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 per kg 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 only slowly from birth to adulthood. Prematurity is associated with severe infections and PMN dysregulation in neonates born before 32 weeks gestation. Our data also show that neutrophil reserves remain low for about three weeks after birth. Term infants, by contrast, have a reduced total neutrophil cell mass at birth. Term infants born before 32 weeks gestation have very low plasma concentrations of the soluble FcRIIa receptor (sFcRIIa). sFcRII is the plasma form of the neutrophil membrane receptor FcγRII which, together with other membrane receptors, is responsible for binding opsonised particles and initiating phagocytosis.

In 1992 we reported that preterm neonates born before 32 weeks gestation have very low plasma concentrations of the soluble FcRIIa receptor (sFcRIIa). This reduction in sFcRII concentration demonstrates reduced 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 prematurity is emphasised by a recent study of patients with idiopathic neutropenia, which has shown that a low sFcRIIa concentration is a more accurate predictor of infection risk than the peripheral blood neutrophil count. Further, 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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Chasing hyponatraemia in preterm infants

Editor,—In a recent issue, Hartnell et al 1 described a baby born at 27 weeks gestation with severe oxygen dependency, body weight, and body composition of preterm infants. According to them, an overzealous approach to correcting hyponatraemia in preterm infants is not scientific. In the same context, I would like to describe a case of a preterm infant who developed symptomatic patent ductus arteriosus after supplementation with sodium.

The infant was a 1060 g baby boy born at 29 weeks of gestation with no significant antenatal history. The first five days were unre- markable. On day 6 of life, he was noted to have hyperglycaemia, which persisted, requiring insulin drip. At the same time, his hyponatraemia was corrected with extra sodium supplementation. On day 11, he was noted to have a murmur, which later was confirmed by echocardiography as patent ductus arteriosus. Table 1 summarises the events.

The case clearly indicates a need for a conservative approach to hyponatraemia in preterm infants, as "chasing" it may lead to fluid retention and development of patent ductus arteriosus.

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Susxamethonium is safe in safe hands; mivacurium should also be considered

Editor,—We thank Whyte et al for detailing the premedication policies for intubation in United Kingdom neonatal units. There is a wide disparity in both the type and dosage of drugs. Whyte et al mention that, as a muscle relaxant, suxamethonium has, important safety benefits. It has a very rapid onset and offset of action, and therefore there is a “get out clause” for the difficult intubation or intubation by an inexperienced operator. However, other considerations need to be taken into account when suxamethonium is used.

Firstly, intravenous premedication is contraindicated in conditions known to be associated with difficult intubations, such as the Pierre Robin sequence. Further, suxamethonium is a depolarising muscle relaxant and therefore can cause sinus bradycardia. Atropine can be given in an attempt to avoid this.

Suxamethonium causes depolarisation at the neuromuscular junction, rarely leading to hyperkalaemic induced cardiac arrest. Undiagnosed myopathic conditions were found in a number of affected children, and paediatric licensing for this drug was therefore removed in the United States. However, no other very rapidly acting drug has been manufactured, and, because of its popularity, suxamethonium was reinstated. Before its use, a detailed family history for myopathic conditions must be taken. In our neonatal unit, we have started to use fentanyl, an opioid, with mivacurium as the muscle relaxant.  

The disadvantage of mivacurium is that it is only a rapidly acting muscle relaxant. Its major advantage is that it is a non-depolarising muscle relaxant and therefore carries no risk of life threatening arrhythmias. The routine use of atropine is not necessary.
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 selective 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. Analgesia and sedation are still used infrequently in nurseries for intubation and other “routine”, but invasive, therapeutic or diagnostic procedures.

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 oxyhaemoglobin saturation were recorded at 10 minute intervals for each infant. The study was terminated when the 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% in 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 because of early termination, our study indicates 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 our study (two have been anonymous) with the lowest and highest survival was 7 (4/56) and 3 (2/58) 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. 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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Letters, Correction

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 in the first 24 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 of 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 week of life. They suggest that the lower limits of mean arterial blood pressure (MAP) for infants between 26 and 32 weeks of 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 mean arterial blood pressure is not a reliable parameter to assess cerebral blood flow, at least two published studies 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 intropopic 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.

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F162

Letters, Correction

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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 hypoten-
sion and IVH, but studies looking at this controversy as to what constitutes hypoten-
sion are required, and extend his conclu-
sion. Their letter raises questions about the
to PTV or SIPPV. Therefore the possibility of confounding by differ-
differences in practice between centres needed to be excluded. If the centres using the SLE as well as the Dräger (positive pressure ventilation, assist control, or synchronised assisted ventilation in infants), with the ventilator set to trigger at each inspiratory effort. No infants in this study were ventilated with SIMV. With the Dräger, we assume the indication for PTV was an intermittent mandatory ventilation) where the baby's breaths, selected during a “time window”, trigger the ventilator with the pressure of breaths to ventilate.

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 trigger systems. Other centres ventilating infants with the Dräger babylong 8000 has a trend to pneumothorax on SIPPV. Differences found were small, the observed pneumothorax rate was higher in infants ventilated with the Dräger babylong 8000, rather than SIPPV from the Dräger babylong 8000. Although the numbers are small, the observed pneumothorax rate was substantially (but not significantly, 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 babylong 8000 than in those ventilated with the SLE 2000. If it therefore seems somewhat illogical to recommend caution in using


Dr Baumer responds on behalf of the trial collaborators:

The interest displayed in the trigger ventilation trial by Burmester and Petsos is wel-
1 Tyszczuk L, Meek J, Elwell C, Wyatt JS. Cerebral blood flow is independent of mean arterial blood pressure in preterm infants undergoing intensive care. Pediatr

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 ultrasonic 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 babylong 8000 should not be recom-

However, we are concerned that this may be a premature conclusion given the signifi-


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Table 2 Numbers of infants of all gestations departing from the assigned mode of ventilation

<table>
<thead>
<tr>
<th></th>
<th>PTV</th>
<th>IMV</th>
</tr>
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<tbody>
<tr>
<td>Dräger without pneumothorax</td>
<td>19 (20%)</td>
<td>20 (17%)</td>
</tr>
<tr>
<td>SLE without pneumothorax</td>
<td>154 (18.5%)</td>
<td>114 (10.9%)</td>
</tr>
<tr>
<td>SLE with pneumothorax</td>
<td>35 (26.1%)</td>
<td>47 (34.6%)</td>
</tr>
</tbody>
</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. 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 triggered by the SLE ventilator.

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 their assigned ventilator.

Bermote and Petsos ask whether centres contributing few patients may have higher morbidity rates, correcting for potential confounding factors by using a logistic regression model. The pneumothorax rate from centres contributing less than 20 patients was the same as the centres contributing more infants.

We have used a model to identify outcome differences in infants randomised within three months of the first infant being entered into the study, correcting for individual centre effects, gestation, birth weight, and mode of ventilation. There was no significant difference in rates of death and chronic lung disease, abnormal cranial ultrasound scan, or duration of ventilation. However, an appreciable and statistically significant difference was found for pneumothorax rates. The 139 infants randomised within three months had a pneumothorax rate of 5% compared with a rate of 13% for those randomised more than three months into the trial (odds ratio 0.38; 95% confidence intervals 0.12 to 0.74; p = 0.009). This was seen equally for both modes of ventilation.

This finding suggests that the initial educational visit by the trial coordinator had a beneficial effect on ventilator management which disappeared as infants continued to be enrolled.

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.

I 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. 1 One is defining the electroclinical 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 before hand. It seems unlikely 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. This would then in effect mean that the electrical pattern is on withdrawal, when we, like others, found that a continuous or intermittent high volt slow wave pattern with or without spikes was typical. 2 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 electrical pattern in pyridoxine dependent seizures.

This study builds on the use of EEGs in neonatal units. Very few of the neonates reported in the literature or in the UK study 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 difficult to detect a specific electroclinical pattern.

A second issue is that the clinical features are not very specific. The neonatal presentation can be: as an acute encephalopathy, followed by later seizures; as seizures accompanied by encephalopathy (both of which introduce a wide differential diagnosis); or, as seizures alone; or, more unusually, as apparent acute abdomen obstruction or respiratory distress, usually accompanied by irritable behaviour, again followed by seizures. In some reports, seizures have been precipitated by sudden sounds or movements, although some of these could be an exaggerated startle response. Although we too found that a variety of seizures, especially generalised tonic and generalised clonic, occurred in most of our cases, some only had one or two seizure types. In the UK study, five of the 20 definite and probable cases with an early onset were reported to have a single seizure type (unpublished data).

The third concern is that Nabbout and colleagues did not include later presenting cases (older than 28 days) in their report. These also appear to be pyridoxine dependent as judged by trials of withdrawal and occurrence in siblings. They accounted for three of the 23 definite and probable cases in the UK population study, although a further five had early seizures that responded to routine anticonvulsants and then remained seizure free for several weeks. In these, both the clinical and electrical features can differ from neonates. For example, the inter-ictal EEG can be normal, or show focal or generalised spikes or sharp waves with or without high voltage slow waves. In these cases, the clinical features include recurrent episodes of status and less varied seizure types. Concentrating on a possible neonatal pattern risks overlooking such cases.

As Nabbout and colleagues emphasise, there needs to be a high index of suspicion. Clinically it is important that pyridoxine dependency should be considered in all early childhood seizures, because (a) 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 Cuttini et al. 1 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 questionnaire 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 Cuttini et al., 2 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 distant referrals. Of the nine units (Aglaia 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 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 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 parent 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 Utu 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 predisposed these infants to develop CLD? Another issue is that in the multivariate analyses, Utu 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 Utu colonisation on RDS before any conclusions can be drawn.

The treatment of Utu 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. We wonder if infants receiving postnatal steroids were confounders in the discrepancy between the incidence of RDS and CLD.

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De 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 significantly lower incidence of respiratory distress syndrome (RDS) in these infants. The fact that intrauterine infection is low grade, may also mean that delivery is delayed for long enough for exogenous steroids to take effect.

The development of chronic lung disease (CLD) is dependent on many factors, and, in our study, it correlated less closely with ureaplasma infection than did RDS. We also noted that other effects of multiple gestation outweighed those of ureaplasma infection on the development of CLD in twins. Presumably, the lower rate of RDS in ureaplasma-infected singleton infants reduced their risk of CLD, directly or indirectly. If so, there must be other respiratory insults severe enough to offset this effect. We were unable to identify factors associated with CLD from RDS and ureaplasma infection. In particular, the incidence of PDA did not differ between infants with and without CLD.

We postulate that the low grade inflammatory effect of intrauterine ureaplasma infection could cause significant irreversible lung damage and predispose to CLD, as well as stimulating the production of surfactant. The outcome is unlikely to be affected by erythromycin therapy after birth, except in the minority of infants in whom there is frank pneumonitis, with progression after birth.

We have not studied the effects of postnatal steroids, but this is unlikely to be a significant confounding factor since RDS was treated before the study was done, steroid therapy was not given until after four weeks of age, when CLD was already established.

Although we agree that further research is needed to confirm our findings, we believe that the inverse relationship between ureaplasma colonisation and RDS 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 [jdm.bmj Journals.com/cgi/content/full/fetalneonatal;81/3/F162/DC1]
The journal would like to apologise for this error.

Dr Gilbert et al respond:

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.

Dr Gilbert et al respond:

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CORRECTION

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Table 6 Relative risk of neonatal death for very low birthweight infants in Scotland and England and Wales compared with Australia

<table>
<thead>
<tr>
<th>500–999 g</th>
<th>1000–1499 g</th>
<th>500–1499 g</th>
</tr>
</thead>
<tbody>
<tr>
<td>Relative risk of neonatal death</td>
<td>95% confidence interval</td>
<td>Relative risk of neonatal death</td>
</tr>
<tr>
<td>Scotland vs Australia 1993–94</td>
<td>1.24</td>
<td>1.09–1.42</td>
</tr>
<tr>
<td>Scotland vs Australia 1993–98</td>
<td>1.43</td>
<td>1.35–1.63</td>
</tr>
<tr>
<td>England and Wales vs Australia 1994</td>
<td>1.15</td>
<td>1.07–1.24</td>
</tr>
<tr>
<td>England and Wales vs Australia 1993–96</td>
<td>1.25</td>
<td>1.19–1.32</td>
</tr>
</tbody>
</table>