Neonatal respiratory distress syndrome

EDITOR,—The report of the working group on the management of neonatal respiratory distress syndrome states that 'normal' limits for blood gas variables cannot be stated, and appropriate levels of arterial oxygen saturation are not yet agreed.6 As a result they recommend that further research includes determining safe levels of SaO₂ in preterm infants with respiratory distress syndrome and chronic lung disease. Despite this area of uncertainty, they state that the recommended range for PaO₂ [arterial oxygen tension] is 6–10 kPa and that acceptable levels for SaO₂ of 85–93% have been proposed.4 These ranges are stated without reference to reported normal values and may in fact be detrimental to an infant.

The fifth centile for levels of SaO₂ in ‘healthy’ preterm infants without lung disease and either ready for discharge from the neonatal intensive care unit,5 or in the first week of life,3 are 95% and 95.5% respectively. These measurements were made with the Nellcor N100/N200 during quiet sleep and excluding the transient drops in SaO₂ after normal respiratory pauses. By definition, hypoxaemia is below these levels and should be corrected when treating respiratory failure.

In order to avoid hypoxaemia (for which definitions vary) in preterm infants receiving mechanical ventilation, the upper limit of SaO₂ must also be specified. Soutar et al, using the Nellcor N100 pulse oximeter, found from 169 measurements in 91 patients, 24 occasions when PaO₂ was >100 mm Hg (≥13.3 kPa); the SaO₂ was >97% on 23 occasions and 95% on one.4 Bucher et al, comparing the Nellcor N100 and the Ohmeda Biox pulse oximeters, reported that the Nellcor N100 identified hypoxaemia (PaO₂ <90 mm Hg or <12.0 kPa) with 100% sensitivity if an alarm level of 95% was chosen.5 The Ohmeda Biox 3700 pulse oximeter, however, showed a sensitivity of only 37% at this alarm level. A more recent study using the Ohmeda Biox 3700 found that the lower limit SaO₂ levels of 95–96% would result in a postductal PaO₂ of ≥99 mm Hg (<3.7 kPa) for 95% of the time.6 Another study, involving 137 hypoxaemic instances (defined as PaO₂ <80 mm Hg or 10.7 kPa) in 50 patients, found that the Nellcor N200, with the alarm limit set at 95%, identified 95% of these instances.7 The highest PaO₂ value not identified by the pulse oximeter was 104.5 mm Hg (13.9 kPa).

Thus, when using the Nellcor pulse oximeter, hypoxaemia may only be avoided with sufficient certainty if the upper alarm limit is kept at 95–97%. This implies that SaO₂ values above 95–97%, although obviously 'normal' for healthy preterm neonates, cannot be recommended for preterm infants receiving respiratory support. Because of this unfortunate overlap between normal SaO₂ levels, and those that may be associated with a 'dangerously high' PaO₂, we would recommend that baseline SaO₂ is kept between 94 and 96% in preterm infants receiving additional inspired oxygen and monitored using the Nellcor pulse oximeter. In addition, and as the working group stresses, the monitoring of arterial line PaO₂ values will remain essential to assess the effect of respiratory support and to be certain of avoiding hypoxaemia.

Lower levels of SaO₂ may be detrimental: firstly, preterm infants with a low baseline SaO₂ desaturate further with apnoea pauses than those who are adequately oxygenated.8 Secondly, hypoxaemia as a result of lung hypoxia increases both pulmonary vascular and bronchial smooth muscle tone,9 10 Such changes may prolong ventilatory and oxygen dependence, increase the risk for severe hypoxaemic episodes, and result in infants being treated with diuretics and bronchodilators. Inspired oxygen is a potent pulmonary vasodilator and may also prevent bronchospasm.

We agree with the recommendation in appendix A that one of the most important steps forward here would be a randomised controlled trial looking at the levels of oxygenation recommended for infants being treated with respiratory distress syndrome. Such a study should include information concerning retinopathy as well as major outcome variables, such as death, chronic lung disease, the duration of inspired oxygen, and levels of required ventilation.

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Varicella zoster virus infection in pregnancy

EDITOR.—The recent annotation describes the possible effects on the fetus from maternal varicella zoster infection, including the embryopathic effects of first trimester infection.1 The authors quote Alkalay et al2 as suggesting, in their review of all published reports of the fetal varicella syndrome (FVS), that the presence of cicatrificial skin lesions corresponding to a dermatome distribution is essential. We have reviewed one well documented case had other features of FVS but no skin lesions,3 and we have recently seen a similar case.

A baby boy was born at 36 weeks’ gestation, with a birth weight of 3.0 kg, below the third centile. His mother had had chicken pox at 16 weeks. At birth, apart from being severely growth retarded, there were no abnormal external features. No antiviral therapy had been taken by either the mother or father during the infection. General condition was adequate, and there were no immediate complications. The baby remained well over the next 3 weeks. His development was marginal and he had mild feeding difficulties. A review of all the imaging and tests at that time failed to reveal any evidence of a neural tube defect. A careful review of all the imaging and tests during the pregnancy failed to identify any fetal abnormalities. Only at the age of 9 months did he begin to show the characteristic features of varicella zoster virus infection, including skin rash, ataxia and failure to thrive. The disease has responded well to valacyclovir. We believe that this case is a further indication of the deleterious nature of acute maternal varicella zoster infection in the first trimester of pregnancy, and is completely consistent with the embryopathic effects proposed by Alkalay et al.2 We also believe that the absence of cutaneous correlate of varicella zoster virus infection found in the fetus in this case does not contraindicate the diagnosis of varicella zoster virus infection in pregnancy.

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