an impaired increase in PGE\textsubscript{2} in gastric juices which is thought to reflect the amount of PGE\textsubscript{2} in the gastric mucosa during steroid treatment in children.\textsuperscript{3} Moreover, Marino \textit{et al} have reported that the PGE\textsubscript{2} concentration in the gastric secretion in premature infants was significantly lower than that in full term infants.\textsuperscript{4} On the basis of these findings, we suggest that a decrease or impaired increase in PGE\textsubscript{2} in the gastroduodenal mucosa is one of the important factors in the development of gastroduodenal mucosal lesions and perforation in premature babies treated with dexamethasone.

This suggestion may have a therapeutic implication when it comes to breast feeding, although Ng \textit{et al} described it in only one of four cases, \textsuperscript{5} the administration of oral PGE\textsubscript{2} analogues has been shown to protect the gastric mucosa from steroid induced damage.\textsuperscript{6} In addition, a considerable quantity of prosta- glandins including PGE\textsubscript{2} has been identified in human milk but not in infant formula.\textsuperscript{7}

Consequently, breast feeding may have an important protective effect on gastroduodenal mucosal lesions in preterm infants during steroid treatment.

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The prophylactic use of ranitidine in babies treated with dexamethasone

\textbf{Str}.—The Collaborative Dexamethasone Trial Group recently reported an increase in gastro-intestinal bleeding among dexamethasone treated preterm infants compared with controls, although this did not reach statistical significance.\textsuperscript{1} This trial did not report any babies with gastrointestinal perforation but two recent papers in this journal estimated an incidence of perforation of the order of 2–3%, and it was associated with considerable morbidity and mortality.\textsuperscript{2}

The mechanism by which steroids cause gastrointestinal ulceration is unclear, although it is generally accepted that they reduce the effectiveness of the protective mucosal barrier. In this situation normal acid production may be sufficient to cause gastrointestinal perforation. Since we noted this complication we have aquired intraduodenal pH in babies treated with dexamethasone and various doses of ranitidine.

We report the effect of using a ranitidine infusion of 0.0625 mg/kg/hour in seven babies treated with dexamethasone and not receiving enteral feeding. Patient characteristics and results are presented in the table. This dose of ranitidine caused a significant increase in gastric pH (p<0.001).

In order to test whether or not the routine use of an H\textsubscript{2} antagonist would significantly reduce the incidence of gastrointestinal bleeding or perforation due to dexamethasone a controlled trial involving more than 2000 babies would have to be performed. Although this would be desirable, it would allow adverse as well as beneficial effects to be looked for, it is unlikely to be done. In the meantime, because of its demonstrated effectiveness in reducing gastric acid secre- tion, we currently administer ranitidine prophylactically to all babies treated with dexamethasone.

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Non-invasive assessment of pulmonary arterial pressure in healthy neonates

\textbf{Str}.—Recent correspondence in this journal referred to the assessment of pulmonary artery pressure by Doppler using the ratio of time to peak velocity (TPV); right ventricular ejection time (RVET).\textsuperscript{1} We should like to point out that the position of the regression line relating TPV:RVET to pulmonary artery pressure is influenced by whether the pulsed Doppler sample is taken from the right ventricular outflow tract proximal to the pulmonary valve or from the main pulmonary artery. If TPV:RVET, measured from the main pulmonary artery, is plotted on a regression line derived from measurements made in the right ventricular outflow tract then an inappropriately high mean high pulmonary artery pressure will be obtained. This explains why Skinner \textit{et al}, commenting on our letter,\textsuperscript{2} found an impos- sibly high pulmonary artery pressure of 100 mm Hg when they plotted our measurement of TPV:RVET (sampled from main pul- monary artery) on a regression line based on Kitabatake's measurements (sampled from right ventricular outflow tract).\textsuperscript{2}

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Respiratory support using patient triggered ventilation in the neonatal period

\textbf{Str}.—I should like to comment on your recent timely article on patient triggered ventilation in the neonatal period.\textsuperscript{1} The authors indicate that a recent study patient triggered ventil- ation (PTV) was successful only in three out of 16 infants with chronic lung disease because of asynchrony or poorly sustained respiratory effort in these infants. It may be more appropriate to use a longer inspiratory time (0.6 seconds) when ventilating these babies in trigger mode as this has been shown to be associated with an increase in tidal volume due to the recruitment of more alveoli.\textsuperscript{2} Similarly, if such an infant is being weaned using PTV, doing so by decreasing peak inspiratory pressure may result in progressive alveolar collapse. We have recently had difficul- ties weaning an infant with chronic lung disease in this way. PTV using a ventilator with a built in refractory period resulting in inactivation of the trigger for some of the babies' own breaths enables peak inspiratory pressure and inspiratory time to be maintained and might be more appropriate for weaning infants with chronic lung disease.

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Is 28 weeks of gestation sufficient to 1000 g of birth weight?

\textbf{Str}.—With the rapid development of neonatal intensive care, many tiny and premature babies are now surviving. In 1979 the World Health Organisation published a number of recommendations on the methodology of reporting perinatal mortality statistics.\textsuperscript{1} It is recommended that the numerator and denominator of all rates are restricted to liveborn infants only, and that gestational age (or, where birthweight is unavailable, the corresponding gestational age (28 weeks) or body length (35 cm crown-heel)). These recommendations have been strongly endorsed by the International Federation of Gynecolo- gists and Obstetricians.\textsuperscript{2} In the 9th revision of the \textit{International Classification of Diseases Clinical Modification},\textsuperscript{3} extreme immaturity (code 765.0) is defined as 'Usually implies a birthweight of less than 1000 grams and/or a gestation of less than 28 completed weeks', and other preterm infants (code 765.1) as 'Usually implies a birthweight of 1000–2499
grams and/or a gestation of 28–37 completed weeks. However, these definitions and criteria may be flawed. The issue here is whether 28 weeks of gestation is equivalent to 1000 g of birth weight. Although the mean birth weight at 28 weeks varies from country to country, most of the published data show that infants of 28 weeks weigh more than 1000 g on average, suggesting that 28 weeks of gestation is not a good surrogate for 1000 g of birth weight (references available from author).

Although births with weight around 1000 g or 28 weeks of gestation account for a very small proportion of total births, perinatal mortality in this group is very high. Therefore, in statistical reporting of perinatal mortality, adoption of different criteria (1000 g or 28 weeks) may yield different results. In perinatal research, especially that focusing on preterm births or very low birthweight infants, inconsistency of criterion for sample selection may introduce misclassification into the study, which could bias results in either direction. Thus, only one criterion should be used in statistical reporting of perinatal mortality and perinatal research.

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