LETTERS TO THE EDITOR

Low soluble FcRIII receptor demonstrates reduced neutrophil reserves in preterm neonates

EDITOR,—Studies of human neonate granulopoiesis have been hampered by the lack of a marker of overall neutrophil cell mass. Assumptions about neonate granulopoiesis have therefore largely been extrapolated from rat data. Direct measurement of total neutrophil cell mass (in terms of neutrophils by g body weight) in newborn rats has shown that they have about one quarter the neutrophil mass of adult animals and that their neutrophil mass increases, in response to insults by the time they are 4 weeks old.1 In addition, newborn rodents do not have the reserve pool of quiescent granulocyte progenitors, as found in adults, to recruit into production during sepsis. Circumstantial evidence for a similar immaturity of neutrophil production in human neonates comes from the low proportion of quiescent progenitors in cord blood and the frequent occurrence of postnatal neutropenia in preterm infants. Some additional insight comes from a study of mid-trimester abortuses,2 which showed minimal numbers of neutrophils in haemopoietic tissue before 24 weeks gestation. Until now, direct quantification of total body neutrophil cell mass in human neonates between 24 weeks and term has remained elusive.

In 1992 we reported3 that preterm neonates born before 32 weeks gestation have very low plasma concentrations of the soluble FcRIII receptor (sFcRIII). sFcRIII is the plasma form of the neutrophil membrane receptor FcγRIII which, together with other membrane receptors, binds to cell surface membrane antigens and initiates phagocytosis. We observed that, between 24 and 32 weeks gestation, sFcRIII concentrations are about 15% of normal adult values (mean (SE) 15.3 (1.3)%), while sFcRIII concentrations in fetal samples collected between 19 and 23 weeks are even lower (mean (SE) 8.3 (1.4)%). Between 33 and 36 weeks in utero, sFcRIII increases rapidly so that by 36 weeks term have concentrations similar to adults. In babies born before 32 weeks, sFcRIII concentrations gradually after birth to achieve adult concentrations by the fourth week of postnatal life. At the time, we interpreted these data to indicate reduced FcRIII production by individual cells and thus immaturity of neonate neutrophil function. Since we published these data, further work has clarified the origins and significance of plasma sFcRIII. These studies have shown that sFcRIII is derived from apoptotic neutrophils, and its concentration in plasma reflects the total body neutrophil cell mass as well as the overall production of neutrophils in the bone marrow.4 This new understanding of sFcRIII alters the interpretation of our original observation. It is now apparent that our data confirm what was previously only suspected: preterm human infants of less than 32 weeks gestation have reduced neutrophil production and a reduced total neutrophil cell mass at birth. Our data also show that neutrophil reserves remain low for about three weeks after preterm birth. Term infants, by contrast, have neutrophil production and stores that are similar to those of adults.

The reduced neutrophil mass of preterm neonates explains the common occurrence of postnatal and sepsis induced neutropenia. The relevance of this finding to the high sepsis incidence associated with preterm neonatal life is emphasised by a recent study of patients with idiopathic neutropenia, which has shown that a low sFcRIII concentration is a more accurate predictor of infection risk than the peripheral blood neutrophil count.5 Furthermore, this new insight into neonatal immune development suggests that stimulation of neutrophil production by colony stimulating factors early in postnatal life would reduce the incidence and severity of sepsis in preterm neonates by accelerating expansion of their deficient neutrophil reserves.

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REFERENCES
1 Erdman SH, Christensen RD, Bradley PP, Rothstein G. Supply and release of storage granules and initiating phagocytosis. We observed that, between 24 and 32 weeks gestation, sFcRIII concentrations are about 15% of normal adult values (mean (SE) 15.3 (1.3)%), while sFcRIII concentrations in fetal samples collected between 19 and 23 weeks are even lower (mean (SE) 8.3 (1.4)%). Between 33 and 36 weeks in utero, sFcRIII increases rapidly so that by 36 weeks term have concentrations similar to adults. In babies born before 32 weeks, sFcRIII concentrations gradually after birth to achieve adult concentrations by the fourth week of postnatal life. At the time, we interpreted these data to indicate reduced FcRIII production by individual cells and thus immaturity of neonate neutrophil function. Since we published these data, further work has clarified the origins and significance of plasma sFcRIII. These studies have shown that sFcRIII is derived from apoptotic neutrophils, and its concentration in plasma reflects the total body neutrophil cell mass as well as the overall production of neutrophils in the bone marrow.4 This new understanding of sFcRIII alters the interpretation of our original observation. It is now apparent that our data confirm what was previously only suspected: preterm human infants of less than 32 weeks gestation have reduced neutrophil production and a reduced total neutrophil cell mass at birth. Our data also show that neutrophil reserves remain low for about three weeks after preterm birth. Term infants, by contrast, have neutrophil production and stores that are similar to those of adults.

The reduced neutrophil mass of preterm neonates explains the common occurrence of postnatal and sepsis induced neutropenia. The relevance of this finding to the high sepsis incidence associated with preterm neonatal life is emphasised by a recent study of patients with idiopathic neutropenia, which has shown that a low sFcRIII concentration is a more accurate predictor of infection risk than the peripheral blood neutrophil count.5 Furthermore, this new insight into neonatal immune development suggests that stimulation of neutrophil production by colony stimulating factors early in postnatal life would reduce the incidence and severity of sepsis in preterm neonates by accelerating expansion of their deficient neutrophil reserves.

6 Homberg CHE, de Haas M, von dem Borne AEGKr. Clini-}
We agree that a randomised trial needs to be undertaken to prove that premedication has the desirable end point of reducing neonatal morbidity, particularly intraventricular haemorrhage.

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Premedication for intubation in neonates

EDITOR,—We were very interested in the recent papers by Bhutada et al and Whyte et al on the use of premedication for sedative intubation in neonates. It is now well accepted that term and preterm neonates tolerate awake intubation poorly, often exhibiting hypoxia, bradycardia, and systemic and intracranial hypertension during nasotracheal or orotracheal intubation.1 Analgesia and sedation are still used infrequently in nurseries for intubation and other “routine”, but invasive, therapeutic or diagnostic procedures.2

We recently performed a randomised, double blind, placebo controlled trial to assess whether sedation with midazolam in premature infants would improve physiological stability and the success rate of endotracheal intubation. Eight premature infants underwent 16 intubation procedures after being randomly assigned to one of three groups: I (n = 3), the control group, received placebo only; II (n = 6) received atropine and placebo; III (n = 7) received atropine and midazolam. Infants could be randomised again to a different group after completion of each intubation procedure. Heart rate, blood pressure, and oxyhaemo-globin saturation were recorded at 10 minute intervals for each infant. The study was terminated when the study data were reviewed early because of concerns over adverse events. The number of episodes of oxyhaemoglobin desaturation around the time of intubation was significantly greater in group III (86%) than in group I (0%; p = 0.01). Cardiopulmonary resuscitation was required in 29% of group III compared with 0% of group II (p = 0.16).

In our trial, premature infants who received midazolam were at increased risk of oxyhaemoglobin desaturation during tracheal intubation. There was also a trend toward an increased need for cardiopulmonary resuscitation in these infants. Although regrettably underpowered due to the early termination of the study, our findings indicate caution in the use of midazolam as routine premedication for tracheal intubation in premature infants. Further investigation with a larger number of infants is needed to clarify its safety and efficacy profile.

Although we agree that an important goal in neonatal medicine is to minimise pain and stress, more information is needed on the effects of intravenous anaesthetic agents, such as thiopental and methohexitol, in both stable and unstable preterm neonates before these medications can be recommended for routine use. Further randomised controlled trials are needed to help formulate specific premedication guidelines for the variety of noxious procedures that infants undergo in neonatal intensive care units.

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Incidence of severe retinopathy of prematurity

EDITOR,—We were interested to read the article of Vyas et al on the incidence of severe retinopathy of prematurity (ROP) in 11 neonatal units in five cities in England in 1994. We have published similar data from eight neonatal units in New South Wales (NSW) Australia in 1993 and 1994.3 These data were prospectively collected in the NSW Neonatal Intensive Care Unit’s data collection and is stored and maintained in the NSW Centre for Perinatal Health Services Research, University of Sydney, NSW.

For infants of < 29 weeks gestation, there was no significant difference in severe ROP (≥ stage 3) between the five cities in England and NSW (table 1).

Unlike Vyas et al, we could not find an association between improved survival and the development of severe ROP. In six of the neonatal units in our study (two have been excluded from this analysis as they are children’s hospitals and have very few small premature infants), survival in infants of < 27 weeks gestation ranged from 51.3% to 68.8%. The percentage with severe ROP for the two units with the lowest and highest survival was 15 (3/20) and 24 (5/21) respectively, while the range of severe ROP in the six neonatal units was 15% (3/20) to 36% (9/25).

In infants of 27–28 weeks gestation, survival ranged from 85.1% to 96.7% and the percentage with severe ROP for the two units with the lowest and highest survival was 7 (4/56) and 3 (2/58) respectively, while the range of severe ROP in this group of infants was 2% (1/50) to 7% (4/56) (unpublished observations). We have also shown that, despite an increase in survival of preterm infants following the introduction of surfactant, there was no significant impact on the incidence or severity of ROP.

In infants of 29–31 weeks gestation, six of 443 infants (1.4%) developed severe ROP and one required Cryo/Laser treatment.4 This infant was 30 weeks gestation with a birth weight of 1305 g. We therefore agree with Vyas et al that there should be no reduction in the upper limit of gestation or birth weight for screening for ROP.

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Blood pressure standards for very low birthweight infants

EDITOR,—Based on careful invasive blood pressure measurements in a cohort of 61 very low birthweight (VLBW) infants monitored for up to 6 hours of life, Lee et al report on blood pressure standards in this population. The quality of the blood pressure readings was assessed using continuous video recordings during the first wave forms. The inclusion of a relatively large number (n = 28) of extremely low birthweight infants is another strength of this study.

In an attempt to define normal blood pressure values in a population that by definition is not “normal”, the authors have excluded very ill infants and infants requiring inotropic support. They have also excluded infants who developed intraventricular haemorrhage (IVH) grades III and IV during the first 6 hours of life. They suggest that the lower limits of mean arterial blood pressure (MAP) for infants between 26 and 32 weeks gestation are numerically similar to the gestational ages. With the exclusion of infants who developed severe IVH, it became impossible to detect any association between blood pressure and IVH. Decreases in cerebral blood flow may play an important role in the pathogenesis of IVH, and, although this is not a reliable parameter to assess cerebral blood flow,5 at least two published studies6 7 have shown that the incidence of IVH was higher in infants with lower MAP. Perhaps the authors could indicate how many infants...
with IVH grades III and IV were excluded and what blood pressures were recorded in these infants. For similar reasons, it remains uncertain whether the proposed blood pressure standards are applicable to very ill infants or infants who require isotropic support. It is conceivable that cerebral vascular resistance in sick preterm infants differs from that in the study patients, and consequently the application of the suggested blood pressure standards to sick preterm infants would result in different—and possibly inadequate—cerebral blood flows.

The data presented add to our knowledge on blood pressure in VLBW infants, but do not allow the conclusion that the suggested blood pressure standards are safe for all VLBW infants. THOMAS M BERGER

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Drs Juin, Rajadurai, and Wee respond:

We would like to thank Dr Berger for his comments. Our objective was to define the normal range of blood pressures for very small infants and this would logically exclude those with IVH and those on inotropes, both of which could affect blood pressure.

We did not compare infants with and without IVH because that was not our aim. There may very well be a correlation between hypotension and IVH, but studies looking at this phenomenon have not been reported. Tyszczuk et al. determined that cerebral blood flow was independent of blood pressure in preterm infants, but again the selection of a mean arterial pressure of 30 mm Hg as the cut off is open to debate. We think that choosing such an arbitrary number was not appropriate and more such studies are needed with particular attention being paid to the very different normal blood pressures among preterm infants with various birth weights and gestational ages.


Patient triggered ventilation in neonatal respiratory distress syndrome

EDIToR.—Baumer reports the results of a large multicentre study comparing the effects of patient triggered ventilation (PTV) with conventional ventilation (IMV).1 There appears to be no benefit from PTV compared with IMV in death rate, development of chronic lung disease, pneumothorax rates, and cerebral ultrasound abnormality. In addition, because of an increased trend toward a higher pneumothorax rate, Baumer concludes that, at present, PTV delivered with either the SLE 2000 or the Dräger babylog 8000 should not be recommended for infants of less than 28 weeks gestation with respiratory distress syndrome (RDS).

However, we are concerned that this may be a premature conclusion given the significant difference in PTV delivered by the two main ventilators used and the potential heterogeneity of clinical practice within the different centres involved, despite agreed ventilation protocols. Dimitriou et al. showed that neonates and infants trigger a significantly lower proportion of breaths using the SLE 2000, an airway pressure triggered ventilator that provides synchronised intermittent positive pressure ventilation (SIPPV). Attempts to optimise the trigger rate of the SLE 2000 ventilator by increasing pressure sensitivity often results in an increased trigger rate, as discussed by Baumer. Therefore the PTV modes of the two ventilators are substantially different. This prompts us to ask whether the findings of this multicentre study are only applicable to PTV provided by the SLE 2000? Would there have been a different outcome if all the triggered babies had received SIPPV?

It is not known how many of the 40/213 babies of less than 28 weeks gestation who had pneumothorax were receiving SIPPV. As only 11% (52/465) of all triggered babies ever received SIPPV, we surmise that very few of the 40 were ventilated in this way. Is it fair to conclude that the Dräger babylog 8000 has a trend to pneumothorax on SIPPV?

In a separate smaller study, Baumer also reports 120 patients in three centres randomly assigned to either the Dräger babylog 8000 or the SLE 2000 ventilator, and found a non-significant trend to higher pneumothorax rate, chronic lung disease, and death for the former group. But we are not told how many of the babies were actually on trigger mode (PTV or SIPPV). They should all have been receiving IMV or IMV + trigger (SIPPV) ventilation. What is the trigger rate of the SLE 2000? Is it possible that SIPPV is not being tested?

Further evidence was that a significantly higher percentage (124/465 versus 108/40) of all triggered babies that departed from their assigned mode of ventilation, 45 of these failed to trigger their ventilator. Were they all on the SLE 2000 ventilator, as Dimitriou et al. would predict?

Finally, we note that 10 of the 22 neonatal units each recruited less than 20 patients over the four year period, one contributing only one patient. Could the technique of PTV ventilation in units contributing so few be different from those enrolling 60–136 patients over the same period, despite prior visits from the trial coordinator? Would a logistic regression for morbidity against centre e0000? Would a logistic regression for morbidity against centre four possible factors such as gestation.

We do not compare those using the SLE 2000 and those using the Dräger babylog 8000, because with present technology we cannot be extrapolated to another. However, both ventilators were in PTV mode (sometimes referred to as synchronised intermittent positive pressure ventilation, assist control, or synchronised assisted ventilation in infants), with the ventilator set to trigger at an inspiratory effort. In this study were ventilated with SIMV and IMV modes (intermittent mandatory ventilation) where the baby’s breaths, selected during a “time window”, trigger the ventilator with the preset number of breaths per minute.

The results were reported in the way that they were for a reason. The original study design allowed a four way randomisation between the two makes of ventilator and the two modes of ventilation. As was reported, only three centres had enough of both ventilators to allow this to occur. Other centres ventilating infants with the Dräger babylog had a two way randomisation between PTV and IMV.

Therefore the possibility of confounding by differences in practice between centres needed to be excluded. If the centres using the SLE 2000 were as well as the SLE 2000 different outcomes from those using only one make of ventilator, this may have led to inappropriate conclusions being drawn if all infants being ventilated with one ventilator were simply compared with those being ventilated with another. A logistic regression model was therefore used to allow for possible centre effects (as well as other significant factors such as gestation).

With that caveat, I have contacted the numbers from the database giving details of the crude observed rates of pneumothorax in the infants of less than 28 weeks gestation, separately reported for the two makes of ventilator (table 1).

The observed rate of pneumothorax was substantially (but not significantly, χ² 2.8, p > 0.05 < 0.1) higher in the infants ventilated in PTV mode than in IMV mode using the SLE 2000. Although the numbers are small, the observed pneumothorax rate was higher in infants ventilated with the Dräger babylog 8000 than in those ventilated with the SLE 2000 ventilator. It therefore seems somewhat illogical to recommend caution in using...
PTV, patient triggered ventilation; IMV, intermittent mandatory ventilation.

Table 1 Rates of pneumothorax in infants of less than 28 weeks gestation using two different makes of ventilator

<table>
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<tr>
<th></th>
<th>PTV</th>
<th>IMV</th>
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<tr>
<td>Dräger without pneumothorax</td>
<td>19 (20)</td>
<td>20 (20)</td>
</tr>
<tr>
<td>SLE without pneumothorax</td>
<td>154 (114)</td>
<td>14 (114)</td>
</tr>
<tr>
<td>SLE with pneumothorax</td>
<td>35 (18.5%)</td>
<td>14 (10.9%)</td>
</tr>
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</table>

PTV, patient triggered ventilation; IMV, intermittent mandatory ventilation.

the SLE 2000 in PTV mode in infants less than 28 weeks gestation, but not extend this caution to ventilating infants with the Dräger babylog 8000.

Given that none of these differences were statistically significant, no clear recommendation can be given. This is why the wording used in the publication was “it might be prudent to avoid…”.

As regards the number of infants departing from their assigned mode of ventilation, several points should be emphasised. The study protocol permitted changing the mode of ventilation at the discretion of the attending clinicians. This was inevitably interpreted differently by each clinical team. Departure from the assigned mode of ventilation was not an intended outcome, and it is evident that this occurred more commonly in the more immature infants and those that subsequently died. High rates of departure from the assigned mode cannot therefore readily be interpreted as evidence of failure of the assigned mode. Table 2 shows the numbers of infants of all gestations departing from the assigned mode of ventilation.

There was therefore a higher crude rate of departure from the assigned mode of ventilation in infants ventilated with the Dräger babylog 8000, with a similar proportion departing from the SLE.

It would be difficult to interpret the pneumothorax rates for infants who were actually being ventilated with their assigned mode of ventilation. Some infants were switched to another mode of ventilation after sustaining their pneumothorax. Most of the pneumothoraces occurred while infants were receiving their assigned mode of ventilation, and this included infants being trigger ventilated using the Dräger babylog 8000.

Burmester and Petros ask whether centres contributing few patients may have higher pneumothorax rates. The 139 infants randomised within the same as the centres contributing more infants.

In summary, there is no evidence from this study of any trend towards better outcomes with the Dräger babylog 8000 ventilator, although the small numbers enrolled make any conclusions less robust. There is evidence that suggests there may have been a short term reduction in pneumothorax rates from the educational package offered at the start of the trial.

In conclusion, there was no convincing evidence of a beneficial effect of a policy of using PTV in preterm infants with RDS with the ventilators used. Regular attention to staff education on ventilator techniques is recommended.

We would like to use this opportunity to pay tribute to the two trial coordinators (Sue Ellis and Tom Mill), to the trial statistician (David Wright), and to the data monitoring committee (David Field and Diana Elbourne), whose details were inadvertently omitted from the final paper and without whom, together with the trial collaborators, the study would not have been possible.

Pyridoxine dependent epilepsy: a suggestive electroclinical pattern

Editor,—There are several problems with describing the electroclinical pattern in pyridoxine dependent seizures. One is defining the electrical features. Until now all published reports (including four of the five cases of Nabbout and colleagues) have described the electroencephalogram (EEG) in neonates who have received other anticonvulsants beforehand. It seems that anticonvulsants could affect the EEG, as suggested by the infant with the most pronounced burst suppression pattern in the paper of Nabbout and colleagues. The other method of assessing the electrical pattern is on withdrawal, when we, like others, found that a continuous or intermittent high voltage slow wave pattern with or without spikes was typical. It would be very useful to know the EEG features of the patient who did not receive any other drugs before pyridoxine, as this would be the first description of the true electroclinical pattern in pyridoxine dependent seizures.

This study shows electroencephalographie use of EEGs in neonatal units. Very few of the neonates reported in the literature or in the UK study1 had EEGs before receiving pyridoxine. Presumably this reflects difficulties in obtaining EEGs out of hours or there is no facility attached to the neonatal unit, as well as the desire to treat without delay. As a result, for some neonatal units it may be misleading and (b) early treatment does appear to be beneficial.

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Parental visiting in neonatal units

Editor,—We read with interest the paper by Cattini et al. Although policy on parental visiting is an easier issue to evaluate, parental participation in decision making, particularly in decisions with strong ethical overtones, is much more complex. It is difficult to evaluate with accuracy, and by its nature so much more controversial. The paper does not stress that data collected from each participating unit, through a structured questionnaire completed by the unit coordinator, represent policies—that is, the intention and stance of each unit towards the particular issue for evaluation. Data collected through questionnaires and interviews involving both unit staff and parents would have provided a better understanding of the actual practice of each participating unit.

No unit from Greece took part in the study by Cattini et al., but Greece is briefly mentioned in the Discussion, using results from a previous study in which, in a sample...
of 38 units from 11 European countries, it was shown that the nine units that imposed visiting restrictions were in France, Greece, Italy, and Portugal.

We would like to provide further information on visiting policy in Greek neonatal intensive care units (NICUs). There are 15, two of which are private; 12 are attached to maternity hospitals and the remaining three are in children’s hospitals and accept adult patients. In all but one (Agia Kyriakou Children’s Hospital), visiting restrictions are imposed. These allow parents only, and the usual practice is 30 minutes to one hour visiting time in the morning and afternoon (except mothers). The most common reasons given for imposing restrictions are increased danger of infection and a disruptive effect on the unit.

We conducted a survey, through questionnaire and interview, of parents with a baby who had been cared for in another NICU that imposed visiting restrictions before it was transferred to our NICU and/or parents who had a previous baby in another NICU that imposed restrictions. The overwhelming majority (98.6%) said that they preferred our liberal policy on visiting. One mother of a preterm baby with bronchopulmonary dysplasia said that “if I had delivered at term I would be with my baby; if I had not delivered prematurely I would also be with my baby (in my womb); now that I have delivered prematurely why can’t I be with my baby?”

We conclude that in Greece there is a demand for unrestricted parental visiting, but most Greek NICUs do not meet this demand for reasons not based on medical or sociological evidence. It is worth noting that, in Greece, infants beyond the neonatal period have been admitted to children’s wards with their mothers for many years.

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Ureaplasma colonisation and chronic lung disease in neonates

EDITOR.—We read the article by Hannaford et al with interest. We would like to point out that in our recent retrospective analysis, our findings of the colonisation rate of genital mycoplasmas (Ureaplasma urealyticum and Mycoplasma hominis) in the respiratory tract of premature infants of 27% were similar to their study. We also found a higher incidence of chronic lung disease (CLD) in our cohort colonised with genital mycoplasmas.

We are, however, intrigued by the implication of a “protective effect” of Ureaplasma urealyticum colonisation on respiratory distress syndrome (RDS) in this study. The hypothesis that this may be related to the “stimulatory effect of subacute intrauterine infection on lung maturation...” is interesting but unsubstantiated. Current evidence suggests that the sicker infant with RDS has an increased early inflammatory response and a higher rate of CLD. In the present study, the incidence of RDS in Uu colonised infants was lower—but their incidence of CLD at 36 weeks was higher. Were there other factors—for example, increased incidence of PDA, more IV fluid use, that might have predisposed these infants to develop CLD? Another issue is that in the multivariate analyses, Uu colonisation was a significant risk factor for CLD only in singleton infants, but the relationship did not hold true when all infants were analysed. Based on these observations, we wonder if the “protective effect” is real or a statistical aberration? More research needs to be done to study this “protective effect” of Uu colonisation on RDS before any conclusions can be drawn.

The treatment of Uu colonisation was not discussed in this paper. In our experience, erythromycin alone was not effective in clearing the organism from the respiratory tract. We found a short course of postnatal steroids alone or in combination with erythromycin more effective.1 We wonder if infants receiving postnatal steroids were confounders in the discrepancy between the incidence of RDS and CLD.

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Dr Gilbert et al respond:

EDITOR.—We thank Drs Bhandari and Hussain for their interest in our paper. We too were intrigued by the apparently contradictory effects of ureaplasma colonisation on the lungs of affected infants and acknowledge that our hypothesis is unsubstantiated. The possibility that a subacute intrauterine infection could stimulate endogenous surfactant production is a plausible explanation for the inverse relationship between ureaplasma colonisation and RDS but was too strong to be dismissed as a statistical aberration.

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Editor’s note

References 22–30 of the paper by Hannaford et al were incorrect, and each correlate with the number below in the text. Thus reference 22 in the reference list corresponds with reference 21 in the text, and so on. This correction appears online [Arch Dis Child Fetal Neonatal Ed: first published as 10.1136/fn.83.2.F160 on 1 September 2000].

The authors of the paper “Risk adjusted and population based studies of the outcome for high risk infants in Scotland and Australia” Arch Dis Child Fetal Neonatal Ed 2000;82:118–123 have asked us to publish the following corrected version of table 6. The authors apologise for the error.

<table>
<thead>
<tr>
<th>Relative risk of neonatal death for very low birthweight infants in Scotland and England and Wales compared with Australia</th>
<th>500–999 g</th>
<th>1000–1499 g</th>
<th>500–1499 g</th>
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<tbody>
<tr>
<td>Relative risk of neonatal death</td>
<td>95% confidence interval</td>
<td>Relative risk of neonatal death</td>
<td>95% confidence interval</td>
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<tr>
<td>Scotland vs Australia 1993–94</td>
<td>1.24</td>
<td>1.09–1.42</td>
<td>1.20</td>
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<tr>
<td>Scotland vs Australia 1993–96</td>
<td>1.48</td>
<td>1.35–1.63</td>
<td>1.34</td>
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<tr>
<td>England and Wales vs Australia 1994</td>
<td>1.15</td>
<td>1.07–1.24</td>
<td>1.18</td>
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<tr>
<td>Scotland vs Australia 1993–96</td>
<td>1.25</td>
<td>1.19–1.32</td>
<td>1.23</td>
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The journal would like to apologise for this error.