PEDIATRIC NEUROLOGY BRIEFS

A MONTHLY JOURNAL REVIEW

J. GORDON MILLICHAP, M.D., F.R.C.P., EDITOR

Vol. 2, No. 11 November 1988

METABOLIC DISORDERS

NEONATAL HYPOGYCEMIA

The definition of neonatal hypoglycemia has been surveyed at the Department of Child Health, University of Newcastle upon Tyne by reference to 36 textbooks of pediatrics and answers to questionnaires from 178 pediatricians in charge of nurseries with 4 or more intensive care cots. In textbooks there was a wide variation in definition ranging from a glucose concentration of <1 mmol/l to <2.5 mmol/l with a modal value of <1.7 mmol/l for term babies of appropriate weight, and <1.1 mmol/l for babies who were preterm or small for gestational age. Among practicing pediatricians the definitions showed an even greater range from a glucose concentration of <1 mmol/l to <4 mmol/l with a modal value of <2.0 mmol/l for term babies of appropriate weight and <1.1 mmol/l for babies who were preterm or small for gestational age. The site of sampling -- that is, whether capillary, venous, or arterial blood -- was not included in the definition of hypoglycemia obtained from textbooks nor from pediatricians surveyed. There appeared to be no accepted definition of the lower limit of normality for circulating blood glucose concentrations. (Koh THHG, Eyre JA, Aynsley-Green A, Neonatal hypoglycemia -- the controversy regarding definition. Arch Dis Child Nov 1988;63:1386-1398).

COMMENT. A functional definition of hypoglycemia was suggested by the authors to be based on a correlation between objective measurements of neurophysiological

PEDIATRIC NEUROLOGY BRIEFS © 1988 covers selected articles from the world literature and is published monthly. Subscription requests ($28 US or £ 15 UK annually; add $5 (£ 3) for airmail outside North America) may be sent to: Pediatric Neurology Briefs - J. Gordon Millichap, MD, FRCP - Editor, P.O. Box 11391, Chicago, IL 60611, USA, or P.O. Box 1605, London W8 7JD, UK. The Editor, Professor of Neurology and Pediatrics at Northwestern University Medical School, Chicago, is presently at SIU School of Medicine, Springfield, Illinois, USA.

function and blood glucose concentrations. The "safe" blood glucose concentration may vary according to the clinical situation, eg. during hypoxia, polycythemia, or convulsions, and may be independent of the gestational and postnatal age and birth weight. Clinical signs of hypoglycemia in the neonate are not well established and the question of neural dysfunction or damage with asymptomatic hypoglycemia is addressed in the following paper.

HYPOGLYCEMIA AND NEURAL DYSFUNCTION

Evoked potentials were used to measure neural function in relation to blood glucose concentration in 17 children at the Department of Child Health, University of Newcastle upon Tyne. Thirteen children were admitted for investigation of metabolic or endocrine disorders and hypoglycemia was provoked by fasting or insulin administration; 4 had recurrent episodes of spontaneous hypoglycemia. Abnormal brainstem auditory evoked potentials were recorded in 9 and abnormal somatosensory evoked potentials in 1 of the 11 children whose blood glucose concentration remained above 2.6 mmol/l; 5 of these 10 children were asymptomatic and 5 became drowsy. No change in evoked potentials was recorded in the 6 children whose blood glucose concentration remained above 2.6 mmol/l. Of the 10 children with abnormal evoked potentials, 6 had normal latencies immediately after the IV administration of 25% dextrose (2ml/kg) or the IM administration of glucagon (20 ug/kg), whereas 4 had persistently prolonged I-V latencies or absent wave V for 1 hr, 1.5 hrs, 16 hrs, and 2 days. The authors suggest that blood glucose concentration should be maintained above 2.6 mmol/l to ensure normal neural function in newborns and children irrespective of the presence or absence of abnormal clinical signs. (Koh THHG, Aynsley-Green A, Tarbit M, Eyre JA, Neural dysfunction during hypoglycemia. Arch Dis Child Nov 1988;63: 1353-1358).

COMMENT. A prospective controlled study of neonates with episodes of hypoglycemia has shown that long term follow up may reveal impairments in intellectual function (Pildes et al. Pediatrics 1974;54:5). Short term follow up studies of infants who have suffered hypoglycemia may be misleading. Clinical signs of hypoglycemia and disturbances of neural function in the newborn baby may be subtle and difficult to recognize, and the distinction between "symptomatic" and "asymptomatic" hypoglycemia may require recordings of evoked potentials to demonstrate neural dysfunction. Whether the transient abnormalities in evoked potentials demonstrated in the above study can be predictive of permanent neural damage remains to be determined. The neurological signs resulting from abnormal cerebral metabolism secondary to hypoglycemia may depend on the rate of fall of blood sugar, the duration and degree of hypoglycemia, and the age of the patient. (Etheridge JE.