Management of gastroenteritis

SIR,—Professor Walker-Smith's recommendations on the dietary management of infantile gastroenteritis are commendably clear.¹ Unfortunately they do not correspond to the advice in the accompanying article by Jenkins and Ansari, which takes general practitioners to task for 'inappropriately' continuing milk feeds (this category contributed half of the inappropriate management total).² Before we chide primary care doctors for their deficiencies we should examine the consistency of our own advice. Official WHO guidance on diarrhoea management is to continue feeding in a breast fed baby, and for bottle fed infants to continue the usual formula every three hours (except during the three to six hour rehydration period),³ yet paediatricians and major textbooks commonly advise the withdrawal of milk feeds and food for a temporary period.

It is, therefore, hardly surprising that general practitioners do not show a uniform approach. In addition, none of the three papers about gastroenteritis management in the same issue give clear guidance as to whether solid foods should be stopped. Jenkins and Ansari indicate that normal diet should be ceased for 24 hours, but say that this regimen is under debate. WHO advice is that 'food intake should never be restricted during or following diarrhoea'.³

It is time that our own guidance is brought in line with that adopted in developing countries, and we recommend continued milk and solid feeding in the outpatient management of gastroenteritis.

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1 Walker-Smith JA. Management of infantile gastroenteritis. *Arch Dis Child* 1990;65:917-8.

2 Jenkins HR, Ansari BM. Management of gastroenteritis. *Arch Dis Child* 1990;65:939-41.

3 World Health Organisation. *Programme for the control of diarrhoeal diseases. A manual for the treatment of diarrhoea*. Geneva: WHO, 1990.

Professor Walker-Smith comments:

It is certainly difficult for general practitioners to have a uniform approach when there is at present no general agreement among paediatricians concerning dietary management of gastroenteritis in infancy. Dr Waterston cites WHO advice that has been aimed at developing communities. In such communities nutritional status is often already seriously compromised whereas in developed communities it is usually not. Furthermore gastroenteritis is usually less severe in the latter. While there is general agreement that breast feeding should continue in infants who develop gastroenteritis with additional oral rehydration therapy as appropriate, there is no such general agreement about bottle feeding. There is, for example, no satisfactory published study from the UK comparing the regimen of 24 hours oral rehydration solution alone and no feeding to a regimen where bottle feeding is continued unrestricted. Whereas there are studies to show that after such a 24 hour period there is no advantage for regrading rather than returning to full strength feeds. Until such a study is published most paediatricians would

agree with Jenkins and Ansari, that in this country the normal diet should cease for 24 hours. They are correct to say that this regimen is under debate.

Drs Jenkins and Ansari comment:

We thank Dr Waterston for highlighting an important area that needs clarification. We would wish to remind him that, at the time we carried out our study, the recommendations regarding the dietary management of gastroenteritis in the UK were clear and unequivocal and were published by Professor Walker-Smith and his colleagues.¹ However, as we clearly stated in our paper, the guidelines may well require updating in the light of further experience. We fully endorse the WHO guidelines (1990) for the developing world where cessation of feeding may mean the difference between life and death, although these guidelines are not necessarily appropriate for the developed world where the disease is relatively mild. Until clear evidence is available from controlled studies that similar guidelines are to be recommended for the UK we will continue our present practice of providing oral rehydration solution and stopping milk feeds and solids for a period of time (6-24 hours) after which full strength feeds and solids may be restarted.

1 Wharton BA, Pugh RE, Taitz LS, Walker-Smith JA, Booth IW. Dietary management of gastroenteritis in Britain. *BMJ* 1988;296:450-2.

Drs Mandal and Dunbar comment:

Dr Waterston has taken us to task for our failure to give clear guidance to general practitioners on whether solid foods should be stopped temporarily during the initial phase of gastroenteritis management. However, our paper was not about the general aspect of management of gastroenteritis. We were looking at a very specific issue—that is, whether the currently widely given advice on graded reintroduction of an infant's usual feeds are necessary for children below the age of 6 months. Thus our study was specifically designed to establish whether there is any advantage in such regrading, with particular reference to recurrence of diarrhoea, effect on weight, and duration of hospital stay. We did not find any advantage in the traditional approach of regrading patients below the age of 6 months with gastroenteritis. We also found that children with lactose intolerance can be satisfactorily managed with a lactose free soy based preparation. Professor Walker-Smith has expressed reservation about this approach because of the risk of soy protein allergy but the incidence of soy intolerance has been no more than 1% or so in our unit despite its use over many years.

We do not accept that there has been any confusion over the advice given to British doctors over the general management of gastroenteritis. The recommendations on the dietary management of gastroenteritis have always included withdrawal of the infant's usual formula feeds and solids and their replacement with an oral rehydration solution for 24 hours after which the feeds should be reintroduced. In developing countries where multiple attacks of gastroenteritis are common, often leading to malnutrition, continued milk and solid feeding has been practised with no apparent ill effects, but as in the

case of what is the most appropriate sodium concentration and carbohydrate content for the use in developed countries, this also needs to be examined in properly designed studies before there is uncritical adoption of continued milk and solid feeding approach in the management of gastroenteritis in Great Britain.

Helicobacter pylori

SIR,—The regular review on *Helicobacter pylori* makes interesting reading.¹ However the treatment recommended for duodenal ulcer disease is confusing. Colloidal bismuth subcitrate is no longer recommended in either the *British National Formulary* (September 1990) or the manufacturer's data sheet for use in children.

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1 Drumm B. *Helicobacter pylori*. *Arch Dis Child* 1990;65:1278-82.

Professor Drumm comments:

Dr Campbell's letter highlights a major area of confusion in relation to the treatment of gastritis and duodenal ulcer disease using bismuth containing compounds. It is true that the *British National Formulary* and the manufacturer's data sheet do not recommend colloidal bismuth subcitrate for use in children. The formulary requires that pharmacokinetic data for children must be available if the drug is to be recommended for use in this age group. This information is not available.

There is, however, no evidence that bismuth subcitrate has adverse effects in children other than those already described in adults. These include encephalopathy, which is reversible after withdrawal of the drug, and acute renal impairment after ingestion of an overdose of this drug.^{1 2}

Colloidal bismuth subcitrate is certainly not contraindicated in children. A more significant risk during the treatment of *Helicobacter pylori* gastritis may be the development of pseudomembranous colitis as a result of the use of an antibiotic in the treatment regime. We, and others, as noted in the review, have used bismuth preparations in children. Adverse reactions to bismuth used in the treatment of *H pylori* associated gastritis have not been reported in the studies published to date.

1 Hudson M, Ashley N, Mowat G. Reversible toxicity in poisoning with colloidal bismuth subcitrate. *BMJ* 1989;299:159.

2 Taylor EG, Klenerman P. Acute renal failure after colloidal bismuth subcitrate overdose. *Lancet* 1990;335:670-1.

A clinical trial of two parenteral nutrition solutions in neonates

SIR,—Professor McIntosh and Dr Mitchell, while hypothesising that the use of Vamin 9 for parenterally fed preterm infants may have contributed to death through high plasma concentrations of aromatic amino acids,¹ have

also illustrated some of the difficulties in attempting comparisons of different amino acid solutions.

We have previously drawn attention to abnormal plasma amino acid profiles in Vamin 9 fed infants.² We considered this to be a reflection of the amino acid composition of Vamin 9 rather than an idiosyncratic reaction, as the pattern occurred consistently, and was much less evident when a modified solution containing less phenylalanine was used.³ One factor which McIntosh and Mitchell do not address in detail, but which may have considerable influence on plasma amino acid profiles, is the amount of enteral nutrition being given. In our original index case of hyperphenylalaninaemia,² small volumes of breast milk given as a supplement to parenteral nutrition produced large changes in plasma phenylalanine. The patients of McIntosh and Mitchell received either breast milk or preterm formula in addition to their intravenous nutrition. In fact, by day 5 of the study, the only day during parenteral nutrition on which plasma amino acid profiles were measured, the figures given for mean enteral feed in both infants receiving Vamin 9 and those fed with MB233G represent approximately 60% of the total nitrogen intake. It is therefore difficult to see how valid conclusions can be drawn about the suitability of either amino acid solution as a parenteral nitrogen source.

Although 68 patients were recruited to the study, some died, some were excluded, and some had inadequate blood sampling or blood taken on the wrong day. As a result, the data derive solely from a single plasma amino acid analysis, performed on only seven patients in the Vamin 9 group and five in the MB233G group. Moreover, individual amino acid concentrations are reported as mean (SD) suggesting normal distribution of data. However, the fact that -2SD would give a negative value for 50% of the amino acids measured indicates non-parametric distribution of results, consistent with similar studies.³ It would therefore be interesting to be able to compare mean (after log transformation to 'normalise' the data) and ranges of individual amino acids.

Finally, single measurements of plasma amino acid profiles may not allow adequate comparison of different solutions as plasma concentrations can vary considerably. This is illustrated by the following plasma concentrations of phenylalanine and tyrosine measured every two weeks in a child with gastroschisis receiving total parenteral nutrition with a constant nitrogen intake: phenylalanine 149, 500, 200, 49, 63, 983, and 260 $\mu\text{mol/l}$ and tyrosine 95, 60, 80, 58, 86, 86, and 145 $\mu\text{mol/l}$.

The theoretical basis for the formulation of MB233G is of interest, but it remains to be seen whether or not this new solution offers any advantages over alternative preparations.

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- McIntosh N, Mitchell V. A clinical trial of two parenteral nutrition solutions in neonates. *Arch Dis Child* 1990;65:692-9.
- Puntis JWL, Edwards MA, Green A, Morgan I, Booth IW, Ball PA. Hyperphenylalaninaemia in parenterally fed newborn babies. *Lancet* 1986;ii:1105-6.
- Puntis JWL, Ball PA, Preece MA, Green A, Brown GA, Booth IW. Egg and breast milk based nitrogen sources compared. *Arch Dis Child* 1989;64:1472-7.

Professor McIntosh comments:

Drs Puntis and Booth suggest that the enteral component of nutrition may be important in the development of hyperphenylalaninaemia. They point out that on day 5 both groups of infants in our study received approximately 60% of their nitrogen intake enterally. We agree with this, as at this time the intravenous nitrogen intake was about 40% of the total! The point of difference in our two groups was the composition of the intravenous amino acid mixtures and it would seem therefore to be a 'non-sequitur' to suggest the high concentrations of phenylalanine, tyrosine, serine, proline, and asparagine were due to the enteral intake.

There were in fact 16 and 14 patients with 'good' samples for amino acid analysis taken on day 5—see original table 5. We accept that the results were not in a Gaussian distribution—this is why Wilcoxon's signed rank test was used when comparing amino acid concentrations the two groups.

We note the information in the last paragraph which we find very interesting.

We believe that the apparent ability of babies given MB233 to keep their plasma aminogram in the reference range of cord blood aminograms makes it an attractive alternative to Vamin 9 glucose where the aminograms at five days approached the values of infants with untreated phenylketonuria and hereditary tyrosinaemia.

Virological investigations of acute encephalopathy in India

SIR,—Rabies should be considered in the differential diagnosis of acute encephalopathy in children living in endemic areas. It may present as a non-specific encephalitis without the pathognomonic features such as hydrophobia and a clear history of exposure is not always present. It was therefore surprising that it received no mention in the series of Kumar *et al*¹—could it have accounted for some of the cases among the 40% in whom they found no cause?

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- Kumar R, Mathur A, Kumar A, Sethi GD, Sharma S, Chaturvedi UC. Virological investigations of acute encephalopathy in India. *Arch Dis Child* 1990;65:1227-30.

Dr Kumar comments:

Rabies is endemic in the Indian subcontinent. It produces an acute encephalitic illness usually with a pronounced psychological component. We agree that occasionally none of these pathognomonic features may be found and therefore it may account for a few of the fatal 'unexplained encephalopathies' in this study. Although we have discussed other viral encephalitides as a cause in this group we have not specifically mentioned rabies. We therefore appreciate Dr Hall's comment.

SIR,—Dr Kumar and his colleagues in reporting their investigations into the cause of acute encephalopathy in children in Lucknow have not considered the possibility of cerebral cysticercosis in their differential diagnosis.¹

Cysticercosis in India is not uncommon² and parenchymal involvement, the most common form in children,³ may present acutely. Acute diffuse parenchymatous disease presents with generalised cerebral oedema often severe enough to cause an acute rise in intracranial pressure with deterioration of consciousness and cerebral shifts. Acute focal parenchymal disease presents with localised patchy oedema often resulting in convulsions.

The diagnosis of cerebral cysticercosis may present difficulties. Serology is beset with the problems of poor sensitivity and specificity and cerebrospinal fluid changes are often non-specific and non-diagnostic. Computed tomography has improved diagnostic accuracy but it not always readily available in India. Where there has been the opportunity to perform enhanced computed tomography on Indian patients presenting with seizures, lesions suggestive of cysticercosis have been reported in 26% of cases,⁴ excision biopsy confirming the diagnosis in a further series of cases.⁵

A diagnosis of cerebral cysticercosis should be considered in all children presenting with 'acute encephalopathy' in India.

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- Balasubramaniam V, Kanaka TS, Ramamurthi B. Cerebral cysticercosis in India. *Int Surg* 1971;56:172-81.
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- Wadia RS, Makhale CN, Kelkar AV, Grant KB. Focal epilepsy in India with special reference to lesions showing ring or disc-like enhancement on contrast computed tomography. *J Neurol Neurosurg Psychiatry* 1987;50:1298-301.
- Chandy MJ, Rajshekhar V, Prakash S, *et al*. Cysticercosis causing single, small CT lesions in Indian patients with seizures. *Lancet* 1989;ii:390-1.

Auditing community screening for undescended testes

SIR,—With interest we read the article of Tamhne *et al* about the screening for undescended testes. We were surprised by reported high cumulative rates of orchidopexy especially as the authors state that some of the children in the younger cohort would not have their undescended testes detected and operated on.¹

In The Netherlands during the period from 1976 to 1986 about 3% of boys between the ages of 0 and 14 underwent orchidopexy.^{2,3} Because this percentage is higher than the generally accepted estimate of the prevalence of truly non-descended testes (about 1%⁴) we are making efforts to reduce unnecessary orchidopexy.

As we have reason to believe that in the past the high orchidopexy frequency was related to inaccurate registration of the testes localisation, in some parts of The Netherlands a card for the testes registration was introduced. On this card the localisation of the testes is registered by the person who assists the delivery. If the testes are not scrotal at birth the boys are followed up. After introduction of this card in one region the number of operations for orchidopexy was reduced to an acceptable level.³

We chose registration soon after birth because the cremaster reflex is still absent at