an impaired increase in PGE₂ in gastric juices which is thought to reflect the amount of PGE₂ in the gastric mucosa during steroid treatment in children. Moreover, Marino et al have reported that the PGE₂ concentration in the gastric secretion in premature infants was significantly lower than that in full term infants. On the administration of oral PGE₂ analogues has been shown to protect the gastric mucosa from steroid induced damage. In addition, a considerable quantity of prostaglandins including PGE₂ has been identified in human milk but not in infant formula feeds. Consequently, breast feeding may have an important protective effect on gastroduodenal mucosal lesions in preterm infants during steroid treatment.

The prophylactic use of ranitidine in babies treated with dexamethasone

Str,—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. 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 which was associated with considerable morbidity and mortality.

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 measured gastric acid production, with the H₂ receptor antagonist ranitidine, in all babies treated with steroids. To determine the effectiveness of this practice we have serially monitored 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<0·001).

In order to test whether or not the routine use of an H₂ 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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The characteristics and results of the patients studied

<table>
<thead>
<tr>
<th>Gestation (weeks)</th>
<th>Weight (g)</th>
<th>pH before ranitidine</th>
<th>pH while on ranitidine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Range 24–31</td>
<td>579–1171</td>
<td>1–3–2</td>
<td>4–2–6</td>
</tr>
<tr>
<td>Mean 27–77</td>
<td>849</td>
<td>1–7–1</td>
<td>4–9</td>
</tr>
</tbody>
</table>


Respiratory support using patient triggered ventilation in the neonatal period

Str,—I should like to comment on your recent timely article on patient triggered ventilation in the neonatal period. The authors indicate that 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 alveoli. Similarly, if such an infant is being weaned using PTV, do 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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In 28 weeks of gestation equal to 1000 g of birth weight?

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. 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 born alive (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. In the 9th revision of the International Classification of Diseases Clinical Modification, 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.'