Neonatal respiratory distress syndrome

EDITOR,—Professor Southall and colleagues1 question the comments of the working group on the management of neonatal respiratory distress syndrome regarding parameters of oxygenation.2 We write in support of the provisional and practical suggestion of an arterial oxygen saturation (Sao2) range of 85–93% and suggest that much of the data quoted by Southall et al is not applicable to this clinical setting.

Professor Southall and colleagues suggest an upper limit for Sao2 of 96% and quote four studies in support of this choice. In two of these papers,1,2 the majority of the patients were neonatal unit infants who would have been treated using Ohmeda Biox oximeters on a population under 33 weeks' gestation found that if the saturation value was 93% or below the arterial oxygen tension (PaO2) was never greater than 12 kPa.3 To maintain the PaO2 below 10 kPa, which is the upper limit suggested by the working party, requires a saturation of 92% or less. Certainly, when using an Ohmeda oximeter, an upper limit of 93% seems very reasonable.

When discussing a lower limit for Sao2, Professor Southall recommends 95%, based on a study of well preterm infants.4 These infants bear little relation to the sick, low birthweight infants who, until very recently had experienced a physiological, umbilical venous oxygen tension (Po2) of approximately 4–5–6 kPa.

With regard to the adverse effects of hypoxia, there is no firm evidence which defines the optimal range for any of the indices of oxygenation. There are almost no modern data relating levels of oxygenation to outcome; the data to be quoted by Professor Southall regarding hypoxia describe physiological responses to lower levels of oxygenation and not outcome.

Limited by the lack of scientific data, the working party have had to make reasonable and, equally important, practical recommendations. We feel that there is little evidence to support a range of 94 to 96% and in addition would be surprised to find any significant difference to be quoted by Professor Southall regarding hypoxia within such narrow limits. With the marked fluctuations in oxygenation seen in preterm infants receiving intensive care, it would be difficult to make sufficiently frequent adjustments to the parameters quoted in these limits. Based on our own data we currently employ the range 86 to 94%, similar to that mentioned in the working party report and ask for further studies to give us more guidance on this difficult question.


Professor Southall and colleagues comment:
The upper limit for Sao2 that we recommend is based on several studies correlating PaO2 and saturation levels.1,3 Although the two papers mentioned by Cochran and Shaw did include older infants,2 the potential bias of the correlation method of calculating PaO2 from pulse oximeter's sensitivity to hyperoxia — the higher proportion of fetal haemoglobin in preterm infants would result in a higher PaO2 at any given PaO2. With regard to avoidance of hyperoxia (PaO2>13 kPa), the Nellcor N100 has been found to identify hyperoxia with a 95% sensitivity at saturations of 96% and above; the Ohmeda Biox 3700 had a similar sensitivity, but at saturations of 89%.5

The different averaging times, and algorithms of available pulse oximeters must be taken into account when discussing acceptable ranges of Sao2 in the neonatal population. Until manufacturers standardise their technology, it would be safer to (a) use oximeters that have been validated for use in the neonatal population, with demonstrated sensitivity for the detection of hyperoxia but avoiding a range that avoids the risk of hypoxaemia, (b) ensure that when defining acceptable ranges for Sao2, the range is model specific to avoid inadvertent hyperoxia or hyperoxia by using inappropriate limits.

Our recommendation for saturation was originally based on a study of ‘well’ preterm infants at discharge from hospital. It has now been confirmed in a study of non-distressed preterm neonates investigated during their first week of life.6 It does not seem unreasonable to aim to achieve the same range of Sao2 in ‘sick’ preterm infants. The argument that the latter group had recently experienced a physiological intratracheal PaO2 of 6–5–6–0 kPa, the light of 4–5–6–0 kPa the range of arterial oxygen saturation that avoids the risk of hyperoxaemia, but not of Oxygen consumption. Finally, large changes in Sao2 will be seen in an infant if lower Sao2 values are maintained. This is because of the shape of the dissociation curve, and the resultant effects of hypoxaemia and hyperoxia on pulmonary vascular and bronchomotor reactivity.


Blood pressure and blood volume in preterm infants

EDITOR,—We read with interest the article by Bauer et al on blood volume and blood pressure in preterm infants.1 We have looked at a very similar group of infants and assessed the blood volume but with different results.

In 31 infants the mean arterial blood pressure (MAPB) was measured by invasive arterial methods. Blood volume was assessed using an indocyanin green dye dilution method described previously in this age group.2 The infants had a median gestational age of 26 weeks (range 25–31 weeks) and a median birth weight of 900 g (range 580–1380 g). Results were all obtained in the first three days of life.

The median blood volume observed in our infants was 85 ml/kg (range 46–131 ml/kg), very similar to both the 83 ml/kg of Bauer et al and to previously published results from a median MAPB of 36 mm Hg (range 18–47). We found no significant relationship, on regression analysis, between observed blood volume and MAPB, p=0.42 (figure).

Thus, we have not confirmed the third order polynomial relationship described by Bauer et al. This may be for a number of reasons, but it may be of note that the majority of the samples in Bauer et al’s mean arterial pressure control for MAPB from their group by a non-invasive method, that has previously criticised in the sick very low birthweight infant.3 We would agree with the conclusion of Bauer et al that blood pressure assessment alone is not a poor measure of absolute hypervolaemia in very low birthweight infants. It is important that more than one cardiovascular parameter is considered in the assessment of the hypervolaemic infant so that different expansion and inotropes are used appropriately in this age group.