1991; 88:115-120). Only 11 (31%) of 36 children had normal development upon reassessment in early childhood. With or without major complications, extremely low birth weight places children at risk for emerging development problems with age. Head circumference was not addressed in this study.

PRENATAL ETIOLOGY OF CEREBRAL PALSY

The incidence of minor malformations in a group of 137 adults with cerebral palsy, living in a New York State residential institution, is reported from the Division of Developmental Pediatrics, Robert Warner Rehabilitation Center, State University of New York, Buffalo, NY. Subjects were classified in three etiological groups: (1) prenatal onset, (2) postnatal onset, and (3) perinatal onset. Criteria for enrollment in group 1 included a known prenatal cause (i.e., CNS malformation, chromosomal aberration or congenital infection) or fullterm birth with unremarkable delivery and neonatal course. Of 109 patients with CP of prenatal origin, 82% had unknown causes, 10% CNS malformation, 5% chromosomal aberration and 3% congenital infection. Of 28 patients with postnatal causes, 39% had bacterial meningitis, 32% encephalitis, 11% metabolic disease, 7% cardiopulmonary arrest and 7% trauma. Spastic quadriplegia, profound mental retardation, and seizures occurred in 80-90% of the total sample, and the neurological findings were similar in the two groups. The prenatal group with known etiology and a subgroup with unknown etiology had significantly more minor malformations than the postnatal group. Individuals with CP and multiple minor malformations, in the absence of definitive postnatal or perinatal factors, have CNS developmental dysfunction caused most likely by prenatal influences. (Coorssen EA et al. Multiple minor malformations as a marker for prenatal etiology of cerebral palsy. Dev Med Child Neurol August 1991; 33:730-736).

COMMENT. Increased numbers of minor malformations are indicative of aberrant embryonic development of the CNS (Illingworth R. "Why blame the obstetrician? A review." BMJ 1979; 1:797-801) Less than 10% of CP is caused by perinatal asphyxia or events related to delivery (Blair E, Stanley F. "Intrapartum asphyxia: a rare cause of cerebral palsy." J Pediatr 1988; 112:515-519).

INFANTILE SEIZURES

INFANTILE SPASMS AND PARTIAL SEIZURES

The concurrence of infantile spasms and partial seizures was studied in 11 infants using time-locked video electroencephalography at the Department of Pediatrics, Children's Hospital, Ohio State University School of Medicine, Columbus, OH. Partial seizures preceded the onset of infantile spasms in 7. In some, the focal EEG ictal activity was overriding the generalized slow-wave transient and in others was visible only during the generalized decrement. Interictal EEGs showed hypsarrhythmia in 6 patients and modified hypsarrhythmia in 5. Brain malformations occurred in 3 and tuberous

sclerosis in 2. Accurate clinical identification of both types of seizures was not made without resort to video EEG. (Donat JF, Wright FS. Simultaneous infantile spasms and partial seizures. J Child Neurol July 1991; 6:246-250).

COMMENT: The authors refer to only one previous account in the literature of the simultaneous occurrence of the two seizure types (Hrachovy RA et al. Epilepsia 1984; 25:317-325). The concurrence of these seizures might support both cortical and subcortical mechanisms in the genesis of infantile spasms. Information on the relative effect of ACTH and other forms of treatment of these seizure types would be of interest.

A long term follow-up study of 42 patients with West syndrome treated with high doses of sodium valproate is presented from the Neuropaediatric Unit, Hospital de Ctuces, Bilbao, Spain. The hypsarrhythmia EEG pattern was controlled after two weeks treatment with VPA 100 to 300mg/kg/daily. In 80% of patients relapses occurred most often in those treated with doses lower than 200 mg/kg/day. Other types of seizures developed in 50% of patients followed beyond two years of age. Side effects included thrombocytopenia, vomiting and somnolence. Hepatic enzymes were elevated in 3 patients and returned to normal when treatment was ended (Prats JM et al. Dev Med Child Neurol August 1991; 33:617-625). The authors recommend that high dose valproate requires careful monitoring and admission to hospital. Somnolence, vomiting, thrombocytopenia and coagulation defects are of concern and warrant further evaluation before more wide acceptance of this therapy.

SEIZURES AND PARTIAL HYPOPARATHYROIDISM

Neonatal hypocalcemia and seizures in three infants who developed recurrence of hypocalcemia later in childhood are reported from the Department of Paediatrics, The Hospital for Sick Children, Toronto, Ontario, Canada. Hypocalcemia resolved in 1 week, 3 months, and 14 months and recurrences were noted at 4, 7 and 12 years of age. Plasma parathyroid hormone concentrations were low but detectable. One patient born full term with normal birth weight became irritable and developed tonic-clonic convulsions on the first day of life. The plasma calcium was 1.43 mmol/L; magnesium, 0.86 mmol/L; and phosphate 1.95 mmol/L. She was hospitalized again at 4 weeks of age for asymptomatic hypocalcemia. Except for generalized development delay first noted at 7 months of age, she had no clinically important illness until 12-1/2 years of age when she experienced difficulty bending her fingers and numbness of her hands and feet. Several months later, following a minor viral illness, she lost consciousness while watching television and developed convulsions. The plasma calcium was 1.35 mmol/L and ionized calcium 0.69 mmol/L. She has received therapy with calcitriol for the last 3 years and has maintained a normal plasma calcium concentration. In patients 2 and 3 the childhood hypocalcemia developed gradually over several years. The authors caution that congenital