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. 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 formulas. 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

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 it 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 reviewed gastric acid production, with the H2 receptor antagonist ranitidine, in all babies treated with steroids. To determine the effectiveness of this procedure we have been serially measuring gastric pH in babies treated with dexamethasone and various doses of ranitidine.

We report the results of using a ranitidine infusion of 0.0625 mg/kg/hour in seven babies

The characteristics and results of the patients studied

<table>
<thead>
<tr>
<th>Gestation</th>
<th>Weight</th>
<th>pH before</th>
<th>pH while</th>
</tr>
</thead>
<tbody>
<tr>
<td>(weeks)</td>
<td>(g)</td>
<td>ranitidine</td>
<td>ranitidine</td>
</tr>
<tr>
<td>Mean</td>
<td>27-77</td>
<td>849</td>
<td>1-7</td>
</tr>
<tr>
<td>Range</td>
<td>24-31</td>
<td>579-1171</td>
<td>1-3-2-0</td>
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</table>

Non-invasive assessment of pulmonary arterial pressure in healthy neonates

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). 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, found an impossibly high pulmonary artery pressure of 100 mmHg 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).

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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 the prolonged inspiratory period.

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 baby's 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?

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 neonatal mortality rates (birthweight 0-2500 g) be calculated for both liveborn infants and stillbirths; and separately for liveborn infants weighing 2501-3000 g, 3001-3400 g, 3401-3700 g, 3701-4000 g, 4001-4400 g and 4401-4800 g.
The prophylactic use of ranitidine in babies treated with dexamethasone.

E J Kelly, K G Brownlee, P C Ng and P R Dear

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