an impaired increase in PGE_2 in gastric juices which is thought to reflect the amount of PGE_2 in the gastric mucosa during steroid treatment in children.[3] Moreover, Marino *et al* have reported that the PGE_2 concentration in the gastric secretion in premature infants was significantly lower than that in full term infants.[4] On the basis of these findings, we suggest that a decrease or impaired increase in PGE_2 in the gastroduodenal mucosa is one of the important factors in the development of gastroduodenal mucosal lesions and perforation in preterm babies treated with dexamethasone.

This suggestion may have a therapeutic implication when it comes to breast feeding, although Ng *et al* described it in only one of four cases. The administration of oral PGE_2 analogues has been shown to protect the gastric mucosa from steroid induced damage.[2] In addition, a considerable quantity of prostaglandins including PGE_2 has been identified in human milk but not in infant formulas.[5] 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

SIR,—The Collaborative Dexamethasone Trial Group recently reported an increase in gastrointestinal bleeding among dexamethasone treated preterm infants compared with controls, although this did not reach statistical significance.[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.[2 3]

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 tried to reduce acid production, with the H_2 receptor antagonist ranitidine, in all babies treated with steroids. To determine the effectiveness of this practice we have been serially monitoring gastric pH in babies treated with dexamethasone and various doses of ranitidine.

We report the effects 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 ($p<0·0001$).

The characteristics and results of the patients studied

	Gestation (weeks)	*Weight (g)*	*pH before ranitidine*	*pH while on ranitidine*
Mean	27·7	849	1·7	4·9
Range	24–31	579–1171	1·3–2·0	4·2–6·0

In order to test whether or not the routine use of an H_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, as 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 secretion, 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

SIR,—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).[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 pulmonary artery pressure will be obtained. This explains why Skinner *et al*, commenting on our letter,[1] found an impossibly high pulmonary artery pressure of 100 mm Hg when they plotted our measurement of TPV:RVET (sampled from main pulmonary artery) on a regression line based on Kitabatake's measurements (sampled from right ventricular outflow tract).[2]

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Respiratory support using patient triggered ventilation in the neonatal period

SIR,—I should like to comment on your recent timely article on patient triggered ventilation in the neonatal period.[1] The authors indicate that in a recent study patient triggered ventilation (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 recruitment of more collapsed alveoli.[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 difficulty 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 equivalent to 1000 g of birth weight?

SIR,—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[1]: 'It is recommended that countries should present, solely for international comparisons, "standard perinatal statistics" in which both the numerator and denominator of all rates are restricted to fetuses and infants weighing 1000 g or more (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 Gynecologists and Obstetricians.[2] In the 9th revision of the *International Classification of Diseases Clinical Modification*,[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