Histidinaemia is an autosomal recessive condition in which there is a deficiency of histidase and persistently raised blood levels of histidine. Family studies show a wide variation in the clinical picture from complete normality to severe retardation. The association between the biochemical condition and any neurological abnormality could be coincidental. In the present study, infants with raised histidine levels by Guthrie technique were followed without dietary treatment. In the first year 110,000 infants were screened and 10 had persistently raised histidine levels (i.e. incidence 1 in 11,000). The oldest is now 15 months and is within the normal range in all development.

Urinary FDP in children with renal disease.

William Uttley introduced by J. K. Brown. Department of Child Life and Health, University of Edinburgh.

Vassalli and McCluskey (1971) reviewed the current status of coagulation processes and fibrin deposition in the pathogenesis of renal disease.

We have examined a series of 96 patients, one-third of whom were children with a wide spectrum of renal disease, comparing the demonstration of fibrin in renal biopsy specimens using 3 techniques—standard histochemistry, electron microscopy, and immunofluorescence. A good correlation existed between the most reliable of these methods (immunofluorescence) and the estimation of the maximum amount of fibrin/fibrinogen degradation products (FDP) in the urine (Clarkson *et al.*, 1971) before biopsy.

In children, normal values of UFDP have been obtained (0.0–0.5 $\mu\text{g/ml}$). Results obtained are in the Table.

Significant amounts of UFDP were also found in patients with urinary tract infection and the haemolytic uraemic syndrome. Illustrative cases were discussed. It was concluded that the urinary excretion of FDP does not support the hypothesis of significant fibrin deposition in nephrosis (Duffy *et al.*, 1970), but reflects periods of episodic coagulation in glomeruli in proliferative nephropathy.

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In vivo effect of adenosine 3', 5'-monophosphate on Ehrlich ascites tumour cells. Mary J. Seller and Philip F. Benson. Paediatric Research Unit, Guy's Hospital Medical School, London SE1 9RT.

In some tumour cells there is diminished activity of adenylyl cyclase. There is evidence which suggests that this causes deficient production of cyclic AMP which in turn results in uninhibited cell division. When tumour cells are cultured *in vitro* the addition of cyclic AMP to the medium restores some of the properties of density dependent inhibition of growth.

We have investigated the carcinostatic effect of cyclic AMP. 10 million Ehrlich ascites tumour cells were injected either subcutaneously to produce a solid tumour, or intraperitoneally to produce the ascitic form. 3 days later drugs were injected intraperitoneally twice daily for 4.5 days. Two regimens were used. (1) Cyclic AMP 10 mg/kg and theophylline ethylenediamine (TED) 50 mg/kg (cyclic AMP + TED). (2) TED 50 mg/kg. The control group was injected with saline only.

Eight days after receiving tumour cells the cyclic AMP+TED animals had significantly smaller solid tumours than the controls ($0.001 > P > 0.01$). Those with the ascitic form had significantly fewer ascites cells (548 million) than the controls (1076 million) ($P = 0.001$), and significantly lower mean packed tumour cell volume ($0.001 > P > 0.01$). The TED group had intermediate values.

Ascites and abdominal wall infiltration were much less marked in the cyclic AMP+TED animals than in controls. Treatment also produced changes in tumour cell morphology.

It may be concluded that cyclic AMP inhibits growth and tissue invasion of Ehrlich ascites tumour cells in mice.

Organic anions and stool volume in the newborn.

Michael Tarlow and Hazel Thom introduced by G. Russell. Department of Child Health, University of Aberdeen.

The factors controlling stool volume in normal individuals are unknown. A large proportion of stool electrolyte consists of short chain fatty anions (e.g. acetate, lactate, butyrate). These presumably arise in the colon from bacterial fermentation of undigested foodstuffs. Since the colonic wall is relatively imper-

TABLE

Disease	No. of patients	No. of specimens	UFDP ($\mu\text{g/ml}$)			
			0–0.5	0.5–1	1–2	> 2
Nephrosis	14	143	123	14	4	2
Henoch-Schönlein purpura	16	77	70	5	1	1
Other forms of proliferative glomerulonephritis	19	129	83	23	12	11

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.

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