

formation of concretions and continued absorption of the drug. Prolonged repeated use of activated charcoal after carbamazepine overdose in comatose patients is not beneficial and carries a hazard of aspiration.

VALPROATE-INDUCED CEREBRAL EDEMA

A case of cerebral edema resulting from acute sodium valproate poisoning in a 19 year old male is reported from Northwick Park and East Birmingham Hospitals, UK. On admission the patient was unconscious and the plasma valproate concentration was 900 mg/L. He had taken 1200 mg of sodium valproate along with 1500 mg of aspirin. The salicylate blood level was 70 mg/L. CT showed gross cerebral edema with slit-like ventricles and absence of cortical sulci and basal cisterns. Gastric lavage, fluid restriction and IV dexamethasone resulted in slow recovery. Acute complications included liver and renal dysfunction, hypocalcaemia and generalized muscle spasms. (Khoo SH, Leyland MJ. Cerebral edema following acute sodium valproate overdose. Clin Toxicol June 1992; **30**:209-214.) (Reprints: Dr. S.H. Khoo, Monsall Hospital, Newton Heath, Manchester, M108WR, UK.)

COMMENT. External leakage from feeding gastrostomies was reported in 4 of 8 children who received valproate sprinkle at the Hennepin County Medical Center, Minneapolis, MN (Jones-Saete C et al. Epilepsia July/Aug 1992; **33**:692-695). Adherence of undissolved valproate particles to the exterior of the tube appeared to prevent the close approximation of the tube to the gastrostomies stoma. The problem was reduced by changing the tube more frequently or using a larger size tube.

SEIZURE DISORDERS

PYRIDOXINE DEPENDENT EPILEPSY

Four children with pyridoxine dependent seizures beginning at 2 to 19 months are reported from the Loyola University Medical Center, Maywood, IL. Patients were identified out of 51 treated routinely with pyridoxine for refractory seizures and seen over a 6 year period. The dose of pyridoxine was 50 mg orally twice daily. The seizure types were atypical absence, myoclonic, generalized clonic and simple partial and complex partial. The authors suggest that pyridoxine should be tried in all children with seizure disorders with onset at any age who are poorly responsive to anticonvulsant drugs. (Coker SB, Postneonatal vitamin B₆ - dependent epilepsy. Pediatrics Aug 1992; **90**:221-223.) (Reprints: S.B. Coker, M.D., Loyola University Medical Center, 2160 S. First Ave., Maywood, IL 60153.)

COMMENT. Pyridoxine dependent epilepsy may present after the neonatal period and may be manifested by many seizure types. Many of us have been discouraged by failure to uncover pyridoxine dependency in drug resistant epilepsies in children. Obviously, persistence rewards, but the relationship between pyridoxine and epilepsy is complex.

The wide range of reactivity of vitamin B₆ with a number of drugs and other chemicals encountered in the environment must be considered in the diagnosis. These interactions involve a condensation between the xenobiotic and pyridoxal phosphate, and vitamin B₆ absorption, coenzymes, and specific vitamin B₆-dependent enzymes can also be affected. Isoniazide is probably the most extensively studied drug with respect to effects on vitamin B₆ metabolism. Other drugs reported to interact with vitamin B₆ include ampicillin, anticonvulsants, levodopa, oral contraceptives and ethanol. The mechanism of anticonvulsant drug and vitamin B₆ interaction is not well determined. One study showed that the vitamin was decreased only in males treated with anticonvulsants. Another showed that pyridoxine supplements resulted in lower plasma phenytoin and phenobarbital concentrations. An interaction between the vitamin and dyes added to medications during manufacture has also been demonstrated (Dubick MA. Interactions of vitamin B₆ (pyridoxine) and xenobiotics. In Nutritional Toxicology, Vol III, ed. JN Hathcock. New York, Academic Press, 1989.)

DISCONTINUING MEDICATION: RECURRENCE RISK FACTORS

The results of antiepileptic drug discontinuation in epileptic children who were seizure free for at least 2 years are reported from the Services of Child Neurology and Psychology, Department of Neurology, Clinics Hospital University of Sao Paulo Medical School, Sao Paulo, Brazil. Twenty children (28%) had a recurrence, 75% during discontinuation or less than 6 months after discontinuation of AED. The risk factors related to seizure recurrence were 1) more than 10 seizures before seizure control, 2) abnormal EEG in the year before discontinuation, 3) focal neurologic signs and/or mental retardation, and 4) a mixed seizure pattern. Two or more risk factors were associated with recurrence of seizures in 14 children (70%), whereas patients with none or only 1 risk factor had no recurrence (72%). (Gherpelli JLD et al. Discontinuing medication in epileptic children: a study of risk factors related to recurrence. Epilepsia July/Aug 1992; 33:681-686.) (Reprints: Dr. J.L.D. Gherpelli, Service of Child Neurology, Department of Neurology, Hospital das Clinicas da F.M.U.S.P., Sao Paulo 01000, P.O. Box 8091, Brazil.)

COMMENT. Risk factors may be of help in determining the safety of discontinuing antiepileptic medications in children who are seizure free for a period of at least 2 years. There was no correlation between age at seizure onset and recurrence in this study, which is at variance with some other reports.

A finding of a higher sleep tendency in children with epilepsy than in controls, even 4-5 months after drug discontinuation, was unexpected in a study from the University of Lund, Sweden (Palm L et al. Epilepsia July/Aug 1992; 33:687-691). A long time had passed since the last reported seizure and the EEG was free of epileptiform activity in most cases, so that the sleepiness could not be a direct effect of epileptic seizures.