

Phenobarbitone in neonatal jaundice

Sir,

In their paper 'Relative roles of phototherapy and phenobarbitone in treatment of nonhaemolytic neonatal jaundice' (*Archives*, 1973, **48**, 704) Drs. Wong and Wood rightly call attention to the role of ethanol in studies on the effect of phenobarbitone on enzyme induction. They point out that linctus simplex, which we (Levin, McMullin, and Mobarak, *Archives*, 1970, **45**, 93) used as excipient and placebo, also contains ethanol. I think it should be made clear that linctus simplex contains ethanol in a concentration of 0.63% v/v, so that the maximum dose of ethanol given to control and treated babies alike was 0.1045 ml in 24 hours, a very small dose indeed. Such a dose, in so far as it had any effect, tends to obscure any effect of phenobarbitone.

Wong and Wood also stated that we found no significant drop in bilirubin levels when compared to controls. This is not quite correct. We did find a small difference at 24 hours which was significant at the 5% level. If the one infant who required exchange transfusion 24 hours after entry into the trial is included in the analysis at 24 hours, the difference between control and treated bilirubin levels becomes significant at the 1% level using Student's 't' test. Nevertheless, we did conclude that there was no place for phenobarbitone in the routine management of established neonatal jaundice. I would also endorse the suggestion that phenobarbitone may mask the signs of serious underlying conditions giving rise to jaundice.

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Muscular performance and vitamin E in cystic fibrosis

Sir,

The report from Vancouver (Darby, Davidson, and Desai, 1973) on 'Muscular performance in cystic fibrosis patients and its relation to vitamin E' confirms studies performed by Professor Harry Gordon and his co-workers some 15 years ago when they reported on 'Evaluation of muscle strength and effect of tocopherol administration in children with cystic fibrosis' (Levin *et al.*, 1961).

Patients with cystic fibrosis have tocopherol deficiency presumably as a result of steatorrhoea. Evidence for

possible muscular involvement in cystic fibrosis patients is clinical (muscular weakness and wasting in many patients), biochemical (creatinuria on a creatine-poor diet, with reversal of this finding after ingestion of tocopherol esters, accompanied by a decrease in creatine in plasma and an increase of creatine in muscle), and pathological (focal lesions in skeletal muscle resembling nutritional muscular dystrophy, and ceroid pigment in smooth muscle).

Muscle strength was measured with a bulb ergograph in 45 patients with cystic fibrosis attending the clinics at the Babies' Hospital, Columbia Presbyterian Medical Center, New York. In a double-blind study, in which half the cases received tocopherol orally, and the remainder placebo, no significant increase in muscle strength was found in those patients receiving tocopherol over a period of 6 months as compared to the placebo group. Blood tocopherol levels in the treated group returned to normal. This report may have been missed by your contributors.

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Blood urea in infants

Sir,

It may interest your readers to know that 25 years ago while describing the rise of blood urea in dehydrated infants (Doxiadis, 1948), I had also recorded, as in a recent better documented report (Davies and Saunders, 1973), that a high protein intake could cause high blood urea values not only in sick but also in healthy infants. That work of mine had been greatly helped then by the advice of today's senior Editor of the *Archives*.

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