Oxygen saturation and retinopathy of prematurity

EDITOR,—The observations of Tin et al1 have led them to suggest that babies may have better overall outcomes when unit policies aim at oxygen levels of 70–90%, much lower than current practice in most NICUs. While I would support their call for further well designed research into this question, I have major concerns that this concept of beneficial hypoxia might creep into clinical practice, and even be extended to the older survivors. The authors are clearly aware of the limitations of their study. There are obviously many possible alternative reasons for the differences in outcome between the nurseries; table 2 of the study suggests widely divergent policies on a number of issues apart from oximetry levels. There are no data supplied regarding the actual oximetry levels maintained in the nurseries, which makes conclusions about the safety of a saturation of 70% rather speculative.

My main concern is the potential risk to older babies with chronic lung disease who might once again be subjected to chronic hypoxia. Since the more widespread acceptance that babies with chronic lung disease require similar oxygen levels to their more fortunate brethren we have largely abolished the high first year mortality in these babies, and the high incidence of hypoxaemia and the pulmonary hypertension which was previously seen. One observational study of differing oximetry levels within a single unit confirmed the high risk of even mild chronic hypoxia in this group of infants,2 showing a high rate of apparently life threatening events in the hypoxic infants.

While there is continued uncertainty about the optimum oximetry levels in the early life of a preterm baby, there is no justification for maintaining subnormal levels of oxygen in babies beyond 34–36 weeks of age with chronic lung disease, and I trust that this paper will not encourage such practice.

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Two sacred cows of neonatal intensive care

EDITOR,—I read the descriptive study of Tin et al1 with considerable interest. In essence it challenges two sacred cows of neonatal intensive care, whether intra-arterial monitoring is necessary, and what is the appropriate PaO2 at which to nurse critically ill babies.

ARTERIAL MONITORING

They do not give us accurate details of arterial catheter use. There is a hint that they are used for fewer days in the more affluent babies at SpO2 and capillary measurements. Nor do they tell us what analgesia is used for multiple capillary samples.

A fundamental principle of neonatal intensive care is minimal handling, and indwelling arterial catheters allow all samples to be taken with no or minimal disturbance, and if the catheters are umbilical, they can also be used safely for virtually all infusions including TPN. Furthermore, if the sampling is done using oscillometric techniques, they allow accurate blood pressure recordings.

Surprisingly, the literature, and my own clinical experience, show that various complications of UAC are much more common in term and near-term babies than in more preterm infants. Until such data show that babies kept at 3.3–6.0 kPa for days and weeks during a vulnerable period for brain development do as well as those kept adequately oxygenated, I would regard the practice as described by Tin et al2 as of unproven benefit and possibly dangerous.

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Two sacred cows of neonatal intensive care—Authors’ response

EDITOR—I am glad to have a chance to respond to Dr Roberton’s assertion that the care of the babies nursed using oximeter settings of 70–90% was “negligent”, since I was responsible for those children, but time and space does not allow a full response. Neither does space allow me to respond to the criticism implicit in your own introductory statement that such care “breaches BAPM guidelines.”

Dr Roberton says the cerebral palsy rate is “irrelevant”, but parents might not agree. Parents might also be glad that, while 4 children monitored using an oximeter alarm set at 88–98% went blind, no child in the other group went blind. They might also be glad that half were born with ankyloglossia and that half were born with Pierre Robin syndrome. This, however, is far superior as it enables the child to lie prone and is less liable to get knocked or blocked through kinking. We suggest that this approach overcomes these issues.

Dr Roberton mentions the outcome of a study of 38 children offered corrective surgery for transposition 6 months to 5 years after birth.1 Those operated on early had a better outcome, but Dr Roberton does not mention the fact that these children had a mean saturation of 68% before operation, and that 8 had a history of acquired central nervous system damage. Hypercapnia, the main thrust of Dr Roberton’s letter is that lack of an arterial line subjected these babies to unnecessary pain. This overprotects the fact that morphine was given to discourage ventilation. The main issue of concern here is the cost of preventing retrolental fibroplasia.

In the original description the protruding part of the airway is cut into four strands and the upper cut off. We found the remaining strands rather thick, so cut them to half the width. We found that leucoplast tape was the only tape that held the strands together with no additional benefit resulting from the use of Tinc Benz. The strands rubbed badly where they curved over the edge of the nostril. To overcome this a piece of suction catheter (9F) of just sufficient length to extend over the lateral and medial walls of the nostril is tied transversely across the tube with a 3/O silk tie (fig 1). This lifts the three strands off the edge of the nostril and prevents any rubbing.

In our patient blockage of the airway occurred after formula but not breast milk feeds. It is therefore advisable to suction the airway after each feed. We used a suction catheter with graduation marks enabling insertion no further than the tip of the airway. If inserted further the pharyngeal stimulation usually caused vomiting.

If a nasogastric tube is also required this can be taped to one of the strands rather than the face. The airway was changed every four to six days, immediately before a feed and alternating between nostrils. Using 1% lignocaine drops and smearing the tube with lignocaine jelly reduced crying time after insertion.

Compared to the use of an unmodified tube with a large connector attached to the end, we feel that the technique described by Masters et al is far superior as it enables the child to lie prone and is less liable to get knocked or blocked through kinking. We suggest that the modifications we have described will further improve the acceptability of the technique.

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Using a modified nasopharyngeal airway in Pierre Robin syndrome

EDITOR—Masters et al describe how a modified endotracheal tube can be used as a nasopharyngeal airway in infants with Pierre Robin syndrome.1 We describe further modifications made to overcome some problems which we encountered in using the technique in one of our patients.

In the original description the protruding part of the airway is cut into four strands and the upper cut off. We found the remaining strands rather thick, so cut them to half the width. We found that leucoplast tape was the only tape that held the strands together with no additional benefit resulting from the use of Tinc Benz. The strands rubbed badly where they curved over the edge of the nostril. To overcome this a piece of suction catheter (9F) of just sufficient length to extend over the lateral and medial walls of the nostril is tied transversely across the tube with a 3/O silk tie (fig 1). This lifts the three strands off the edge of the nostril and prevents any rubbing.

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Figure 1 The modified nasopharyngeal airway in place.


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Use of the black area on the tube tip for rapid estimation of insertion depth of endotracheal tubes in neonates: a potential hazard

EDITOR—I would like to report on a premature neonate who was intubated unilaterally as a result of improper use of the black area at the endotracheal tube tip. At 29 weeks gestational age, a 1020 g boy was born by emergency caesarean section to a mother who presented with preclampsia. He was intubated immediately for signs of severe respiratory distress with a 3.0 mm ID tube via the naresotracheal route by the resident on call. The black area of the tube was inserted full length through the vocal cords with the upper rim positioned at the level of the vocal cords. Breath sounds were auscultated through the tube. The tube was fixated with adhesive tape at the 8.5 cm mark at the nose. A thoracic x ray revealed that the tube was located in the entrance of the right main stem bronchus. The tube was removed and replaced with a 3.5 cm and fixated at 8.5 cm at the nose, after which exogenous surfactant was instilled for treatment of grade 3 idiopathic respiratory distress syndrome. The ensuing clinical course was uneventful and the infant was discharged with signs of mild bronchopulmonary dysplasia several weeks later.

During the evaluation of this incident it was found that the resident who intubated had been taught at another institution that the length of the black area would ensure a full insertion of the endotracheal tube. The resident had not been taught at this other institution that the position of the upper rim of the black area at the level of the vocal cords would ensure a proper insertion length. However, the tube at this other institution was produced by a different manufacturer. This prompted us to measure the actual length of the black area on the neonatal size endotracheal tubes of four major manufacturers. As shown in table 1, the length of the black area varies among the tubes from different manufacturers. One manufacturer has adjusted the length of the black area to the size of the patients for which a particular tube size is indicated. The others added a black area of a fixed length, regardless of tube size and, thus, patient size. The distance from the vocal cords to the carina of a neonate of 1000 g is approximately 30 mm.1 This explains the endobronchial position of the full length insertion of the black area of the endotracheal tube through the vocal cords in our patient. The equal distribution of breath sounds that was used in this case to determine the correct tube position has been shown to be an unreliable parameter for this purpose in neonates.2

In conclusion, this report illustrates that caution is required in the use of the black area at the endotracheal tube tip for rapid estimation of insertion depth of endotracheal tubes in neonates.

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Respiratory distress syndrome and antenatal corticosteroid treatment in premature twins

EDITOR—Randomised, placebo controlled trials of antenatal corticosteroid administration have not shown a significant reduction in the incidence of respiratory distress syndrome (RDS) in premature twins.1 Subsequent retrospective studies examining the effect of steroids on twin pregnancies have shown conflicting results.2

Further to our recent article,3 we have investigated the relationship between respiratory distress and antenatal corticosteroid treatment in premature twins from the same historical cohort selected from the Australia and New Zealand Neonatal Network (ANZNN) 1995 database. To reflect best possible clinical practice, the analysis was restricted to the effects of an optimal steroid course (two doses of corticosteroids given, the first dose of which was received more than 24 hours and less than eight days before the infant’s birth) compared with no steroid treatment.

As shown in table 1, treatment with antenatal steroids resulted in a significantly lower incidence of RDS and surfactant use. The reduction from 18% to 11% in the risk of mortality was not significant (p = 0.08). Recent advances in obstetrics and neonatology could explain the absence of an antenatal steroid associated reduction in mortality.4 There was no statistically significant association between optimal steroid use and the outcome measures of days of intermittent positive pressure ventilation, days of oxygen, oxygen at 36 weeks corrected gestational age, severe intraventricular haemorrhage, or the number of proven infection episodes. Gestation, sex, and birth order did not modify the association between antenatal steroid treatment and RDS incidence.

The sample examined in this study is over twice the size of the most recent retrospective analysis, which reported no antenatal steroid associated reduction of RDS in twins.5 However, the reduction in RDS incidence observed in our study is less than that seen in singleton infants (odds ratio 0.35, 95% confidence interval 0.26 to 0.46).6 Optimal steroid treatment may be less effective in multiple gestation pregnancies because the increased volume of distribution in these mothers may reduce the plasma level of steroids to which the fetuses are exposed.

Table 1 Length of black area at the tube tip of endotracheal tubes for neonatal use as produced by four major manufacturers

<table>
<thead>
<tr>
<th>Tube (ID)</th>
<th>2.5</th>
<th>3.0</th>
<th>3.5</th>
<th>4.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rusch</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>30</td>
</tr>
<tr>
<td>Portex</td>
<td>24</td>
<td>30</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>Vygon (st)</td>
<td>17</td>
<td>19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vygon</td>
<td>25</td>
<td>25</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>Mallinkrodt</td>
<td>30</td>
<td>30</td>
<td>30</td>
<td></td>
</tr>
</tbody>
</table>

Tube ID and length of black area expressed in mm, st, surfactant tube.
Table 1  Effect of antenatal steroids on respiratory distress syndrome (RDS) in premature twins

<table>
<thead>
<tr>
<th>Outcome measure</th>
<th>Optimal antenatal steroids</th>
<th>No antenatal steroids</th>
<th>Odds ratio (95% CI)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RDS</td>
<td>184/310</td>
<td>79/106</td>
<td>0.49 (0.27 to 0.91)</td>
<td>0.02</td>
</tr>
<tr>
<td>Surfactant use</td>
<td>162/333</td>
<td>67/108</td>
<td>0.58 (0.34 to 0.99)</td>
<td>0.05</td>
</tr>
<tr>
<td>Mortality</td>
<td>38/334</td>
<td>20/110</td>
<td>0.53 (0.26 to 1.08)</td>
<td>0.08</td>
</tr>
</tbody>
</table>

Results are number of twin infants affected over number of infants at risk, also expressed as a percentage. Odds ratio and p values were produced by logistic regression with standard errors adjusted for within pair correlation.

Progress in the management of disease in the newborn has carried with it a recognition of the substantial risk of injury to the immature nervous system. The aspiration to localise and prognosticate from neurological signs in the early newborn period is easily understood. The problem is that the signs available to be discerned are in themselves usually insufficient to allow precision. In addition, the child grows and develops, the range and complexity of skills are constantly changing, and the manifestations of the lesion(s) alters, or may become silent, often to reappear later as a different but nevertheless highly significant impairment.

The evaluation of the newborn nervous system was originally based upon concepts learnt from adult neurollogy. The baby was seen as demonstrating little or no cortical or cerebellar activity and the study of primary reflexes predominated. The approach of adult neurollogy, with emphasis on localisation of the lesion, becomes less applicable in the younger child. In the newborn period, focal insults to the brain often give rise to generalised disturbances and, contrarily, generalised disturbances may show focal deviations. Recognition of these phenomena has led to a progression from the concept of localisation based neurology to one which provides quantification of the assessment, a optimality score from the observed items of the assessment. This section deals with the results of a survey of 224 normal term infants. In this study each item of the scheme was plotted, and centile values (and thereby optimality scores) were computed. This provides quantification of the assessment, a sense of the range of findings to be expected, and can be useful in correlating lesions observed on neuro imaging with clinical findings. Chapter six deals with the scheme in relation to findings in infants with recognised brain lesions.

The book is not designed to be a text of neonatal neurology and readers looking for discussion of neurological disease states will be disappointed. As a description of a comprehensive and easily applied system of neonatal neurological examination the new edition succeeds admirably.
Respiratory distress syndrome and antenatal corticosteroid treatment in premature twins

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