meable to these short chain fatty anions, they may act as osmotic purgatives, and regulate the total water content of the stool, i.e. control stool volume.

The relation between 24-hour stool volume and organic anion output has been investigated in over 40 separate 24-hour stool collections on healthy babies in the Newborn Nursery in Aberdeen. A positive correlation exists between stool volume and 24-hour organic anion output in these infants, lending support to the hypothesis that stool organic anions are of physiological importance in controlling stool volume.

Endocrine control of fetal lung development in the rabbit. M. L. Chiswick, P. M. B. Jack, A. Ahmed, and R. D. G. Milner. Departments of Health and Pathology, University of Manchester, Manchester.

In the last 6 days of fetal life in the rabbit important changes occur in the morphological, biochemical, and physical properties of the lung which result in the facilitation of pulmonary expansion and the stabilization of the air spaces. Glucocorticoids given to the fetus towards the end of gestation accelerate the development of normal pulmonary surface properties. We have studied the implication that naturally occurring fetal glucocorticoids are necessary for normal lung development in the rabbit fetus.

The fetal pituitary is necessary for normal development of the adrenal cortex. In a series of experiments, 1 fetus of a litter was decapitated *in utero* on day 24 of gestation and allowed to develop for a further 5 days. The litter was delivered by hysterotomy on day 29 and the lungs of the decapitated fetus were compared with the lungs of the control littermates in terms of histology and physical properties.

The osmiophilic inclusion bodies in the pneumocytes, which are thought to represent stored surfactant, were reduced in the decapitated fetuses to approximately half the concentration seen in control littermates. The results of pressure-volume studies, and the examination of the stability of bubbles squeezed from the lungs, showed that the surface-active properties of the alveolar lining were normal in decapitated and control fetuses.

The reduction in inclusion bodies caused by decapitation is probably due to fetal adrenal atrophy. It may

be concluded that a full complement of inclusion bodies is not necessary for the development of normal physical properties of 29-day fetal rabbit lung.

Pulmonary function in the infant of the diabetic mother. Robert Dinwiddie and George Russell. Department of Child Health, University of Aberdeen.

Pulmonary hypoperfusion has been implicated in the pathogenesis of hyaline membrane disease (Chu *et al.*, 1967), and is one of the changes in respiratory function which occur in this disease, in addition to reduced lung volume and compliance.

The infant of the diabetic mother has an increased risk of developing hyaline membrane disease, and because pulmonary hypoperfusion might precede or might occur in the absence of other functional or clinical manifestations of 'stiff lungs', it was of interest to study the results of pulmonary function tests including measurements of effective pulmonary blood flow in such infants. Pulmonary function in a small group of infants born to diabetic mothers has been investigated and compared to a control group of normal infants.

Results were as follows. Mean values for tidal volume, respiratory rate, lung volume, specific compliance, and work of breathing were closely similar for the 2 groups.

Mean effective pulmonary blood flow was significantly lower ($P < 0.05$) in the infant of the diabetic mother ($131 \text{ ml/kg per min SD} \pm 22$) than in the control group ($164 \text{ ml/kg per min SD} \pm 31$).

In one diabetic's infant with hyaline membrane disease evidence was found of reduced specific compliance, lung volume, and effective pulmonary blood flow and increased work of breathing, with a return to normal values on clinical recovery.

It is concluded that in the infants studied effective pulmonary perfusion was significantly reduced, even in the absence of respiratory symptoms and that this may be a factor contributing to the increased incidence of hyaline membrane disease in such infants.

REFERENCE

- Chu, J. *et al.* (1967). Neonatal pulmonary ischaemia. *Pediatrics*, **40**, 709.