neurologic exams (7.7%). Relapse was related to the age of onset of the seizure disorder; the younger the age of onset the greater the risk of relapse. Of the 22 children who relapsed 67% had a seizure while drug therapy was being withdrawn or within one year of withdrawal, and 86% relapsed within two years. Of 49 children who had EEGs before drug withdrawal the incidence of relapse was 19% in the normal EEG group (37) and 25% in the abnormal group (12) and the difference was not significant. The authors concluded that prewithdrawal EEG for a neurologically normal child is not of prognostic benefit. Since some relapses occurred as late as eight years after withdrawal the authors recommended that follow-up should continue for ten years. (Ehrhardt P, Forsythe WI. Prognosis after grand mal seizures: A study of 187 children with three-year remissions. Dev Med Child Neurol October 1989; 31:633-639).

COMMENT. Recommendations for the withdrawal of anticonvulsant treatment vary, some employing a two year remission, others a three year remission, and some four, five, and even ten year remission before withdrawing treatment. The present paper was more specific than some regarding the relationship of the clinical characteristics of the patients in relation to prognosis. The group of children with neurological dysfunction and mental retardation was small comprising only 13 of the total group and the actual risks of drug withdrawal could not be assessed in this investigation. Of those patients who had EEGs before drug treatment was discontinued, the relapse rate was almost double the relapse rate in the total group. In those with abnormal EEGs the relapse rate was 25%. It would be interesting to know the factors involved in deciding which of these patients required EEGs. This group may deserve further study.

A paper reviewed in the last issue of Ped Neur Briefs (1989; 3:83) showed that the relapse rate after drug withdrawal in 425 children with epilepsy was 12% (the same rate of relapse as in the present study) and the risk was greatest in the first year. Factors related to relapse were neurologic abnormalities and organic etiology, mental retardation, seizure type (infantile spasms, absence seizures), and the appearance or persistence of EEG abnormalities during the course of the illness and before discontinuation of the drugs. The value of the EEG before drug withdrawal as a predictive factor for relapse cannot be discounted.

TOXIC AND METABOLIC DISORDERS

LOW DOSE LEAD AND CNS DEFICITS:

The long term effects of exposure to low doses of lead in childhood have been examined in 132 of 270 young adults who had

initially been studied as primary school children in 1975-1978 and the results of an 11 year follow-up are reported from the School of Medicine, University of Pittsburgh; Boston University; and the Neuroepidemiology Unit, Children's Hospital and Harvard Medical School, Boston, MA. Neurobehavioral functioning in the earlier study of school children was found to be inversely related to dentin lead levels. In the subjects reexamined as adults, impairment in neurobehavioral function was still related to the lead content of teeth shed at the ages of six and seven. The persistent toxicity of lead was seen to result in significant and serious impairment of academic success, specifically a sevenfold increase in failure to graduate from high school, lower class standing, greater absenteeism, impairment of reading skills, and deficits in vocabulary, fine motor skills, reaction time, and hand-eye coordination. A dose response relation was demonstrated between exposure and numerous outcome variables. Young people with dentin lead levels greater than 20 PPM had a markedly higher risk of dropping out of high school and of having a reading disability as compared with those with dentin lead levels less than 10 PPM. (Needleman HL et al. The long-term effects of exposure to low doses of lead in childhood: An 11-year follow-up report. N Engl J Med Jan 11, 1990; 322:83-88).

COMMENT. Exposure to lead even in children who remain asymptomatic may have an important and enduring effect on brain function and learning. Since 16% of children in the United States are reported to have elevated blood lead levels (greater than 15 mcg/dl), the early detection and attention to lead in the environment might prevent school failure in a significant number of children in the USA. The agency for Toxic Substances and Disease Registry has defined the threshold for neurobehavioral toxicity for lead as 10-15 mcg/dl. The mean blood level among the subjects reported with high tooth lead levels was 34 mcg/dl.

In the Sydney Lead Study, a prospective investigation of the relationship between low level lead exposure and neurobehavioral development during the first five years of life, average blood lead levels at the fourth year were approximately 10 mcg/dl and this degree of lead exposure was not associated with mental or motor deficits. (Cooney GH et al. Low-level exposures to lead: The Sydney Lead Study. Dev Med Child Neurol 1989; 31:640-649). A meta-analysis of 24 modern studies of childhood exposures to lead in relationship to IQ inferred that low dose lead exposure is closely associated with deficits in psychometric intelligence (Needleman HL, Gatsonis CA. Low-level lead exposure and the IQ of children. A meta-analysis of modern studies. JAMA Feb 2, 1990; 263:673-678). The level of lead exposure in these studies may have been higher than that in the Sydney Lead Study.

In another current study of lead exposure in preschool children, the calcium status of 64 black urban children aged 18-47 months

was evaluated in relation to blood lead levels and behavior, particularly pica. Children with blood levels less than 30 mcg/dl were compared with a group having blood levels greater than 30 mcg/dl. The study verified the positive association between blood lead levels and pica, an association recognized for many years. Decreased calcium intake and three other calcium measures were not related to blood lead levels and calcium intake was not associated with pica scores. (Laraque D et al. Blood lead, calcium status and behavior in preschool children. AJDC Feb 1990; 144:186-189). Pica has been emphasized as a common prelude to plumbism (Millichap JG et al. Lead paint: A hazard to children. Lancet 1952; 2:360) and should prompt the early diagnosis of lead exposure and prevention of neurobehavioral deficits. The identification of children with lead poisoning in need of chelation is possible using unstimulated urinary lead excretion without the necessity of the $CaNa_2EDTA$ provocative test. (Berger OG et al. Using unstimulated urinary lead excretion to assess the need for chelation in the treatment of lead poisoning. J Pediatr 1990; 116:46-51).

POLYCHLORINATED BIPHENYLS (PCBs) AND COGNITIVE DEFICITS

The effects of prenatal exposure to polychlorinated biphenyls (PCBs) and related contaminants on the CNS function of infants born to women who had consumed Lake Michigan sports fish have been investigated in 236 children previously evaluated for PCB-related deficits in infancy and reassessed at four years of age in the Psychology Department, Wayne State University, Detroit, MI and the Michigan Department of Public Health, Lansing, MI. Prenatal exposure, indicated by umbilical cord serum PCB levels, was associated with poorer short term memory function on both verbal and quantitative tests and the adverse effects were dose dependent and not attributed to other variables. Exposure from nursing was unrelated to cognitive performance. The study demonstrates continuation of toxic effects through early childhood. (Jacobson JL et al. Effects of in utero exposure to polychlorinated biphenyls and related contaminants on cognitive functioning in young children. J Pediatr Jan 1990; 116:38-45).

COMMENT. Polychlorinated biphenyls were once used in industrial products and were banned in the United States in 1970. Residues persist in air, soil, water and sediments in lakes and can be detected in residents of industrialized countries. PCB levels are unusually high in sports fish from Lake Michigan and transplacental exposure to PCBs has been documented.

PARTIAL BIOTINIDASE DEFICIENCY

The symptoms, biochemical features and inheritance pattern of partial biotinidase deficiency have been studied at the Departments of Human Genetics and Pediatrics, Medical College of Virginia, Richmond,