Dr. Cooke's work, which showed a significant excess of chronic lung disease in preterm infants, has been the subject of much debate. Cooke's hypothesis that lipid treatment could reduce the risk of chronic lung disease is based on several key findings. In a retrospective study, Cooke and colleagues compared the incidence of chronic lung disease (bronchopulmonary dysplasia, BPD) between lipid-treated and control groups. Admittedly the difference in respiratory support and supplemental oxygen treatment was greater in the lipid-treated group (p < 0.05), 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 BPD and respiratory failure in these infants. Lipid emulsion could only exacerbate fat soluble vitamin deficiency.

Fat is a major nutrient which provides about half of 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 prevention of osteoporosis 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 prescription. Neonatal intensive care certainly is a "continuous experiment" but let us make sure it is adequately controlled.

Factors associated with chronic lung disease in preterm infants

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

In our institution, this analysis demonstrates an 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 status of the premature infant is the most important variable which needs to be considered in any analysis of parenteral lipid consumption or correlation with chronic lung disease.

In support of his hypothesis, Professor Cooke cites a number of clinical trials 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 > 0.1). Admittedly the difference in respiratory support and supplemental oxygen treatment was greater in the lipid-treated group (p < 0.05), 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 BPD and respiratory failure in these infants. Lipid emulsion could only exacerbate fat soluble vitamin deficiency.

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