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