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 should be determined for each neonatal unit. As a result they recommend that further research includes determining safe levels of SaO₂ in infants with respiratory distress syndrome and chronic lung disease. Despite this area of uncertainty, they state that the normal range for PaO₂ [arterial oxygen tension] is 6-10 kPa and that acceptable levels for SaO₂ of 85-93% have been proposed. 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 unit, or in the first week of life, are 97.5% 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, apnoeic 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. Southall 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 Burcher et al, comparing the Nellcor N100 and the Ohmeda Biocare 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 Biocare 3700 pulse oximeter, however, had a sensitivity of only 37% at this alarm level. A more recent study using the Ohmeda Biocare 3700 found that the lower limb SaO₂ levels of 95-96% would result in a postductal PaO₂ of 99 mm Hg (13-2 kPa) for 95% of the time.6 Another study, involving 137 hypoxaemic infants (defined as PaO₂ <80 mm Hg or 10-7 kPa) in 50 patients, found that the Nellcor N200, with the alarm level set at 95%, identified 95% of these infants. 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 hyperoxaemia.

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 bronchiolar 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 oxygen saturation used for ventilated newborn infants 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 cicatrinal skin lesions corresponding to a dermatome distribution is essential. They then state that 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 bursterna cataract, coelomic effusion, profound developmental delay, and a hoarse, weak cry, but he has no skeletal or urinary tract abnormalities. Contrast radiological studies have shown almost complete absence of oesophageal and gastric peristalsis, and he is fed via a jejunostomy tube. He is unable to swallow saliva and has permanent respiratory signs and symptoms due to aspiration.

This is a very rare case and the authors are correct in stating that we are confident that our patient does have FVS, but were misled by the absence of skin lesions and limb abnormalities at birth. It is clear that babies may be born after maternal varicella zoster infection up to 20 weeks' gestation who are severely affected by varicella embryopathy, but in whom the external appearances may be reassuringly normal. It is therefore, very important to examine such infants closely for other features of FVS.

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