Hospital, Birmingham, Alabama (Snead OC, Miles MV. J Pediat 1985;106:323). A loading dose of 20 mg/kg was effective but a marked rise in serum glumatic oxaloacetic transaminase activity occurred in 3 of 7 patients treated, requiring cessation of valproate therapy.

ICTAL VOMITING

Paroxysmal vomiting in 9 patients, 3 children (7, 8, and 11 years of age) and 6 young adults, is reported from the Section of Epilepsy and Clinical Neurophysiology, Cleveland Clinic, Cleveland, OH. Amnesia for the episodes occurred in 8 of the 9 patients. Other ictal phenomena prior to the vomiting included staring, automatisms, eye-blinking, grimacing, eye-rolling, chewing, and swallowing. After vomiting, only one patient regained normal alertness immediately. Four patients had temporal lobectomy, the pathology specimens showing mild inflammatory changes, gliosis, and neuronal heterotopia. (Kramer RE et al. Ictus emeticus: an electroclinical analysis. Neurology, July 1988;38:1048-1052).

COMMENT. The authors note that reports of ictal vomiting recorded electrographically are scarce and that 13 of 14 cases published have an onset or lateralization of EEG findings to the right hemisphere. In one case the vomiting was induced by photic stimulation. Another case may be added to this list from the French literature (Giroud M et al. Un symptome critique epileptique rare: le vomissement. Arch Fr Pediatr 1987;44: 231-4). In this 9 year old boy, the paroxysmal episode of vomiting was synchronized with an epileptiform discharge in the left fronto-temoral area on the EEG. That cyclic vomiting may represent a form of epilepsy in children was proposed in a report of 33 patients, 7 (21%) having a history of grand mal or complex partial seizures in addition, and 25 (76%) with seizure discharges in the EEG, some focal and predominantly in the temporal lobe. (Millichap JG, Lombroso CT, Lennox WG. Pediatrics 1955;15:705). The EEG was not recorded at the time of the vomiting and, in retrospect, some of our cases may have been more correctly classified as migraine.

SEIZURE RECURRENCE AFTER MEDICATION WITHDRAWAL

The relapse rate after withdrawal of antiepileptic medication was investigated in 146 children with epilepsy seen at the Dept of Child Neurology, University Hospital, Rotterdam, and Research Unit for Clinical Neurophysiology, Westeinde Hospital, The Hague, The Netherlands. The cumulative probability of remaining seizure-free after a 2 year period of control and normalization of the EEG was 75%. Three-quarters of relapses occurred during the withdrawal period and in the following 2 years. A significantly higher relapse rate was present in girls and with seizures of known etiology. In patients with partial epilepsy, recurrence may be predicted by the presence of focal neurological signs and/or mental retardation, female sex, a positive family history for epilepsy, and polytherapy. In those with primary generalized epilepsy, no predictive factor was uncovered. The recurrence rate did not change between groups of children who were treated for 2, 3, 4, or 5 years before withdrawal was attempted and EEG epileptiform abnormalities had disappeared.

(Arts WFM et al. Follow-up of 146 children with epilepsy after withdrawal of antiepileptic therapy. Epilepsia May/June 1988;29:244-250).

COMMENT. A relapse rate of 25% in this study is similar to that reported previously, and the relatively short treatment period did not appear to increase the risk of recurrence compared to studies requiring a minimum 4 year seizure-free period before discontinuing medicines. However, the absence of EEG epileptiform discharges at the time of drug withdrawal did not improve the outcome by reducing relapse rate compared to studies in which the EEG was not used in decision to withdraw. The results of this investigation emphasize the dangers of anti-epileptic drug withdrawal in mentally retarded female patients with neurological deficits.

READING EPILEPSY

Precipitating stimuli, including eye-movements, reading aloud versus reading silently, linguistic complexity, and concentration, were investigated in a 24 year old young woman with reading epilepsy seen in the Division of Neurology and Clinical Neurophysiology Laboratory, University of Ottawa General Hospital, and Depts of Psychology and Computer Engineering, Carleton University, Ottawa, Ontario, Canada. A viral illness at 19 years of age was followed by episodic jerking of the jaw while reading silently and a single generalized tonic-clonic seizure. After 2 years freedom from symptoms, the jaw jerking returned while reading, and was aggravated by fatigue or stress. Reading material was presented on a microcomputer video display monitor while the patient underwent EEG radiotelemetry video monitoring. Seizures were most readily elicited by reading aloud material of medium or high linguistic complexity. (Christie S et al. Primary reading epilepsy: Investigation of critical-provoking stimuli. Epilepsia May/June 1988;29:288-293).

COMMENT. Bickford et al., in their original description of reading epilepsy, observed precipitation of seizures by difficult reading matter (Trans Am Neurol Assoc 1956;81:100), and other authors have stressed the cortical or "lexical" aspects of the reading process in seizure provocation. The results of the present study suggest that a combination of factors involved in reading, including saccadic eye movements, articulation, and difficulty of linguistic content, contribute to epileptogenesis. Seizures were more readily provoked when greater demand was placed on each of the subtasks collectively and no factor acted solely as the critical stimulus.

In an article in the same issue of Epilepsia from the National Centre for Children with Epilepsy, Park Hospital for Children, Headington, Oxford, England, Verduyn et al surveyed mothers' impressions of seizure precipitants in children with epilepsy. Reading was not included in the list of precipitants, but stimuli that are somatic, psychological, and sensory were frequently followed by seizures. Mothers reported a relaxed state, emotional state, anxiety, exercise, and drowsiness as precipitants in 60-75% of the sample of 446 children. Sensory-evoked epilepsy in 2.5% was provoked by loud noise, startle, flashing lights, bright light, diet, touch, television, and pattern. (Epilepsia 1988;29:251).