LETTERS TO THE EDITOR

Neonatal hyperoxaemia

SIR,—Although hyperoxaemia is no longer considered the sole risk factor for retinopathy of prematurity,¹ litigants 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 gases 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 preductal 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, 378 (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 hyperoxaemic. 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 hyperoxaemic. We believe this reflected the tendency for PaO₂ to rise on constant respiratory support during recovery from surfactant deficiency. No period was free of hyperoxaemia, but the lowest prevalence was 7.0% (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. The level 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 12.³

If hyperoxaemia is causally related to retinopathy in some babies, it is crucial to know (a) when hyperoxaemia is most likely to occur and if it can be prevented, and (b) if the developing retina is susceptible to hyperoxaemic damage at that time. Our observations confirm that even when supplemental oxygen treatment is carefully controlled episodes of hyperoxaemia are not uncommon.

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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 'captive 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 and become undescended again at a later age.⁴ These 'late descendents' could be detected at neonatal screening and referred for careful surgical supervision.

Secondly, we would agree that late orchidopexy 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 tending towards early 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.^{6,7} 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 assess critically the performance of health visitors in a screening procedure for which they receive absolutely no training and are not even expected 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 to 6 months can 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.

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