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, before and after intervention, have not been defined. 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 recommended 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 intensive care unit, or in the first week of life, 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 or diabetic feeds. By definition, hypoxaemia is below these levels and should be corrected when treating respiratory failure.

In order to avoid hyperoxaemia (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 on 91 patients, 24 occasions when PaO2 was >100 mm Hg (>13.3 kPa): the Sao2 was >97% on 23 occasions and 95% on one.

Bucher et al, comparing the Nellcor N100 and the Ohmeda Biox 3700 pulse oximeters, reported that the Nellcor N100 identified hyperoxaemia (PaO2 >90 mm Hg or >12.0 kPa) with 100% sensitivity if an alarm level of 95% was chosen. The Ohmeda Biox 3700 pulse oximeter, however, had 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 (>13.2 kPa) for 95% of the time.

Another study, involving 137 hyperoxaemic 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. 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, hyperoxaemia 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 dangerous ‘hypoapnoeic’ High Sao2, 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 apnoeic pauses than those who are adequately oxygenated. Secondly, hypoxaemia as a result of lung hypoxia increases both pulmonary vascular and bronchial smooth muscle tone. 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 bronchoconstriction.

We agree with the recommendation in appendix A that one of the most steps forward here would be a randomised controlled trial looking at the levels of oxygen saturation in infants ventilated for 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.

DAVID P SOUTHWALL MARTIN P SAMUELS CHRIStIAN P PFOETS
Academic Department of Paediatrics, University of KwaZulu-Natal, North Staffordshire Hospital Centre, Stoke-on-Trent ST4 6QG


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. The authors quote Alkalay et al1 as suggesting, in their review of all published reports of the fetal varicella syndrome (FVS), that the presence of cicatrization skin lesions corresponding to a dermato- cutaneous distribution is essential. We have found one well documented case had other features of FVS but no skin lesions,1 and we have recently seen a similar case.

A baby boy was born at 36 weeks’ gestation with a birthweight of 2.5 kg, and a neonatal unit stay of 11 weeks. At birth, apart from being severely growth retarded, there were no abnormal external features. No antiviral zoster IgM was detectable in his blood, and this, together with the absence of cicatrization skin lesions or limb abnormalities, reassured us that he had probably escaped the FVS. However, further examination revealed severe chorioretinitis, and at 10 weeks, whilst still on the neonatal unit, he developed a typical shingles rash in the C6 dermatome distribution. There had been no postnatal contact with anyone with chickenpox, and no chickenpox immunity. Skin aspiration of the vesicles identified varicella zoster particles. There was no serological evidence of other congenital infections.

He had chickenpox rash and other features well described in the FVS, including severe gastro-oesophageal reflux and bulbar palsy resulting in several near fatal episodes of aspiration pneumonitis, cortical seizures, 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 jejunojunostomy tube. He is unable to swallow saliva and has permanent respiratory signs and symptoms due to aspiration.

We are confident that our patient does not 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’ gestation2 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.

HEATHER SMITH
Department of Paediatrics, Bishop Auckland General Hospital, Bishop Auckland, County Durham DL14 6AD

SUNIL SINHA
Neonatal Unit, South Cleveland Hospital, Middlesbrough TS4 3BW


Downloaded from http://adc.bmj.com/ by guest on March 3, 2022