Cooke cites disease . . .', though the difference in incidence showed a significant excess of chronic lung disease.  

A programme of systematic and uniform developmental checks for all boys under 5 years is being introduced throughout the district, in keeping with the national directive on child health surveillance.1 If the new programme achieves the desired effect of early detection, testicular screening at school entry may become unnecessary in the future.  

The purpose of our audit was to assess the effectiveness of local screening for undescended testes. General practitioners were unaware that health visitors were not required to check testes, as indeed were two out of the three authors of this study. This fact only became generally recognised as a result of this audit.  

The ineffectiveness of a programme is so often caused by problems which seem completely obvious—in retrospect. The purpose of audit is to reveal these problems, however self-evident they may appear, as frequently this is the only way to motivate change in an organisation.

Factors associated with chronic lung disease in preterm infants  

Str.—Professor Cooke's assertion that . . . 'parenteral lipid emulsions should be restricted to sick premature infants, or to those without respiratory symptoms' is not warranted by the original data he presents or the published work he cites.

In our department, his analysis demonstrates association between chronic lung disease and parenteral lipid therapy but correlation is a notoriously weak proof of cause. In this case why cannot one contend that the longer a baby is ventilated, the more likely he is to receive parenteral lipid? The conclusions of such an analysis also depend on which variables one includes or, more importantly, ignores. The vitamin and lipid imbalance (the causative factor of early gestational maturity) could exemplify the latter category.

In support of his hypothesis, Professor Cooke presents a small proportion of cases of undescended testes were not detected at the neonatal stage but at the school entry examination at 5 years of age. We are therefore compelled to recommend that a 'final' screen at around 5 years is continued at present. Detection at 5 years may not be ideal but it is better than no detection at all.

The conclusions of this paper are invalid. Professor Cooke recommends that parenteral lipid should be restricted to older preterm infants, or those without respiratory symptoms in the belief that the risks of an increased incidence of BPD outweigh 'theoretical gains made from early lipid infusion'. We believe that the balance of risk and benefit should be reversed, and that the harm caused by lipid deprivation may greatly outweigh any theoretical reduction in the incidence of BPD. Undernutrition is a major problem in preterm infants, and the consequences of using lipid emulsions with late use of lipid emulsions and frequent periods of lipid-free alimentation being among the main reasons. Lipid emulsion is energy rich and thus helps avoid the risk of fluid overload while achieving the goal of energy. Moreover, essential fatty acid deficiency quickly develops in preterm babies who are deprived of lipid. Fat soluble vitamins are usually supplemented as an additive to parenteral lipid emulsion. There is increasing speculation that vitamin A deficiency affects the repair of injured squamous epithelium in preterm lungs, and thus contributes to the development of BPD. 

We welcome a multicentre study to test Professor Cooke's hypothesis that lipid emulsion use causes an increased incidence of BPD.


Str.—We read with interest Professor Cooke's paper on factors associated with chronic lung disease (bronchopulmonary dysplasia, BPD) in preterm infants.1 His retrospective study shows an association between lipid use in the first 21 days of life and the incidence of chronic lung disease. Details of lipid use are scanty—there is no information on total duration of lipid emulsion, time of start of emulsion in other than 10 day bands, whether lipid emulsion was reduced as enteral feeds were increased, how often hyperchloremia or hypertriglyceridaemia occurred and if lipid emulsion was reduced accordingly, how many babies received 4 g/kg/day of lipid emulsion (20 mg/kg/day of 20% Intralipid) and so on. An association was also noted between BPD and both gestational age and septicaemia. However, association does not imply causation, and it can only generate a hypothesis to be tested.

The association noted from this retrospective study should be investigated by a prospective randomised controlled study, comparing the current nutritional package in use in the Mersey unit for babies of 24 to 30 weeks' gestation with an intervention in which lipid is used only in babies of this gestational age range who have no respiratory disease. With the quoted (1989/9) rate of BPD in the unit of 66%, an intervention to reduce the rate to 40% would require a sample size of 126 babies in each group for the study to have a power of 90% and significance level of 0.05. As 40% of babies die or are breathing air by 28 days, the total sample size would need to be 420 babies. In a single centre study, this would require 10 years to complete at current rates of preterm birth in the Mersey region. If the intervention reduced the rate of BPD to 25%, a total sample size of 123 babies would be required. Without such a study, we believe that the last paragraph of this paper is invalid. Professor Cooke recommends that parenteral lipid should be restricted to older preterm infants, or those without respiratory symptoms in the belief that the risks of an increased incidence of BPD outweigh 'theoretical gains made from early lipid infusion'. We believe that the balance of risk and benefit should be reversed, and that the harm caused by lipid deprivation may greatly outweigh any theoretical reduction in the incidence of BPD. Undernutrition is a major problem in preterm infants, and the consequences of using lipid emulsions with late use of lipid emulsions and frequent periods of lipid-free alimentation being among the main reasons. Lipid emulsion is energy rich and thus helps avoid the risk of fluid overload while achieving the goal of energy. Moreover, essential fatty acid deficiency quickly develops in preterm babies who are deprived of lipid. Fat soluble vitamins are usually supplemented as an additive to parenteral lipid emulsion. There is increasing speculation that vitamin A deficiency affects the repair of injured squamous epithelium in preterm lungs, and thus contributes to the development of BPD. 

We welcome a multicentre study to test Professor Cooke's hypothesis that lipid emulsion use causes an increased incidence of BPD.