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 proportion of prosta
glandins including PGE₂ has been identified in human milk but not in infant formula. Consequently, breast feeding may have an important protective effect on gastroduodenal mucosal lesions in preterm infants during steroid treatment.

TOSHIKAI SHIMIZU YUICHIRO YAMASHIRO KEIJIRO YABUTA
Department of Paediatrics, University of Tansui, 2-1-1 Hongu Bunkyo-ku, Tokyo, Japan

3 Shimizu T, Shioya T, Yamashiro Y, Sato M, Yabuta K. Impaired increase of prosta
4 Marino LR, Blumer JL, Halpin TC Jr. Prosta

The prophylactic use of ranitidine in babies treated with dexamethasone

SIR,—The Collaborative Dexamethasone Trial Group recently reported an increase in gastro
testinal 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 mor
diety 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 administered acid production, with the H₂ 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).

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 secret
cion, we currently administer ranitidine prophylactically to all babies treated with dexamethasone.

E J KELLY
P C NG
P R F DEAR
Academic Unit of Paediatrics and Child Health, St James’s University Hospital, Beckett St, Leeds LS9 7TF

3 O’Neill EA, Chwals Z. Gastroduodenal ulceration during ventilator treatment dependency: possible life threatening gastro

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). 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 arterial pressure will be obtained. This explains why Skinner et al, commenting on our letter,1 found an impos
ingly 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).2

NICK EVANS
NICK ARCHER
Department of Paediatrics, John Radcliffe Hospital, Headington, Oxford OX3 9DU

1 Evans N, Archer N. Non-invasive assessment of pulmonary arterial pressure in healthy neo

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 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 a slower rate of more complete ventilation.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 baby’s breaths enables peak inspiratory pressure and inspiratory time to be maintained and might be more appropriate for weaning infants with chronic lung disease.

N J SHAW
G A B RUSSELL
Department of Paediatrics, Liverpool Maternity Hospital, Oxford Street, Liverpool L7 8BN

1 Greenough A, Milner AD. Respiratory support using patient triggered ventilation in the neo

Is 28 weeks gestation equivalent to 1000 g 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 and institutions report results, solely for international comparisons, "standard perinatal statistics" in which both the numerator and denominator of all rates are restricted to five or more deaths (with five or more births). If 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 Gyneco
tologists 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
Respiratory support using patient triggered ventilation in the neonatal period.

N J Shaw and G A Russell

Arch Dis Child 1992 67: 471
doi: 10.1136/adc.67.4_Spec_No.471-b

Updated information and services can be found at:
http://adc.bmj.com/content/67/4_Spec_No/471.3.citation

Email alerting service

These include:
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/