born to diabetic mothers with vascular complications, but no diabetic mothers in this study had proliferative retinopathy or nephropathy.

D. B. BARR introduced by PROFESSOR J. O. FORFAR (Edinburgh). ‘An Oral Calcium Test in Infancy.’ An oral calcium test feed has been designed to investigate the handling of dietary calcium as reflected by short-term changes in the serum calcium level.

The response of a normal control group is defined and compared with results in infants suffering from a variety of calcium disorders.

In the acute phase of idiopathic hypercalcaemia, an extremely high and prolonged hypercalcaemia is found after the test feed. The effects of treatment and recovery are followed.

In further infants who were not strikingly hypercalcaemic, abnormal loading tests suggested that the test was a more sensitive diagnostic index than sporadic estimates of serum calcium. The test may be a guide to the need for continuing therapy, and a persistently abnormal loading test has been associated with a poor long-term prognosis.

In patients with nutritional rickets, vitamin D caused a ‘shift to the left’ in the shape of the loading curve. A similar effect was seen in a group with neonatal tetany when those undergoing spontaneous recovery were compared with those given vitamin D. The curves obtained in infants with idiopathic hypercalcaemia were compared with those in infants receiving vitamin D.

J. B. HOUSTON introduced by PROFESSOR J. A. DAVIS (Manchester). ‘Renal Tubular Acidosis and Growth Retardation.’ To be published in full elsewhere.

J. T. HARRIES introduced by DR. JUNE K. LLOYD (London). ‘Studies of Vitamin E Function in Children with Malabsorption.’ Though symptoms of Vitamin E deficiency are easily recognized in animals, the clinical importance of this vitamin in man is less well established. Vitamin E is a powerful antioxidant and probably plays a part in the functional integrity of cell membranes. This paper reported investigations on the vitamin E status of children with various types of malabsorption. The function of the red cell membrane has been studied by estimating autohaemolysis and peroxide haemolysis of the cells, and then correlating these effects with the serum levels of the vitamin. Many of the children with low serum levels of vitamin E (<0.5 mg./100 ml.) had increased red cell haemolysis. Whereas autohaemolysis was increased from 6 to 62%, peroxide haemolysis was even more increased, varying between 20 and 90%. In most patients administration of the vitamin was followed by a prompt fall in haemolysis. The serum vitamin levels, however, took much longer to rise, and in some children low levels persisted for long periods after haemolysis had been restored to normal. Tests of red cell haemolysis appear to be much more sensitive indicators of vitamin E action at the cellular level than are estimations of the serum vitamin concentration, and it is probable that peroxide haemolysis will prove to be even more sensitive than autohaemolysis.


C. PICTON-WARLOW introduced by DR. J. W. SCOPES (London). ‘Peripheral Circulatory Responses to Postural Change in Healthy and Sick Newborn Infants.’ Venous occlusion plethysmography has been used to study forearm blood flow in healthy mature infants, healthy premature infants, and premature infants with respiratory distress syndrome.

In both mature and healthy premature infants, of birthweight varying upwards from 760 g., head-up tilting produces a rise in heart rate, a slight fall or no change in systolic blood pressure, and a reduction of forearm blood flow of 25-30% of the supine value.

Premature infants with severe respiratory distress syndrome have low forearm blood flow and low systolic blood pressure. Head-up tilting is followed by an increase in forearm blood flow. The physiological and clinical significance of this ‘paradoxical’ response to posture was discussed.

B. BERNADETTE KLIENSKY introduced by DR. W. W. PAYNE (London). ‘Role of Hyperkalaemia in Experimental Fetal Asphyxia.’ The levels of cardiac carbohydrate reserves are currently believed to be of prime importance in the ability of the fetus and newborn animal to resist anoxia. Histochemical observations on the hearts of anoxic stillbirths have shown persistence of considerable quantities of carbohydrate, indicating that death may occur before cardiac glycogen is depleted. This observation has prompted investigation of the possible role of hyperkalaemia as a cause of cardiac arrest in fetal anoxia. Changes in total cardiac carbohydrate and cardiac glycogen levels, pH, blood gases, and potassium levels have been studied in fetal rabbits after clamping the uterine vessels. Conscious increases of serum potassium are significantly higher than those reported in other experimental models.

The significance of the results was discussed in relation to the problem of human fetal anoxia.

N. A. J. CARSON introduced by PROFESSOR I. J. CARRÉ (Belfast). ‘Biochemical Response to Oral Pyridoxine in Homocystinuria.’ Homocystinuria is an inherited disorder in the metabolism of the essential sulphur-containing amino acid methionine. In the normal individual, methionine is demethylated to form homocysteine which is then condensed with serine by the aid of the enzyme cystathionine synthetase to form cystathionine. This thioether is then cleaved by cystathionase to form cysteine. The presence of pyridoxine is required as a coenzyme in the latter two reactions.