may reverse both the EEG and cognitive, language, and behavioral regression. The well known tendency to development of tolerance with use of benzodiazepines was considered unusual in the treatment of ESES in this study. Seizures and the ESES may be exacerbated by carbamazepine.

VALPROATE-INDUCED WEIGHT GAIN

To determine the relation of carnitine and insulin levels to weight gain during valproate therapy of epilepsy in children, twenty patients were randomly assigned to receive either carnitine or placebo supplementation, in a study at Hacettepe University, Ankara, Turkey. The mean age was 8.3 and 9.4 years in the two groups. Seizures were completely controlled. Weight gain occurred in both groups after a 3 month period of observation. Weight gain did not correlate with carnitine levels. Mean insulin levels and insulin/glucose ratios were increased during valproate therapy. Weight gain might be related to the decrease in glucose levels and consequent appetite stimulation. (Demir E, Aysun S. Weight gain associated with valproate in childhood. Pediatr Neurol May 2000;22:361-364). (Respond: Dr Sabiha Aysun, Department of Pediatric Neurology, Hacettepe University, Ankara 06100, Turkey).

COMMENT. Weight gain associated with valproate therapy for epilepsy is not related to carnitine deficiency. It may be caused by appetite stimulation, secondary to higher insulin and lower blood glucose levels. VPA may decrease glucose levels by inhibiting gluconeogenesis, and by other possible mechanisms.

TOXIC-METABOLIC DISORDERS

PRENATAL ALCOHOL AND NEUROLOGIC DYSFUNCTION

Of 698 pregnant women interviewed about alcohol consumption at the maternity hospital in Roubaix, France, 156 of the offspring were investigated at age 4 and 1/2 years, using a standardized neuropsychological assessment. A posture score was not related to alcohol consumption, whereas a lower general cognitive index (GCI) and a minor neurological sign score were directly related to consumption of 21 drinks/week (3 drinks/day). The minor neurogic signs found at the neuromotor evaluation included synkinesis or mirror movements, finger-nose incoordination, hopping dyspraxia, and impaired ability to walk on heels. The high number of neurologic abnormalities was associated with prenatal alcohol consumption, after controlling for a lower GCI. (Larroque B, Kaminski M, Dehaene P, Subtil D, Querleu D. Prenatal alcohol exposure and signs of minor neurological dysfunction at preschool age. Dev Med Child Neurol August 2000;42:508-514). (Respond: Beatrice Larroque MD PhD, Institut National de la Sante et de la Recherche Medical Unit 149, 16 Avenue Paul Vaillant, Couturier, 94807 Villejuif Cedex, France).

COMMENT. Children born to mothers who consumed 21 or more alcoholic drinks per week (11% of the sample) had more minor neurologic abnormalities than those exposed to less alcohol. Levels of alcohol consumption lower than those associated with fetal alcohol syndrome can cause impairments of neurologic function recognized on clinical examination.

Twelve-year follow-up of children exposed to alcohol in utero, at the University of Helsinki, found that the longer the exposure and the more severe the fetal alcohol syndrome, the more often the children required special

education and were behaviorally impaired. (Autti-Ramo I. Dev Med Child Neurol June 2000;42:406-411). Follow-up should be focussed not only on the child's cognitive and neuromotor development but on parental and foster family support and counseling and the prevention of secondary behavioral problems.

AMPHETAMINE ABUSE AND INTRACRANIAL HEMORRHAGE

Eight young adults presenting with intracranial hemorrhage at the Department of Neurosurgery, Queen's Medical Center and University Hospital, Nottingham, UK, in a 3 and 1/2 year period, were diagnosed with amphetamine abuse. The time from amphetamine exposure to onset of symptoms ranged from 10 minutes to 2 months (median within 24 hours). CT and cerebral digital subtraction angiography (DSA) showed a parenchymal hematoma in 7 (3 in the frontal lobe), and a subarachnoid hemorrhage in 1. Beading of small and medium-sized arteries, characteristic of vasculitis, also occurred in 1. Four recovered, but 1 died and 3 were hemiplegic.

Of 37 previously reported cases, from 1945 to 1996, 6 died and 17 had hemiparesis. One-third claimed to be infrequent amphetamine users. The first reported death from amphetamine abuse (JAMA 1939) was in a 25 year-old man who had taken the drug as a stimulant before a college examination. Amphetamine use or abuse should be considered in diagnosis of a frontal or parietal lobe hematoma in a young patient. (Buxton N, McConachie NS. Amphetamine abuse and intracranial hemorrhage. J R Soc Med September 2000;93:472-477). (Respond: Mr Neil Buxton FRCS (Ed), Department of Neurosurgery, University Hospital, Queen's Medical Centre, Nottingham NG7 2UH, UK).

COMMENT. Amphetamines taken orally, IV, or rarely, inhaled, have resulted in cerebral vasculitis and intracranial hemorrhage. Unfortunately, the dosages involved are unknown. The outcome is poor in more than half the cases reported. Vasculitis, that can follow only a single exposure to oral amphetamines, is characterized by irregular segmental narrowing or beading of small cerebral arteries.

Perhaps the recent increased popularity of amphetamine drugs in the treatment of ADHD in school-age children should be reappraised.

CELIAC ANTIBODIES AND NEUROLOGIC DISORDERS

The yield of screening for celiac disease in children with common neurologic disorders was evaluated at Tel Aviv University, Zerifin, Israel. Of 167 patients presenting, ages 1 to 16 years, 41 had migraine headaches, 39 ADHD, 36 epilepsy, and 51 hypotonia and motor abnormalities. IgG antigliadin antibodies were positive in 22 (13%) of patients compared to 3 (9%) in the control group. IgA and endomysial antibodies were negative in all patients, and duodenal biopsies were not performed. Routine screening for celiac disease is not recommended in children presenting wsith common neurologic disorders. (Lahat E, Broide E, Leshem M, Evans S, Scapa E. Prevalence of celiac antibodies in children with neurologic disorders. Pediatr Neurol May 2000;22:393-396). (Respond: Dr Eli Lahat, Unit of Pediatric Neurology, Assaf Harofeh Medical Center, Zerifin 70300, Israel).

COMMON. Neurologic complications of celiac disease include peripheral neuropathy, myopathy, cerebellar ataxia, myoclonus, cerebral atrophy, cerebral vasculitis, encephalitis, epilepsy, sometimes associated with cerebral calcification. Studies in adults have shown a significant increase in positive titers for antigliadin antibodies in patients (57%) with common neurologic disorders of undetermined etiology. In children, the association of celiac disease with common