when the diet is finally discontinued, close follow-up being essential over a long period.

V. DUBOWITZ (Sheffield). 'Nerve Conduction Velocity—An Index of Neurological Maturity of the Newborn.' The conduction velocity of the ulnar and posterior tibial nerves has been measured in premature ('short gestation'), dysmature ('small for dates'), and full-term infants. 5 sets of twins were also included. The procedure is a relatively simple one and well tolerated even by newborn small premature infants. It is not influenced by factors such as state of sleep, or time after feed.

Sequential measurements have also been made in premature infants, and the conduction velocity attained at 40 weeks' post-conceptual age compared with the conduction velocity of full-term newborn infants.

There is a highly significant correlation between the motor nerve conduction velocity and gestation. The velocity increases with gestational age. In the twin studies there was no correlation of conduction velocity with weight at constant gestation.

'Small for dates' babies can be readily distinguished from premature infants of similar weight. Sequential studies on the same infants suggest that the rate of increase of conduction velocity after birth may be faster than *in utero*, but the differences are not statistically significant.

Studies in premature infants show that the conduction velocity of the premature infant at 40 weeks' post-conceptual age is significantly lower than that of the full-term newborn infant, suggesting a slower rate of maturation in premature infants.

Nerve conduction velocity is a useful parameter for assessing neurological maturity of the newborn infant for distinguishing premature from dysmature infants. Further data may also provide an accurate estimate of gestational age.

R. H. R. WHITE (Birmingham). 'Hypocomplementaemia and Progressive Glomerulonephritis.' A study of renal biopsy specimens obtained from patients showing clinical features of both the *nephrotic syndrome* and *nephritis* (i.e. haematuria, renal insufficiency and often hypertension) has revealed a specific morphological appearance in the majority. This consists of a combination of mesangial cell proliferation and marked, diffuse capillary wall thickening, due, mainly, to deposits of hyaline and fibrillar material on the *sub-endothelial* aspects of the basement membrane. These features distinguish 'membranoproliferative' glomerulonephritis (GN) from other forms of proliferative GN without capillary wall thickening, and from 'epimembranous' nephropathy, in which the deposits are on the *subepithelial* aspect of the basement membrane and proliferation is absent.

Nineteen children under 16 years of age and 4 adults showed this biopsy appearance; 10 were girls aged 8-15 years. Proteinuria occurred in all patients and was relatively unselective. Serum β_{1C} -globulin levels (estimated immunochemically by Dr. J. S. Cameron,

Guy's Hospital Medical School, London) were persistently lowered in 14 patients, in contrast to the normal levels found almost invariably in other patients with the nephrotic syndrome, and the transient depression observed in acute nephritis.

The illness runs a chronic course and does not respond to corticosteroid therapy. There is some evidence that cytotoxic drugs are beneficial if given early, however, and it is therefore urged that membranoproliferative GN should be recognized as soon as possible after onset, by the clinical, laboratory, and histological features described.

GRAHAM W. CHANCE introduced by PROFESSOR D. V. HUBBLE (Birmingham). 'Plasma Insulin Response to Oral Glucose in the Parents and Sibs of Children with Diabetes Mellitus.' The ratio of the increase in circulating insulin to increase in blood glucose at times of sampling after a glucose load has been empirically termed the 'insulinogenic index'. A low and delayed insulin response has been claimed to indicate a predisposition to diabetes mellitus.

Insulinogenic indices have been calculated for responses to oral glucose loads in the first-degree relatives of children with diabetes mellitus. One-third of the mothers and one-quarter of the fathers and sibs had a low index. In those with a low index the mean values for glucose and insulin suggest that the mothers may possess an antagonist to insulin and the fathers a limited insulin response to glucose. Early results in sibs suggest that a low insulinogenic index may indeed be of predictive value in the detection of early cases of diabetes.

CHRISTINE WATSON introduced by DR. MARY J. WILMERS (London). 'A Follow-up Study of Children Born to Diabetic Mothers, with particular reference to frequency of congenital abnormalities.' The frequency of congenital abnormalities in children of diabetic and non-diabetic mothers is still uncertain.

This study aimed to compare the serious congenital abnormalities in 206 viable infants born consecutively to diabetic mothers at King's College Hospital between 1956 and 1961, with 206 control infants born to non-diabetic mothers in the same hospital, over the same period. Almost all survivors in both groups were examined personally on at least one occasion between the ages of 3 and 11 years. Follow-up was completed on 96% of the 'diabetic' offspring and 87% of the controls.

Congenital abnormalities were found in 21 (10.7%) of 197 infants in the 'diabetic' group and 10 (5.6%) of 179 controls. This difference was not statistically significant, but there was a significant trend for major abnormalities to be associated with maternal diabetes. The frequency of congenital heart disease, cerebral palsy, and mental retardation was particularly striking in the 'diabetic' group. 163 (92%) of their sibs were also followed up. 18 children (11.0%) had congenital abnormalities which showed a similar distribution to those in the main 'diabetic' group.

Defects were not significantly increased in children

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

DAVID 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.

I. 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.

N. R. C. ROBERTON introduced by PROFESSOR J. P. M. TIZARD (London). 'Relation between Arterial Oxygen Tension, Systemic Blood Pressure, and Electroencephalogram in the Respiratory Distress Syndrome.' To be published in full.

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.

BERNARD KLIONSKY 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. Conspicuous 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.

NINA 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.