Cerebral blood flow velocity changes after rapid administration of surfactant

SIR,-We enjoyed reading the paper by Dr Cowan et al which reported a number of problems in relating the findings to the conclusion of the authors. The rationale behind the study appears to be the finding of an increased incidence of severe intraventricular haemorrhage in one study of surfactant replacement.1 The authors do mention two other randomised trials which reported a reduction of intraventricular haemorrhage, to which one might add a further two (this review lists four studies, references 37-40, which demonstrate significant reduction in intraventricular haemorrhage after surfactant treatment). Large intraventricular haemorrhage (IVH) and IV have also been shown to be reduced in four studies (references 37, 39, 50, 51).

A meta-analysis involving 34 randomised controlled trials shows odds ratios (95% confidence intervals) for the effect of surfactant on intraventricular haemorrhage of 0.89 (0.73-1.07) for prophylaxis and 0.79 (0.64-0.97) for rescue studies.2 The body of scientific evidence points to a modest reduction of intraventricular haemorrhage after surfactant treatment.

The authors go on to say that the collaborative European multicentre study of Curosurf found a surprisingly high incidence (26%) of severe intraventricular haemorrhage, but they omit to say just how all these infants were, as they required mechanical ventilation in >60% oxygen within the first 15 hours of life.5 Indeed the control infants had a mortality rate in excess of 50%. The incidence of severe intraventricular haemorrhage in this study is less than that reported in at least three other studies (references 54, 58, 67) by rescue surfactant.

Many of the babies studied by Cowan et al would not have fulfilled the criteria for the collaborative European multicentre study of Curosurf6 as they were treated up to the age of 72 hours. The authors do not say whether any of their infants suffered from severe asphyxia, although we are told that the infants had pancuronium and pethidine. It seems likely that these babies were extremely ill and in the discussion it is said that seven out of eight had poor or absent cerebral autoregulation.1 The authors correctly point out that there was a modest reduction in mean arterial pressure after giving surfactant and on looking at their results it would seem that in some infants mean arterial pressure and cerebral blood flow velocities were actually increased. Mean arterial pressure in the graphs all appear to be above 30 mm Hg which is a level at which the authors had previously noted an increase of cerebral blood flow velocies may all be changing. CBF measured using 133 xenon clearance (G Greisen and A Bell, personal communication) and near infrared spectroscopy (FOR Reynolds and AD Edwards, personal communication) shows no consistent change after Curosurf administration and is related to change in carbon dioxide tension.

The final paragraph of the discussion again suggests that there is a relationship between surfactant treatment and increase in intraventricular haemorrhage where in fact none exists. The reduction in intraventricular haemorrhage is in keeping with our two year follow up studies with Curosurf which show that 80% of treated survivors have normal development compared with 72% of control infants.2 We have performed only one study which has looked at a slow continuous infusion of Curosurf compared with the currently recommended bolus instillation. In this pilot study improvement in blood gases was less and the duration of effect greatly shortened. (PW Naras and C Rudin, personal communication). Perhaps there is a need for larger randomised trials of different methods of surfactant instillation because the haemodynamic changes reported by the authors were due to compromise of their infants before treatment or to their concomitant drug treatment.

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Gastroduodenal perforation in preterm babies treated with dexmethasone for bronchopulmonary dysplasia

SIR,—We read with interest the recent paper by Dr Ng and colleagues1 and would like to add a comment on prostaglandins. The authors did not refer to gastric prostaglandins in patients and preterm infants in maternal breast milk, but these substances may play an important part in the aetiology and treatment of gastroduodenal mucosal lesions and perforation in preterm babies treated with dexmethasone. Prostaglandins, and PGE2 and PGI2 in particular, are known to regulate protective factors in the gastroduodenal mucosa such as mucosal blood flow, mucus production, and bicarbonate secretion. An increase in prostaglandins in the gastroduodenal mucosa will therefore result in the depression of the protective mechanisms, thereby contributing to the development of gastroduodenal mucosal lesions. On the other hand, glucocorticosteroids are known to inhibit prostaglandin biosynthesis through the suppression of phospholipase A2 activity. Nobuhara et al2 have shown that corticosteroids decrease gastric mucosal PGF2, in the gastric mucosa and have demonstrated a good correlation between this decrease and the development of gastric mucosal lesions.2 We have also demonstrated

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