Neurological deterioration in young adults with phenylketonuria

Sir,—Since diet treatment began for phenylketonuria (PKU) 30 years ago several caveats have emerged: (a) neonatal diagnosis is essential and is the basis of successful approach in PKU. However, late diagnosis is no case-control cohort data, such as meta-analyses, that examine essential and is the basis of screening programmes, though this time point for diagnosis is controversial; (b) excessive dietary restriction of essential amino acids is harmful to infant growth and development; and (c) "IQ deterioration" has followed diet termination before 10 years of age in some studies. Dr Thompson and colleagues (Spts 8, p 602) extend the concern over diet termination leading to neurological deterioration in young adults with PKU. While scientific support for these concerns is limited in clinical neuroepidemiological information examined by cost-effective analyses does influence national child health policies. Nonetheless, this model of a treatable, rare metabolic disorder that is limited in clinical neuroepidemiological information examined by cost-effective analyses does influence national child health policies. While neurological symptoms observed by Thompson et al suggest a basal ganglia dysfunction, the appearance of another concurrent or contiguous disease should be considered. In the absence of prevalence data and case-control investigations conflicting viewpoints are not uncommon in PKU management. Specific CNS molecular mechanisms have not been established for this metabolic encephalopathy. There are several caveats, however, that lack scientific support. These caveats, however, lack scientific support. There are, however, several monoaminergic abnormalities. Dystonia is not an explanation of the neurological symptoms. However, the infant... and metabolic disease.7

Phyllanthus amarus and hepatitis B

Sir,—Dr Leelarasamee and colleagues (June 30, p 1600) report the failure of Phyllanthus amarus to eradicate hepatitis B surface antigen from symptomless carriers. Our Lancet paper1 on the effects of P. amarus in chronic carriers of hepatitis B virus (HBV) drew comments in the journal2 and via personal communication. The main criticisms of our preliminary study3 were that HBV-DNA estimations were not done in the pretreatment and post treatment samples of the cleared and refractory carriers; that HBsAg to anti-HBe seroconversion was not fully evaluated; and that long-term carriers (ie, those who carry HBsAg for a year or more) were not studied.

With these criticisms in mind we did an open trial in 1989–90 on 28 symptomless chronic HBV carriers known to have carried HBsAg for at least a year and up to 5 years. They were treated with 250 mg capsules of Phyllanthus amarus thrice daily for 3 months. Serum was obtained before treatment, once a month during treatment, and up to 5 years. They were treated with... and undetectable. Long-term Phyllanthus amarus treatment of HBeAg positive cases showed no beneficial effect. Of the 4 HBsAg cleared cases, 3 seroconverted to anti-HBs. None of the HBsAg cleared cases showed a sequential pattern of HBe seroconversion followed by a reduction in HBV-DNA levels during treatment before HBV-DNA becomes undetectable. Long-term P. amarus treatment of HBsAg positive carriers for at least 1 month at a dose of 500 mg thrice daily might yield better results. P. amarus is being studied in a multi-centre trial in HBV carriers in New Zealand, Vanuatu, Australia, Egypt, Singapore, China, the...