism due to Sotos's syndrome. Apart from the gigantism, both of them have many of the clinical signs of Sotos's syndrome. The diagnosis is more obvious in a child than an adult. This point is exemplified by the fact that until now the syndrome in adults has been detected only through the investigation of their children,⁴ so that the pediatrician plays an important role in detecting adult cases.

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