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:

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

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

Table *Difference in composition of Babymilk Plus and day 6 breast milk related to plasma aldosterone levels*

	*Cow & Gate Babymilk plus**	*Breast milk (day 6)*†
Na^+ (mEq/l)	13	17±5·02
K^+ (mEq/l)	22	16·8±2·54
*p*H	6·53	7·10±0·16
Plasma aldosterone‡	2040 pmol/l (73·44 mg/100 ml)	1012 pmol/l (36·43 mg/100 ml)

*Barrie *et al.* (1975).
†Ansell *et al.* (1976).
‡Dillon *et al.* (1976).

than infants fed cows' milk (Hatemi and McCance, 1961) because of the greater net acid content of cows' milk. Sulyok (1971), investigating the relationship between electrolyte and acid-base balance in term infants found a close association between the renal loss of sodium in the first 2 weeks of life and the development of metabolic acidosis, and suggested that this might be due to transient adrenal mineralocorticoid deficiency; and Pitts (1950) showed that adrenalectomized rats failed to show an increase of ammonium excretion in response to an acid load and that this could be corrected by adrenal cortical extract.

The infant's ability to cope with an acid load seems to be dependent at least in part on adequate renal sodium-hydrogen ion exchange (Sulyok *et al.*, 1972). As aldosterone plays an important part in this, it is not surprising that it is present in higher concentrations in those infants receiving a greater acid intake. Infants fed on Cow & Gate Babymilk Plus with its lower sodium and higher acid content might be expected to have higher plasma aldosterone levels than breast-fed infants as found by Dillon *et al.* (1976) in order to conserve sodium and excrete hydrogen ions.

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Drs. Dillon and de Swiet comment:

We are grateful to Dr. Moore for emphasizing the electrolyte differences between colostrum, transitional milk, and Cow & Gate Baby Milk Plus. We would agree that on the basis of the higher concentration of sodium in early breast milk compared with that in Cow & Gate Babymilk Plus, breast-fed infants may receive more sodium in the first week of life than those artificially fed with this particular milk formula. Though this might have contributed to the lower values of plasma aldosterone (PAldo) in the breast-fed group of infants in our study (Dillon *et al.*, 1976), we were surprised that there were no parallel changes in the values of plasma renin activity (PRA). In our experience PRA is a more sensitive index of sodium status than PAldo, and we would have expected similarly low values in the breast-fed babies if they had been significantly sodium-loaded compared with the artificially-fed group. Furthermore, we failed to show differences in urine sodium excretion on day 6 of life between those babies fed breast milk and those fed Babymilk Plus, suggesting that on day 6 the sodium intake of both groups was very similar.

Aldosterone may well play a part in the newborn infant's acid-base homoeostasis. We are, however, unaware of any clear evidence that this is an important role or that aldosterone secretion is specifically increased in response to an acid load. None the less we accept that in situations where aldosterone secretion is clearly deficient the administration of mineralocorticoids can increase the bicarbonate threshold by improving sodium reabsorption (Oetliker and Zurbrügg, 1970).

It is intriguing to speculate on the reasons for increased activity of the renin aldosterone system in the infant and young child. We would agree with Dr. Moore that this is likely to be related to sodium conservation, as we stated in our recent *Archives* paper. However, it seems that the levels of PRA and PAldo in infancy and childhood are almost entirely dependent on age and only marginally attributable to differences in sodium intake (Dillon and Ryness, 1975). We would therefore not wish to put too great an emphasis on the findings of higher PAldo levels in the bottle-fed babies in our neonatal study. The numbers were comparatively small and the differences only just statistically significant. Further study seems required to clarify the situation.

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