LETTERS TO THE EDITOR

Aerosol drug delivery to intubated neonates

SIR,-We were pleased to read the two articles concerning aerosol drug delivery to intubated neonates by O'Callaghan et al¹ and Grigg et al.² In particular, methods to determine the dosage available from the various devices should prove useful for investigators developing new means for aerosol delivery to intubated patients. We have previously described a device similar to the Aerochamber and not unlike the 'collapsible spacer' of O'Callaghan et al. We characterised its performance using beclomethasone dipropionate.³ As noted in both of the new reports,^{1 2} most of the drug released from the metered dose inhaler (MDI) does not reach the lung. We found just over 50% of the drug was actually aerosolised into the spacer.

With our device, the output from the tip of the endotracheal tube was $2.95\pm0.75\%$ of the MDI dose, similar to that delivered by the collapsible spacer device of O'Callaghan et al. We also noted that this output would translate to a rather high dose/kg compared with adults. We would like to point out that if a spacer like the Aerochamber is shortened, however, then the dosage delivered from the endotracheal tube can be decreased. When the length of the spacer on our device was reduced from 16 cm to 7.5 cm, the output dropped from 1.17 ± 0.29 µg to 0.79 ± 0.19 µg. We are currently evaluating the effect of different spacer and endotracheal tube sizes on drug output. We too believe that drugs like beclomethasone, when delivered directly to the lungs, offer great promise. To that end we have begun a randomised, blinded trial of aerosolised beclomethasone versus systemic dexamethasone for the prevention of bronchopulmonary dysplasia.

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Respiratory support using patient triggered ventilation in the neonatal period

SIR,-Greenough and Milner in their review of patient triggered ventilation in neonates make a serious error in the their description of the use of abdominal expansion as a trigger.¹ They advocate taping the sensor capsule of the respiration detector on to the abdominal wall just below the xiphisternum, which happens to be the worst possible place for it. During inspiration, the costal margin may be pulled in by the contraction of the diaphragm to such an extent as to reduce or even reverse the movement of the abdominal wall, so that the pneumatic signal from the capsule is reduced or even abolished. The capsule should be placed, after careful observation of the respiratory movements, at the position where abdominal expansion is maximal, usually in the lower abdomen. The use of this defective technique may account for the absurdly high delays and poor sensitivity observed by Hird and Greenough.²

Although this is a serious point and emphasises the importance of close attention to detail, it will soon become only of academic interest. With modern techniques and attention to phase angle,³ airways pressure triggering is now easily possible with any intermittent positive pressure ventilator, and will supersede all other techniques. Although a prospective clinical trial would seem desirable, it would be very difficult to carry out, and there is a good case for taking patient triggered ventilation as the null hypothesis and requiring those who advocate other methods to prove their superiority.

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Professor Milner and Dr Greenough comment: We are grateful for Dr Wright's comments in response to our paper. We agree that there are considerable problems associated with the placing of the capsule, and that even when attached to the lower abdominal wall it is possible to achieve a situation in which the ventilator is then triggered by the baby's expiratory rather than inspiratory efforts. We are less optimistic that improvements in airway trigger design will overcome all problems as our experience has shown that in the first 24 hours of life, and in those of less than 28 weeks' gestation, respiratory efforts are often too erratic and insufficiently sustained for this technique.

Although we consider that the place of triggered ventilation is probably secure, we remain convinced that all new techniques introduced into neonatal intensive care must be subject to carefully designed prospective control trials to assess their effects on longterm outcome.

Catabolic effect of dexamethasone in the preterm baby

SIR,-In the recent article by Brownlee et al their assessment of the catabolic effect of dexamethasone in the preterm infant promoted some active discussion.¹ One of the points of debate is whether the increase in blood urea observed was due solely to the effect of occult gastrointestinal haemorrhage that is seen and recognised in steroid treatment.²⁻⁴ The already

falling creatinine concentrations would then be in keeping with these findings.

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Premature labour

SIR,-We read with interest the recent review by Steer concerning premature labour.¹ With regard to the diagnosis of premature labour, he recommends a cervical assessment in all cases and states that in patients with preterm premature rupture of the membranes (PPROM) there is no evidence that up to three digital vaginal examinations have any effect on provoking labour. However, we have found a significant negative correlation between the number of digital examinations performed and the duration of the latent period in patients with PPROM (unpublished data). This observation is supported by several previous studies.²⁻⁴ It has been suggested that digital manipulation when assessing cervical conditions may evoke the release of prostaglandins or cause a subclinical infection that might in turn initiate the release of prostaglandins.³ When a patient with PPROM is not in labour and there are no other indications for immediate delivery, virtually all information necessary to manage the patient can be obtained from a single sterile speculum evaluation. The common practice of performing a digital examination in patients with PPROM who are not in labour seems to shorten the interval between examination and delivery $^{2-4}$ and may expose the patient to increased risk of infection.4

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