uninterpretable without knowledge of the comparability in terms of birthweight, gestation, age at death, and number of infants in the fatal HMD + IVH and HMD-only groups. Even with such information an expression of the spread and frequency of the measurements involved is needed for any scientific evaluation, particularly when dealing with data items such as *p*H and Pao_2. We assume that, even in Oxford, blood gases are not measured continuously from the moment of birth in every preterm infant! From the data given in their original paper it may be presumed that the numbers are too small for any detailed analysis.

Their Table 2 is totally misleading. It compares our Hammersmith figures for liveborn singletons over the 8 years 1966–73 with the Oxford figures for all livebirths over a 25-month period from June 1972-July 1974, restricts consideration to babies weighing 1001–1500 g, and ignores the relatively large number of clinically diagnosed IVH cases that were not subjected to necropsy at Oxford during the period in question as well as the 2 infants with IVH who survived beyond the neonatal period (Roberton and Howat 1975, Table I).

In another passage of their letter, Roberton and Howat refer to the infants with IVH without HMD. We have recently completed a paper on this group of infants (Wigglesworth *et al.*, 1977) showing that many of them were indeed given alkaline buffer therapy in similar dosage to that administered to infants with HMD of similar gestational age.

Infants born at Hammersmith in 1972–74 who developed HMD did of course live longer than the average for the period 1966–73. The mean age at death was 34 hours and 30% lived for 48 hours or more.

We reiterate that in our large group of singleton infants of 30–37 weeks' gestation who died with HMD, there was no criterion recorded in the case notes by which we could determine that those who developed IVH were sicker than those who died with HMD only, and that episodes of collapse diagnosed clinically as due to IVH were seen commonly in each group. One of the most convincing arguments for a role of alkaline buffer therapy in causation of IVH in babies with HMD is perhaps the association we have shown with unruptured germinal layer haemorrhage, where it seems difficult to imagine that the drug can have been given as a consequence of the infant's collapse.

We do however agree with Roberton and Howat that infants who die tend to be sicker than those who survive! Our only reason for including information on the alkali dosage administered to survivors was to show that the use of very large alkali doses ($\geq$ 10 mmol/kg in 12 hours) had never (at Hammersmith) been associated with the survival of an infant who required mechanical ventilation for RDS. It seems difficult to 'justify' the use of any drug in a dosage which is invariably associated with death, unless perhaps for euthanasia.

Careful reading of our paper will show that we do not believe that hypernatraemia is the most likely mechanism by which bicarbonate may elicit IVH and in that respect we also agree with Roberton and Howat. Nor would we advocate that sodium bicarbonate solutions should be entirely banished from the newborn nursery.

We do consider that we have produced sufficient evidence to warrant caution in the use of a potentially lethal drug and believe that Roberton and Howat do no justice to themselves by their carping criticisms based on misleading and anecdotal data. If they seriously wish to compare their data with ours we will be happy to co-operate in carrying out a correctly designed and statistically valid study.

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Improved method of attaching Po_2-electrode to fetal scalp

Sir,

Adequate fixation to the fetal scalp of surface electrodes for continuous measurement of fetal Po_2 during labour has been problematic. We constructed a ring (Fig.) from soft silicone rubber (silicone compound Rhodorsil

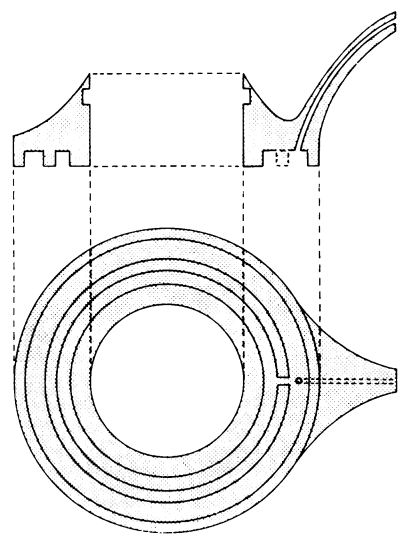

Fig. *Silicone rubber ring.*

Caf 3, Rhône-Poulene, Paris), of height 6 mm, internal diameter 10 mm, and external diameter 20 mm. The suction area is 0·98 cm^2, area of contact 1·37 cm^2, and weight 0·8 g. The Po_2-electrode measuring 5 mm in height and 10 mm in diameter is inserted in the ring. Fixation of the ring to the fetal scalp is completed by applying a negative pressure of 50–100 mmHg.

The ring was tested on 5 fetuses in vertex position during labour. Its application was possible from 2 cm dilatation. Testing time varied from 2 to 8 hours during the first stage, and from 15 to 45 minutes during the second stage of labour. In all cases the ring stayed in place. Owing to its very small weight and size, the ring causes only minimal skin traction. After removal, slight transient hyperaemia of the skin was observed.

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Upper airway resistance

Sir,

We are interested in the paper by Purcell (*Archives*, 1976, **51,** 602) on the response of the newborn to raised upper airway resistance, and while accepting the general validity of his conclusion concerning behaviour responses, we are somewhat perturbed by his numerical data. An almost identical experiment, conduced to measure nasal resistance in infancy, was reported by Lacourt and Polgar (1971) who pointed out that the equation governing total pulmonary resistance (R_{total}) is:

$$R_{total} = R_{lower\ airway} + \left(R_{small\ nostril} \times R_{large\ nostril}\right) \Big/ \left(R_{small\ nostril} + R_{large\ nostril}\right).$$

Applying this equation to Purcell's data from his text, R (lower airway) is a *negative* value of −20 cm H_2O/l per second and applying it to the data in his Table, assuming equal nasal resistances, we again calculate a negative value of −54 and −71 cm H_2O/l per second for the two sleep states. Lacourt and Polgar calculated a much more likely value of +17·9 cm H_2O/l per second and much lower nasal resistances. Clearly a negative lower airway resistance is impossible and we can only speculate that there was some technical error in Purcell's otherwise interesting study. Our own experience, and that of others, is that measurement of total pulmonary resistance using the oesophageal balloon technique is very unreliable, especially in the supine infant as studied by Purcell.

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Reference

Lacourt, G., and Polgar, G. (1971). Interaction between nasal and pulmonary resistance in newborn infants. *Journal of Applied Physiology*, **30,** 870–873.

Dr. Purcell comments:

The method of raising airways resistance used, placing a finger on alternate nostrils, makes movement of the soft nasal septum of the newborn almost inevitable with some obstruction of the opposite nostril as well. This would exaggerate the contribution of the nostrils to the total airways resistance and the formula used by Lacourt and Polgar is not applicable. The technique was chosen as a simple way of raising the airways resistance to assess the respiratory response. Lacourt and Polgar in their study of the nasal airway were at pains to avoid any deflection of the septum, occluding the nostrils with a plug of cotton wool soaked in silicone lubricant. (Their infants were also supine and intrathoracic pressure was measured with an oesophageal balloon.)

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Plasma aldosterone levels in bottle-fed infants

Sir,

In a recent paper, plasma aldosterone levels on day 6 of life were found to be higher in bottle-fed infants than in those breast fed, though there was no difference in the mean values in cord blood (Dillon *et al.*, 1976). The authors were unable to account for their finding, but there are several differences between breast milk and Cow & Gate Babymilk Plus, the formula given to the bottle-fed infants, which could be responsible (Table).

While the sodium content of Babymilk Plus is similar to that of mature breast milk (Macie, 1949), it is considerably lower than the values obtained for colostrum and transitional milk in this laboratory. Colostrum (days 1–3) was found to have a mean sodium concentration of 23·75 mEq/l (23·75 mmol/l) and a mean potassium concentration of 18·5 mEq/l (18·5 mmol/l), which fell by the sixth day post partum to 17 mEq/l and 16·78 mEq/l respectively (Ansell *et al.*, 1976). By day 6 of life a breast-fed infant will have had a greater total sodium intake than an infant fed on Babymilk Plus from birth, and it may be argued that the bottle-fed infants had a relative deficiency of sodium and thus a greater stimulus to the aldosterone sodium-conserving mechanism than the breast-fed infants.

The difference in *p*H between Cow & Gate Babymilk Plus and breast milk is considerable (Table). It has been shown that breast-fed infants excrete fewer hydrogen ions