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. We considered this to be a reflection of the amino acid composition of Vamin 9, and we have previously demonstrated that the amino acid profile of Vamin 9 is markedly different from that of plasma phenylalanine. 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 ear dose fed in both infants receiving Vamin 9 and those fed with MB233 represent approximately 60% of the total nitrogen intake. It is therefore difficult to see why 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 MB233 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. 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 in a single child with gastrochisis receiving total parenteral nutrition with a constant nitrogen intake: phenylalanine 149, 500, 200, 493, 500, and 260 μmol/l and tyrosine 95, 60, 85, 58, 86, and 145 μmol/l.

The theoretical basis for the formulation of MB233 is of interest, but it remains to be seen or whether this new solution offers any advantages over alternative preparations.

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Professor McIntosh comments:

Dr Punts 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 them that 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 solution. We would therefore be able to study this difference in much more detail.

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 amino in the range of cord blood aminograms makes it an attractive alternative to Vamin 9 glucose where the amino in the aminograms at five days approximates the values of infants with untreated phenylketouria and hereditary tyrosinaemia.

Virological investigations of acute encephalitis in India

Sir,—Rabies should be considered in the differential diagnosis of acute encephalitis 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?

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.

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Cysticercosis in India is not uncommon2 and parenchymal involvement, the most common form in children,3 may present in adults. The disease is caused by the larval form of the tapeworm, Taenia solium, which produces a cycle of larval development within the human host. The life cycle is completed when the cysticercus, the larval stage, is ingested by an appropriate intermediate host which then becomes infected. Further development occurs in the muscle, subcutaneous tissue and brain, the latter resulting in the characteristic brain abscesses. Cysticercus cellulosae is a widespread and common human parasite. The disease is common in the tropics, particularly in Latin America and the southern United States, and is believed to be common in the Indian subcontinent. 2 Puntis JWL, Edwards MA, Green A, Morgan I, Booth JW, McIntosh N. Hypophenylalaninaemia in parenterally fed newborn babies. Lancet 1986;ii:1105-6.


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