ing the newborn population in Northern Ireland (birth rate 33,000 per year) for hyperphenylalaninaemia with the ‘Guthrie’ technique over a two-year period are presented and compared with our previous results of ferric chloride testing of urine.

It is now recognized that not all patients with hyperphenylalaninaemia are instances of classical PKU. The importance in distinguishing between the two groups of patients lies in the fact that a phenylalanine restricted diet given unnecessarily will in itself cause failure to thrive and perhaps even brain damage. Distinction has been attempted between classical PKU and its hyperphenylalaninaemia variants; the incidence of the two forms and our experience of their management are recorded.


‘The natural history of phenylketonuria diagnosed by screening in infancy is obscured by the effect of early dietary treatment. It is, however, known that a ‘normal’ IQ may occur in untreated phenylketonuria. Cases of phenylketonuria referred to hospital on account of mental retardation do not represent an unbiased sample. A study of sib pairs among a series of 176 consecutive cases allowed us to avoid this bias, and enabled an estimate to be made of the prevalence of a ‘normal’ IQ in the untreated condition.

†The series provided a total of 38 sib pairs. In 18 of these the index case was diagnosed because of mental retardation, and the sib was discovered by chemical screening of the family at the time of diagnosis of the index case. The IQ range of the index cases was 20–55 with a mean of 37, and the IQ range of the sib cases was 20–100 with a mean of 49, 3 out of the 18 siblings having IQs above 80. In this small group of 18 cases, in which the chemical diagnosis was made not because of mental retardation but because of the discovery of an affected sib, 1 in 6 of the children had an IQ within the ‘normal’ range.

A. H. CAMERON, J. H. EDWARDS, and I. MYSKOVA (Birmingham). ‘Placentalation and Fetal Growth in Twins.’ This paper described an analysis of 1000 twin pairs born in Birmingham. The birthweight and morbidity were related to the different types of placentalation. The two main types of vascular communication within the placenta and their effects on the fetus were described.

D. J. GIRLING introduced by DR. J. S. WIGGLESWORTH (London). ‘Pulmonary Fibroplasia in Newborn Babies Treated with Oxygen and Artificial Ventilation.’

Pulmonary fibroplasia developing in babies subjected to intensive care for respiratory problems in the neonatal period has been ascribed variously to O2 toxicity, ventilator therapy, or the natural progression of severe hyaline membrane disease. In order to assess the relative importance of these factors a clinicopathological study has been performed on all babies who died in the Neonatal Unit of the Hammersmith Hospital from November 1965 to October 1970, who received oxygen therapy and who lived for 48 hours or more.

Of the 81 babies studied 39 had hyaline membrane disease and 64 received artificial ventilation. Unequivocal changes of pulmonary fibroplasia were present in 23 cases.

The main findings were as follows:

1. Of babies treated only with less than 60% O2, none developed pulmonary fibroplasia.
2. Of babies treated with over 80%, O2 for more than 105 hours, all developed pulmonary fibroplasia.
3. Pulmonary fibroplasia was seen in a few babies who had less than 48 hours artificial ventilation and was absent in several subjected to prolonged ventilation.
4. The most severe histological changes of pulmonary fibroplasia were almost invariably associated with hyaline membrane disease.
5. Gestational age had no effect on the development of pulmonary fibroplasia.

The implications of these findings for O2 therapy of the newborn were discussed.

M. A. LE DUNE introduced by DR. I. RILEY (Glasgow). ‘Insulin and Glucose Estimations in Hypoglycaemic Infants.’ To be published.

H. STEINER introduced by DR. G. NELIGAN (Newcastle). ‘The Quality of Survivors of Very Severe Perinatal Problems.’ To be published elsewhere.

B. J. N. Z. DANESH introduced by DR. C. H. M. WALKER (Dundee). ‘Influence of Acid Citrate in Stored Blood on the Level of Electrolytes in Plasma.’ Citrate intoxication resulting from multiple transfusions of citrated blood is a well known hazard. Convolutions and cardiac arrest leading to death have been reported in neonates receiving exchange transfusions for erythroblastosis fetalis. These dangers are greater in the presence of hepatic and renal dysfunction, hypothermia, and when there is mechanical obstruction of hepatic circulation during cardiac surgery.

To gain more understanding of the influence of acid citrate on the level of electrolytes in plasma, studies were performed using semipermeable membranes and the technique of equilibrium dialysis.

It was found that citrate not only decreased the ionized fraction of plasma calcium and magnesium by forming citrate salts, but it also appears to combine with the protein bound fraction which, in the absence of citrate, would dissociate to take the place of ionized fraction which has become complexed with citrate. This changes the normal pattern and the total value of these electrolytes present in the blood.

In conditions where citrate intoxication is an impending danger, rapid transfusions of citrated blood should be avoided.