

(50%) and Lennox-Gastaut (43.7%) syndromes. Grand mal and complex partial seizures recurred in 27.8% and 23.3% of patients, respectively. The majority of relapses were single isolated seizures and occurred in the first year after drug withdrawal.

Risk factors for seizure relapse included 1) delay in initiating anticonvulsant therapy, 2) a symptomatic etiology, 3) mixed seizure types, 4) status epilepticus occurrence, and 5) signs of permanent neurologic damage. (Oller-Daurella L, Oller, F-V L. Suppression of antiepileptic treatment. Eur Neurol 1987; 27:106-113).

COMMENT: The 5 year seizure-free period before drug withdrawal in this study is longer and approximately twice that employed by other investigators. Shinnar S et al (N Engl J Med 1985; 313:976) reported the same 25% relapse rate among 89 children who had been seizure free for only 2 years.

The withdrawal of antiepileptic medication should not be determined by a set seizure-free time-period for all types of epilepsy. Each patient should be evaluated individually, having regard to several factors, including psychological, predictive of potential relapse. Withdrawal is probably contra-indicated or likely to be unsuccessful in patients with the following: 1) symptomatic epilepsies with radiological or neurological evidence of structural cerebral defect, mixed seizure types, Lennox-Gastaut syndrome, focal seizures, complex partial with secondary generalized tonic-clonic patterns, 3) history of status epilepticus and 4) psychological stress, especially in adolescents and young adults.

The prognostic value of the EEG is controversial; these authors and Thurston JH et al (N Engl J Med 1982; 306:831) found a negative correlation whereas others regard the EEG as an important factor, predictive of a good outcome when normal (Shinnar S et al 1985). An abnormal EEG was one of the most significant predictors of relapse after drug withdrawal in the study by Emerson R et al (N Engl J Med 1981; 305:1125).

CARBAMAZEPINE AND COGNITIVE IMPAIRMENT

Members of the Departments of Pediatrics, Neurology, and Clinical Pharmacology and Toxicology at the Children's Hospital and Ohio State University, Columbus, OH 43205, assessed neuropsychological function before and after carbamazepine monotherapy at low (<7.5,ug/ml) and moderate (>8.0,ug/ml) plasma levels in 11 children (4 boys 7 girls, mean age 9.8 yrs) with controlled complex partial epilepsy. Carbamazepine caused significant impairments (P<.03) of efficiency in learning of new information (Paired Associates Test) and short-term memory scanning (Sternberg Memory and Reaction Time Paradigm) that were associated with moderate plasma concentrations within the therapeutic range. The decline in performance was not accompanied by a greater abnormality on the EEG or carbamazepine-induced seizure exacerbation (see Pediatric Neurology Briefs, Vol 1, No 1). A mild beneficial effect on speeded eye-hand coordination was suggested at moderate plasma levels but only in the nonpreferred hand. Except for a trend toward more rapid memory scanning, there was no change in performance from the baseline to low drug level assessment. (O'Dougherty M, Wright FS et al. Carbamazepine plasma concentration. Relationship to cognitive impairment. Arch Neurol 1987; 44:863-867).

COMMENT: These results are in agreement with previous studies in adults that have shown impairments in concentration and memory-processing with higher

but therapeutic serum concentrations of carbamazepine (Thompson PJ, Trimble MR. J Neurol Neurosurg Psychiatry 1983; 46:227-233). It has been suggested that the so-called "psychotropic" effect of carbamazepine reported in cross over antiepileptic drug studies may have been related to the discontinuance of previous drugs rather than a positive carbamazepine effect and that cross over studies are potentially open to error by practice effects (Schain RJ et al. Neurology 1977; 27:476-480). The present study confirms the importance of comprehensive neuropsychological assessments to evaluate possible adverse cognitive side effects of antiepileptic drugs in children particularly at higher dose levels. The theoretical advantages of monotherapy, notwithstanding, the tendency to rigid persistence of large and potentially toxic doses and delay in change to alternative therapy may result in subtle deficits in learning that might be avoided by selective combination therapies at lower dose levels.

BEHAVIOR AND LEARNING DISABILITIES

HARMFUL EFFECTS OF LEAD ON LEARNING

Members of the Depts. Community Medicine, Education, Geology, and Med. Statistics Unit, Univ. Edinburgh, have investigated the effect of blood-lead on cognitive ability and educational attainment in a sample of 855 boys and girls aged 6-9 years from 18 primary schools in central Edinburgh. The mean blood-lead level was 10.4 ug/dl. Multiple regression analyses of individual test scores showed a significant negative relation between blood-lead and British Ability Scales combined scores, number skills, and word reading, with 33 possible variables accounted for. The dose-response relation between blood-lead and test scores showed no evidence of a threshold or safe level. It was concluded that lead at low levels of exposure probably has a small harmful effect on the performance of children in cognitive ability and attainment tests. (Fulton M et al. Lancet 1987; 1:1221).

COMMENT: This finding is in agreement with that of a previous study in the USA (Needleman et al. N Engl J Med 1979; 300:689-95) showing lead-related deficits in neuropsychological and classroom performance of children with elevated dentine lead levels. Exposure levels in the UK were lower than in the US study. Water and dust were the main sources of lead, attributed to a plumbosolvent water supply and lead plumbing in Edinburgh. Reports of research (1979-83) on the neuropsychological effects of lead in children are reviewed by the Medical Research Council, London, 1984.

A case of schizophrenic-like psychosis is an unusual manifestation of moderate lead intoxication (blood level of 60 ug/dl) reported in a 14 year old boy who had sniffed gasoline for 3 months. He was treated at Duke Univ Med Cntr, Durham, N Carolina, using a Ca EDTA challenge and 4 days chelation with dramatic clearing of agitation and psychotic symptoms. He had a history of dyslexia, visual-motor incoordination and conduct disorder. His IQ was 83 at 9 years of age and 69 on recovery from the lead intoxication. A possible psychobiological vulnerability to lead intoxication in children with learning problems, ADD, or mental retardation is proposed. (McCracken JT. J Amer Acad Child Adol Psychiat 1987; 26:274-276).

NEUROPSYCHOLOGICAL SEQUELAE OF REYE'S SYNDROME

The author, a pediatric neurologist at the U. of Kansas School of Medicine, Wichita, Kansas, reviews the sequelae and risk factors in survivors of Reye's syndrome. He reports a