LETTERS TO THE EDITOR

Neonatal hyperoxaemia

Sir,—Although hyperoxaemia is no longer considered the sole risk factor for retinopathy of prematurity,1 litigation still frequently cite it as the cause, arguing that too much oxygen was given and that hyperoxaemia could have been prevented. In order to understand the timing and frequency of hyperoxaemia, we reviewed all arterial blood gas samples on babies <33 weeks' gestation who received supplemental oxygen for respiratory distress by headbox, continuous positive airway pressure or ventilation, and had repeated arterial samples. Before establishing an arterial line babies were nursed to maintain predectal transcutaneous oxygen tensions of 7–12 kPa, and thereafter to maintain similar arterial oxygen tensions (Pao2).

A total of 1836 arterial samples were taken from 90 babies. Their median (range) birth weight was 1220 (480–2550) g and median gestation 29 (24–32) weeks. The median number of samples from individuals was 15 (2–170). On day 1, 452 (25%) samples were taken, 707 (39%) on days 2–4, 578 (21%) on days 5–10, and 299 (16%) thereafter. Fourteen babies were excluded from the analysis because they died within 48 hours. They had a total of 94 arterial samples before they died, and only one had a Pao2 >12 kPa. Among the other 76 babies, 64 (84%) had at least one Pao2 >12 kPa, the level considered to be hyperoxic. Hyperoxaemia was most frequent at the first arterial sample, when 19 (25%) of babies had levels above the threshold. Fifty two (16.5%) of the 315 other samples on day 1 had a Pao2 >12 kPa, a higher proportion than any other day. A second peak was observed at days 5 to 10, when 44 (11.6%) samples were hyperoxic. We believe this reflected the tendency for Pao2 to rise on constant respiratory support during recovery from surfactant deficiency. No patient was free of hyperoxaemia, but the lowest prevalence was 7% (21 of 299 samples) after day 10 (figure). These trends were similar when thresholds of 14 and 16 kPa were used.

There is no universally agreed definition of hyperoxaemia in preterm infants. The upper limit of our target range was 12 kPa and was therefore the empirical definition.1 Because the lower limit of 12 kPa is 4 SD above the mean (SD) Pao2 of 7–9 (1–0 kPa) in normal premature infants at 3 to 5 hours of age,2 Thibeault et al did not record a Pao2 >12 kPa in air breathing non-distressed babies of 1201–1600 g until day 19.3 If hyperoxaemia is causally related to retinopathy in some babies, it is crucial to know (a) when hyperoxaemia is most likely to occur and (b) if the developing retina is susceptible to hyperoxic damage at that time. Our observations confirm that even when supplemental oxygen treatment is carefully controlled episodes of hyperoxaemia are not uncommon.


Screening for undescended testes

Sir,—While the intentions of Rao et al in auditing and thereby seeking ways to improve the screening for undescended testes are commendable, it would seem they have failed to grasp the significance of the two ‘windows of opportunity’, one for diagnosis and the other for possible effective treatment, that are available in this problem.1

First, the best time to examine a child for undescended testes is in the newborn period because the cremasteric reflex is absent, there is little subcutaneous fat, and the scrotum is proportionately at its largest.2 At this time you also have a ‘capitive market’ as, in a developed country, practically all newborns will be examined at some stage by at least one health professional. The presence of testes and their positions can be recorded and all cases of abnormality or doubt should be referred for surgical opinion as soon as possible. Efforts at improving screening will most profitably be directed at this age group rather than at a later age when physical characteristics and logistics combine to make screening less effective as was shown in their study.

Evidence exists to support the phenomenon of the apparently acquired ‘ascending testis’ in which a previously descended testis subsequently ascends to a truly undescended position.3 Neonatal screening may miss this group and this may be where later screening can have a limited but useful role. Even so, there is evidence to suggest that some of these ascending testes occur in boys with incompletely descended testes at birth which subsequently descend in the first few months of postnatal life only to ascend again at a later age.4 These ‘late descenders’ could be detected at neonatal screening and referred for careful surgical supervision.

Secondly, we would agree that late orchidopaxy beyond 2 years of age appears to have little or no influence on fertility or the risk of malignancy.5 In more recent years, paediatric surgeons have been performing orchidopaxy at earlier operations before the age of 2 years but the results of this approach still remain to be seen because of the obviously long follow up required. Histological studies, however, have shown that the undescended testis appears to be normal in the first year, but by the end of the second year pathological changes are present, and progress rapidly.6 Logic therefore dictates that if surgery is to have any influence on the development of the undescended testis it should be performed before the age of 2 years. This renders invalid the authors’ proposal that screening be performed at the preschool age of 5 years.

It seems invidious, and certainly irrelevant, to even attempt to critically perform the health visitors in a screening procedure for which they receive absolutely no training and are not even attempting to perform in their routine assessments of children (LE Morgan, personal communication), as was indeed mentioned in the paper. If you are not even looking for something it should surprise no one that you do not find it. It would also not be surprising if most of the referrals from health visitors resulted from prompting by parents who had noticed a problem as over 40% of the cases in the study were self referrals by parents.

A screening programme directed primarily at 5 year old preschool children is too late. It can only serve to detect the ascending testes and the small number of undescended testes that have escaped detection in early infancy. We would advocate examination of all neonates at birth to document testicular position. Those with an empty scrotum should be seen again at 3 and 6 months, at which time the testes of premature babies and later ‘ascending testes’ will be down. Those babies with an empty scrotum at 3 months should be referred for surgery at 1 to 2 years, while those with delayed descent should be followed up actively to see if the testis reascends.

6 Mengel W, Wronecki K, Zimmerman FA. Comparison of the morphology of normal and
Dr Rao, Wilkinson, and Benton comment: We are grateful to Dr Koh and Dr Hutson for their comments. We agree that tecticular screening should start at the neonatal examination. Tecticular 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 undetected cases 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 on around 5 years is continued at present. Detection at 5 years may not be ideal. 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. If the new programme achieves its desired effect of early detection, tecticular screening at school entry may become unnecessary in the future.

The purpose of our audit was to assess the effectiveness of local screening for undetected testicular atrophy. To achieve this it was necessary and appropriate to examine the involvement of all the professionals 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 an 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 seriously ill preterm infants, or those without respiratory symptoms” is not warranted by the original data he presents or the published work he cites.

It does not follow that 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 virulence of lipid therapy is the size of the actual cohort (including most parental nutrition) could comfortably exceed the latter category.

In support of his hypothesis, Professor Cooke cites another study 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 1/20, control group 10/22, p<0.1). Admittedly the duration of 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 both BPD and retinopathy in the group who avoided vitamin A. Lipid emulsions 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 prescription. Neontal intensive care certainly is a ‘continuing experiment’ but let us make sure it is adequately controlled.

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3 Shenai JP, Kennedy RA, Chyil F, Stahlman M. Clinical significance of lipid emulsions for babies who received lipid treatment with late use of lipid emulsion and frequent periods of lipid-free alimentation being among the main reasons. Lipid emulsions are energy rich and thus helps avoid the risk of fluid overload while achieving the energy goal. Moreover, essential fatty acid deficiency quickly develops in preterm babies who are deprived of lipid. 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. 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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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 of a 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, i.e. the decision 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 detail but it is clear that only some infants, probably the sickest, are adversely affected by very early lipid infusion.