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

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SEIZURE DISORDERS

HIGH-RISK MARKERS FOR ASPHYXIAL NEONATAL SEIZURES

The value of clinical high-risk markers in detecting neonates having seizures within the first hour of life following intrapartum asphyxia was evaluated in term infants admitted to the neonatal intensive care unit at the University of Texas Southwestern Medical Center, Dallas, TX. Seizures developed in 5 (5.2%) of 96 infants with hypoxia ischemia or asphyxia. High-risk markers included fetal heart rate (FHRT) abnormalities only (36), FHRT abnormalities and meconium-stained amniotic fluid (MSAF) (20), MSAF only (23), 5 or less 5 min Apgar scores (21), umbilical cord arterial pH of 7 or less (21), and base deficits of -14 mEq/L (19). Significant relationships with seizures occurred with a combination of low 5-min Apgar scores, and the need for intubation in the delivery room in association with severe fetal acidemia. (Perlman JM, Risser R. Can asphyxiated infants at risk for neonatal seizures be rapidly identified by current high-risk markers? *Pediatrics* April 1996;97:456-462). (Reprints: Dr JM Perlman, University of Texas Southwestern Medical Center at Dallas, 5323 Harry Hines Blvd, Dallas, TX 75235).

COMMENT. The combination of postnatal high-risk markers, 1) low 5-min Apgar score, and 2) severe fetal acidemia and intubation in the delivery room, will identify within the first hour after birth those infants at high risk for seizures resulting from perinatal asphyxia. Infants with subclinical seizures may have been overlooked since EEGs were not done routinely.

Neonatal seizures caused by asphyxia carry a poor prognosis; 43% had a poor outcome in a Dublin Collaborative study reported by Curtis PD et al, 1988. See *Progress in Pediatric Neurology I & II*, PNB Publ, 1991, 1994, for further articles concerning risk factors, prognosis, and the value of the EEG in prediction of continued seizure activity beyond the neonatal period.

RISK FACTORS OF INFANTILE SPASMS

The role of a genetic predisposition and other risk factors for seizures was analyzed by evaluation of records of 80 children with infantile spasms and

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The editor is Pediatric Neurologist at Children's Memorial Hospital and Northwestern University Medical School, Chicago, Illinois.

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compared with 474 children with other types of epilepsy, and 2196 children with febrile seizures, admitted with neurologic disorders at age 1 month to 2 years to 12 Danish pediatric departments during 1967-68 and 1972-73. There was a family history of seizures in 14% of children with infantile spasms, compared to 29% in children with other epilepsies, 26% in those with febrile seizures, and 5% in children admitted for CNS infections. A family history of seizures increased the risk for infantile spasms threefold, but only in the cryptogenic cases that made up 50% of the group. Neonatal hypoxia, neonatal seizures, and CNS malformations were much stronger predictors of infantile spasms than genetic factors. The relatively low incidence of organic cases in this study was explained by the lack of available brain imaging. (Rantala H, Shields WD et al. Risk factors of infantile spasms compared with other seizures in children under 2 years of age. Epilepsia April 1996;37:362-366). (Reprints: Dr H Rantala, Department of Pediatrics, University of Oulu, FIN-90220 Oulu 22, Finland).

COMMENT. A family history of seizures contributes to a 3-fold risk for infantile spasms of the cryptogenic variety. Organic brain disorders and neurologic abnormalities play a much stronger role as precursors of infantile spasms. Neonatal seizures are particularly predictive of possible occurrence of infantile spasms.

Long-term outcome of West syndrome was studied in 214 children with a history of infantile spasms followed for 20-35 years or until death (31%) at the Children's Hospital, University of Helsinki, Finland. (Riikonen R. Epilepsia April 1996;37:367-372). Infection was the most frequent cause of death (31 of 67 patients). Eight children who died during treatment with large doses of ACTH had enlarged adrenal glands and hypertrophic cardiomyopathy. Among survivors, intelligence was normal or near normal in 24%. Factors predictive of a good prognosis included a cryptogenic etiology, normal development before onset of spasms, and a good response to ACTH. Focal abnormalities in the EEG were not necessarily indicative of a poor prognosis.

Treatment with high-dose ACTH (150 U/m²/day) was superior to prednisone (2 mg/kg/day) in suppressing clinical spasms and hysarrhythmia in the EEG in a prospective, randomized, blinded study of 29 patients (22 symptomatic and 7 cryptogenic etiologies) treated for 2 week periods at the University of Southern California, Los Angeles (Baram TZ et al. Pediatrics March 1996;97:375-379). Of 15 patients randomized to ACTH, 13 (87%) responded, compared to 4 (29%) of 14 given prednisone.

LOCALIZING VALUE OF CLINICAL SEIZURE PATTERNS

The value of ictal clinical manifestations in differentiating frontal and temporal lobe partial epilepsies was determined in a prospective study of 252 patients selected according to imaging, EEG, and focal clinical patterns from records at the National Hospital for Neurology and Neurosurgery, London, UK. Cluster analysis gave 14 distinctive clinical types of patterns, but these had limited localizing value with the exception of perioral seizures. Two seizure types with prominent, early motor manifestations, especially version and posturing, were associated with frontal lobe abnormalities. Very high seizure frequencies were seen more often with frontal lesions. Seizures associated with temporal lobe lesions were characterized by absences and those with subjective onsets such as fear and orolimentary automatisms. Location of interictal EEG spikes and ictal EEG onsets were generally consistent with lesion sites. Relatively few seizures could be localized reliably on clinical grounds. (Manford M et al. An analysis of clinical seizure patterns and their