fed. None of the babies had received vitamin K at any time before this study. A significant difference in the Thrombotest values of breast fed compared with bottle and mixed fed babies was obtained (mean (SD) Thrombotest values for breast fed babies=64.1 (20.0)%, for bottle fed=80.0 (22.7)%, and for mixed fed=73.9 (21.2)%; $p<0.01$). Four of the 263 breast fed babies had Thrombotest values lower than 30% and these babies may be at risk from intracranial haemorrhage.

We prescribed 2 mg of vitamin K₂ intramuscularly or orally to 125 babies (90 breast fed, five bottle fed, and 30 mixed fed) who had low Thrombotest values of less than 60% at 2 weeks. The mean (SD) Thrombotest values for babies with each feed before and two weeks after the vitamin K₂ administration were 48.3 (10.0)% and 88.2 (17.4)% for breast fed babies, 52.4 (8.4)% and 86.5 (16.7)% for bottle fed babies, and 50.0 (6.4)% and 93.0 (11.2)% for mixed fed babies, respectively. Thus the administration of vitamin K increased Thrombotest values and may be effective for the treatment of vitamin K deficiency in babies who are breast, bottle, or mixed fed. The possibility of the spontaneous resolution of vitamin K deficiency cannot be absolutely excluded. Recently, O'Conner *et al* showed that oral administration of vitamin K is also effective for the baby with vitamin K deficiency.³ In our studies, 110 of the 125 babies who received vitamin K orally showed a similar increase in the Thrombotest values to those of intramuscular administration.

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Neonatal malaria and obstructive jaundice

Sir,

We enjoyed reading Davenport's paper entitled "Neonatal

malaria and obstructive jaundice" in this journal.¹ We question the use of the term "obstructive jaundice". In Davenport's case there was no extrahepatic obstruction with bile plug. We believe, therefore, that the term "cholestatic neonatal jaundice" should be used in such a condition as we have suggested previously.²

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Dr Davenport comments:

Although I can see the objection to the term 'obstructive jaundice'¹ on the basis that a normal cholangiographic biliary tree is perfectly acceptable, I would like to justify the term on two counts.

Firstly, it is a widely used and understood term and seems more elegant than the biochemically equivalent 'conjugated hyperbilirubinaemia'. It implies a certain pattern of liver function tests—that is, conjugated bilirubin concentration, high alkaline phosphatase activity, and near normal hepatic enzyme activities.

Secondly, microscopically, canalicular cholestasis and intracellular biliary stasis point to a functional obstruction to the excretion of bile after intrahepatocellular conjugation, albeit with a patent extrahepatic biliary tree.

Thus it seems that the term can be retained with some justification on pathological grounds.²

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