cryptorchid testes. In: Fonkalsrud ED, Mengel W, eds. *The undescended testis*. Chicago: Year Book Publishers, 1981:57–74.
7 Hadziselimovic F. *Cryptorchidism—management and implications*. Berlin: Springer-Verlag, 1983.

Drs Rao, Wilkinson, and Benton comment:
We are grateful to Dr Goh and Dr Hutson for their comments. We agree that testicular screening should start at the neonatal examination. Testicular screening in our district *does* begin with the neonatal examination—as mentioned in our article. What we discovered as a result of our audit was that a major 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.

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. To achieve this it was necessary and appropriate to examine the involvement of all the professional groups who contributed to the developmental surveillance of those aged 0–5, including health visitors. 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 the 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.

1 Department of Health and Social Security. *Child health surveillance: implementation of the new GP contract*. (EL (90)P/36.) London: Department of Health, 1990.

Factors associated with chronic lung disease in preterm infants

SIR,—Professor Cooke's assertion that . . . 'parenteral lipid emulsions should be restricted to older preterm infants, or to those without respiratory symptoms'[1] is not warranted by the original data he presents or the published work he cites.

I do not dispute that his anlaysis 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 A intake (lowest in the infants receiving most parenteral nutrition) could exemplify the latter category.

In support of his hypothesis, Professor Cooke cites a small randomised study[2] as demonstrating that lipid-treated infants '. . . showed a significant excess of chronic lung disease . . .', though the difference in incidence of bronchopulmonary dysplasia (BPD) between the lipid-treated and control groups was not significant by conventional criteria (lipid group 14/20, control group 10/22, $p<0{\cdot}1$). Admittedly the duration of respiratory support and supplemental oxygen treatment was greater in the lipid-treated group ($p<0{\cdot}05$), but the alveolar–arterial oxygen tension difference was somewhat greater in these infants *before* randomisation. It is true that a randomised study of *vitamin A supplementation* demonstrated a reduced incidence of both BPD and retinopathy,[3] but avoidance of lipid emulsion could only exacerbate fat soluble vitamin deficiency.

Fat is a major nutrient which provides about half the non-protein energy intake in most parenteral nutrition regimens and it could not be omitted without constraining amino acid intake too. There is a temptation to increase glucose intake to overcome this problem but this increases both oxygen consumption and carbon dioxide production, further stressing a ventilated immature baby. Lipid emulsions also supply essential fatty acids, important for growth of the nervous system and the synthesis of prostaglandins and leukotrienes. An infant of 26 weeks' gestation (1% fat by weight) would rapidly develop deficiency.

I do not dismiss the possibility that some relationship between chronic lung disease and lipid infusion exists, but would argue that randomised studies of dosage and solution composition are the way forward, not draconian proscription. Neonatal intensive care certainly is a 'continuing experiment'[1] but let us make sure it is adequately controlled.

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1 Cooke RWI. Factors associated with chronic lung disease in preterm infants. *Arch Dis Child* 1991;**66**:776–9.
2 Hammerman C, Aramburo MJ. Decreased lipid intake reduces morbidity in sick premature neonates. *J Pediatr* 1988;**113**:1083–8.
3 Shenai JP, Kennedy KA, Chytil F, Stahlman MT. Clinical trial of vitamin A supplementation in infants susceptible to bronchopulmonary dysplasia. *J Pediatr* 1987;**111**:269–77.

SIR,—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 hypercholesterolaemia or hypertriglyceridaemia occurred and if lipid emulsion was reduced accordingly, how many babies received 4 g/kg/day of lipid emulsion (20 ml/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 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 (1988/9) rate of BPD in the unit of 60%, 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 babies who develop BPD, with late use of lipid emulsion and frequent periods of lipid-free alimentation being among the main reasons.[2] Lipid emulsion is energy rich and thus helps avoid the risk of fluid overload while achieving the desired energy goal. Moreover, essential fatty acid deficiency quickly develops in preterm babies who are deprived of lipid.[3] Fat soluble vitamins are usually supplied 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.[4] We would welcome a multicentre study to test Professor Cooke's hypothesis that lipid emulsion use causes an increased incidence of BPD.

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1 Cooke RWI. Factors associated with chronic lung disease in preterm infants. *Arch Dis Child* 1991;**66**:776–9.
2 Wilson DC, McClure G, Halliday HL, Reid MMcC, Dodge JA. Nutrition and bronchopulmonary dysplasia. *Arch Dis Child* 1991;**66**:37–8.
3 Cooke RJ, Zee P, Yeh Y-Y. Essential fatty acid status of the premature infant during short-term fat-free parenteral nutrition. *J Pediatr Gastroenterol Nutr* 1984;**3**:446–9.
4 Shenai JP, Chytil F, Stahlman MT. Vitamin A status of neonates with bronchopulmonary dysplasia. *Pediatr Res* 1985;**19**:185–9.

Professor Cooke comments:
Chronic lung disease (CLD) in preterm infants results from lung damage and abnormal healing caused by a wide variety of factors. My study is aimed to discover the cause for a step change in the incidence of CLD in our unit in 1987/8. The only variable examined that satisfactorily explained such a change was the early use of parenteral lipid, introduced at this time because of concern about poor postnatal weight gain in patients with CLD. It would of course be possible to explore this variable further in more detail and it is quite possible that only some infants, probably the sickest, are adversely affected by very early lipid infusion.