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 per 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 by about a factor of two by the time they are 4 weeks old. 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 abortions, 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 reported that preterm neonates born before 32 weeks gestation have very low plasma concentrations of the soluble FcRRII receptor (sFcRII), sFcRIII is the plasma form of the neutrophil membrane receptor FcγRIII which, together with other membrane receptors, is responsible for binding opsonised particles and initiating phagocytosis. We observed that, between 24 and 32 weeks gestation, sFcRII concentrations are about 15% of normal adult values (mean (SE) 19.3 (3.1)%), 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 babies born 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 neonatal neutrophil function.

Since we published these data, further work has clarified the origins and significance of plasma sFcRII. 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.

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 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 and the frequent prematurity is emphasised by a recent study of patients with idiopathic neutropenia, which has shown that a low sFcRII concentration is a more accurate predictor of infection risk than the peripheral blood neutrophil count. 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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Suxamethonium 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 non-depolarising muscle relaxant and therefore carries no risk of life threatening arrhythmias. The routine use of atropine is not necessary.

Table 1 Summary of the events

<table>
<thead>
<tr>
<th>Day no</th>
<th>Total fluids (mL/kg)</th>
<th>Urinary output (mL/kg)</th>
<th>Sodium input (mmol/kg/day)</th>
<th>Serum sodium (mmol/L)</th>
<th>Weight (g)</th>
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<td>3</td>
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<td>15</td>
<td>131</td>
<td>900</td>
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<td>11</td>
<td>206</td>
<td>6.1</td>
<td>15</td>
<td>136</td>
<td>940 (PDA)</td>
</tr>
</tbody>
</table>
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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Birmingham Heartlands Hospital


Preme...
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.

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Switzerland


Drs Juin, Rajadurai, and Wee respond:

We would like to thank Dr Berger for his comments. Our objective was to define the standards for very low birthweight infants during the first year of life. 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.

We would like to thank Dr Berger for his comments. Our objective was to define the standards for very low birthweight infants during the first year of life. 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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Patients triggered ventilation in neonatal respiratory distress syndrome

EDITORS—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 ultrasonography 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 significantly different 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 outcome not 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). Would there have all been receiving IMV of the Dräger babylog 8000? Therefore is it possible that SIPPV is not being tested?

A further finding was a significantly higher death rate (124/463) of 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 contributing 60–136 patients over the same period, despite prior visits from the trial coordinator? Would a logistic regression for morbidity against centre and other variables chosen for the study model therefore used to allow for inappropriately 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 extracted 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 were small, the observed pneumothorax rate was higher in infants ventilated with the Dräger babylog 8000 than in those ventilated with the SLE ventilator. If it therefore seems somewhat illogical to recommend caution in using...
PTV, patient triggered ventilation; IMV, intermittent mandatory ventilation.

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

<table>
<thead>
<tr>
<th>Make</th>
<th>PTV</th>
<th>IMV</th>
</tr>
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<tbody>
<tr>
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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 remaining with the Dräger 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 the Dräger ventilator.

Burr mest and Petros 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 0.30; 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 beforehand. It seems probable 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 is why another method of asssessing the EEG is on withdrawal, when we, like others, found that a continuous or intermittent high voltage slow wave pattern with or without spikes was typical.1 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 also suggests a low prevalence 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 the fact 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 difficulty is that the clinical features are not very specific. The neonatal presentation can be: as an acute encephalopa-thy, followed by later seizures; as seizures accompanied by encephalopathy (both of which introduce a wide differential diagnosis); as seizures alone; or, more unusually, as apparent acute abdominal 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 these 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. 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) delay in starting pyridoxine 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 is 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 Cuttini et al., but Greece is briefly mentioned in the Discussion, using results from a previous study in which, in a sample

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Letters, Correction

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

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Letters, Correction

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

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<td>15</td>
<td>15</td>
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<tr>
<td>Dräger departing</td>
<td>31</td>
<td>27</td>
</tr>
<tr>
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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 urgent referrals. About one (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 (excluding 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 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.

**H D DELLAGRAMMATICAS**

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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,1 our finding of the colonisation rate of genital mycoplasmas (Ureaplasma urealyticum and Mycoplasma hominis) in the respiratory tract of premature infants of 27% was similar to their study. We also found a higher incidence of chronic lung disease (CLD) in our cohort colonised with genital mycoplasmas.7

We are, however, intrigued by the implication of a “protective effect” of Ureaplasma urealyticum or colonisation on respiratory distress syndrome (RDS) in this study. The hypothesis that this may be related to the “stimulatory effect of subacute intratracheal 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.1 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 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.3 We wonder if infants receiving postnatal steroids were confounders in the discrepancy between the incidence of RDS and CLD.

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**CORRECTION**

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:F118-123 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>
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<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>
</tr>
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<td><strong>Scotland vs Australia 1993–94</strong></td>
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<td>1.09–1.42</td>
<td>1.20</td>
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<tr>
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<td>1.35–1.63</td>
<td>1.34</td>
</tr>
<tr>
<td><strong>England and Wales vs Australia 1993–94</strong></td>
<td>1.15</td>
<td>1.07–1.24</td>
<td>1.18</td>
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<tr>
<td><strong>England and Wales vs Australia 1993–96</strong></td>
<td>1.25</td>
<td>1.19–1.32</td>
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