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NEONATAL ENCEPHALOPATHY

CLASSIFICATION OF NEWBORN ENCEPHALOPATHY

Investigators from the Department of Neurology, Children's Hospital and Harvard Medical School, Boston and the Neuroepidemiology Branch, Bethesda take issue with the terms hypoxic-ischemic and post-asphyxial encephalopathy when the etiology is frequently in doubt. These authors prefer the simple descriptive terms, neonatal or newborn encephalopathy for infants with a clinically defined syndrome of impaired neurologic function, which includes lethargy, coma, hypotonia, impaired primitive reflexes, depressed or absent respirations and seizures. Difficulties in initiating respiration and maintaining tone and consciousness may be caused by factors that predate birth or onset of labor. Neonatal encephalopathy grading schemes previously described in the literature (Sarnat and Sarnat, Low et al, Levene et al, Amiel-Tilson and Ellison, Fenichel) are considered qualitative rather than quantitative and their potentially correctable limitations are discussed. (Leviton A, Nelson KB. Problems with definitions and classifications of newborn encephalopathy. Pediatr Neurol March/April 1992; 8:85-90.) (Correspondence: Dr. Leviton, Children's Hospital, 300 Longwood Avenue, Boston MA 02115.)

COMMENT. In a previous issue of Pediatric Neurology (1991; 7:317-325), Dr. Alan Hill of the University of British Columbia Children's Hospital, Vancouver, reviewed the diagnosis, classification, pathology and management of hypoxic-ischemic cerebral injury in the term newborn. Grade III severe HIE, manifested by coma, hypotonia, seizures, abnormal brain stem and autonomic function, and increased intracranial pressure, is associated invariably with death or severe neurologic sequelae. Grade II moderate HIE, with lethargy/stupor, hypotonia, suppressed reflexes and seizures, has a poor outcome in 20-40% of infants. Grade I mild HIE, characterized by hyperalertness, jitteriness, exaggerated Moro and stretch reflexes, is transient with

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duration less than 24 hours and recovery is complete. The author emphasizes that the clinical features of hypoxic-ischemic encephalopathy are not specific and that similar symptoms may be caused by metabolic disorders, infection or cerebral dysgenesis.

BIRTH ASPHYXIA, CEREBRAL METABOLISM AND HEAD SIZE

Studies of cerebral oxidative metabolism were carried out by phosphorus magnetic resonance spectroscopy during the first week of life in 52 infants with birth asphyxia admitted to the Neonatal Unit at University College Hospital, London. Cerebral phosphocreatine/inorganic phosphate concentration ratio was used as an index of oxidative metabolism and correlated with neurodevelopmental outcome and head growth at 1 year. (Roth SC et al. Relation between cerebral oxidative metabolism following birth asphyxia, and neurodevelopmental outcome and brain growth at one year. Dev Med Child Neurol April 1992; 34:285-295.) (Correspondence: Dr. Simon C. Roth, Department of Paediatrics, University College and Middlesex School of Medicine, The Rayne Institute, 5 University Street, London WC1E 6JJ, England.)

COMMENT. The use of these neuroimaging and biochemical techniques should help in the prediction of outcome following neonatal encephalopathy. However, the complexity of the techniques may detract from their value in practice. (Bax M. Editorial. Birth asphyxia. Dev Med Child Neurol 1992; 34:283-284.)

Central diabetes insipidus as an unusual complication of hypoxic brain damage is described in 2 children at the Department of Pediatrics, Juntendo University School of Medicine, Tokyo, Japan (Arisaka O et al. Child's Nerv Syst March 1992; 8:81-82). Both patients developed cardiopulmonary arrest after choking and both had hypernatremia and low urinary antidiuretic hormone concentrations in the terminal stages. The most common causes of central diabetes insipidus are tumors or trauma in the neurohypophyseal area.

NEUROMUSCULAR DISEASES

MYOTONIC DYSTROPHY: SEVERITY AND MATERNAL AGE

The severity of myotonic dystrophy in 17 affected sibling pairs from 15 families in which 2 or more affected children were born to mothers with myotonic dystrophy is reported from the Hospital for Sick Children, London and the Prince of Wales Children's Hospital, Sydney, Australia. In 13 of 17 sibling pairs the younger child was more severely affected than the older child. Increasing age difference between the affected siblings paralleled increasing age for each mother and showed a positive correlation with the difference in disease severity between siblings. Infants born to older mothers suffered more severe myotonic dystrophy. Maternal age at delivery correlated with the age at which the infant sat alone and walked alone. In addition, the incidence of neonatal feeding difficulties, neonatal respiratory difficulties, surgery for talipes, and scoliosis were directly related to maternal age at delivery. (Andrews, PI, Wilson, J. Relative disease severity in siblings with