lymphadenopathy in 63%, and cardiomyopathy in 30%. Failure to gain weight predated the onset of encephalopathy in infected infants. HIV infected children without encephalopathy had a lower incidence of hepatosplenomegaly (29%) and cardiomyopathy (2%), diagnosed in the first 3 months of life. Risk of encephalopathy was related to a high viral load in infancy. (Cooper ER, Hanson C, Diaz C et al. Encephalopathy and progression of human immunodeficiency virus disease in a cohort of children with perinatally acquired human immunodeficiency virus infection. J Pediatr May 1998;132:808-812). (Reprints: Ellen R Cooper MD, Finland Laboratory, Boston Medical Center, 774 Albany St, Boston, MA 02118).

COMMENT. Encephalopathy is a frequent complication of perinatally acquired HIV infection, occurring in one in 5 infants. A high viral load during the neonatal period, failure to thrive, and early signs of organomegaly and lymphadenopathy are risk factors for HIV encephalopathy.

NEUROLOGIC COMPLICATIONS OF E. COLI H-UREMIC SYNDROME

The association between bacterial genotype of E coli O157:H7 and CNS manifestations of childhood gastroenteritis-associated hemolytic uremic syndrome (D+HUS) was studied in 51 patients with HUS treated at the British Columbia's Children's Hospital, Vancouver, Canada. Of 51 children with HUS, 11 suffered neurologic complications that included encephalopathy in 6 and seizures in 7, with 2 deaths. No association with bacterial genotype was demonstrated. (Cimolai N, Carter JE. Bacterial genotype and neurological complications of *Escherichia coli* O157:H7-associated haemolytic uraemic syndrome. Acta Paediatr May 1998;87:593-594). (Respond: Dr N Cimolae, Room 2G6, Department of Pathology and Laboratory Medicine, British Columbia's Children's Hospital, 4480 Oak Street, Vancouver, British Columbia, Canada).

COMMENT: Seizures, encephalopathy, visual disturbances, and transient hemiparesis are the neurologic complications of E coli hemolytic uremic syndrome. Seizures, the most common symptom, occur from 4 to 12 days after onset of diarrhea. A number of factors may cause the CNS symptoms, but a specific bacterial genotype of E coli is not implicated. Prevention or timely diagnosis and treatment may diminish the incidence of neurologic complications of E coli O157:H7 infection.

Risk of hemolytic uremic syndrome after sporadic E coli O157:H7 infection: Results of a Canadian collaborative study. (Rowe PC et al. J Pediatr May 1998;132:777-782). Of 205 children with HUS, 77% had E coli O157:H7 infection. A further 582 had E coli gastroenteritis. The risk of HUS in Alberta was 8.1%, compared to 31% in other Canadian referral centers.

SEIZURE DISORDERS

COGNITIVE AND BEHAVIORAL OUTCOMES OF FEBRILE SEIZURES

Of approximately 14,000 children enrolled in a British Child Health and Education Study, 398 identified with febrile convulsions (FC) were assessed at age 10 years at Addenbrooke's Hospital, Cambridge, and the University of Bristol, UK. Measures of academic progress, intelligence, and behavior in the FC patients were not significantly different from controls without FC. Patients with simple FC (287) and complex FC (94) showed similar results. The outcomes in those with recurrent FC and those with a single seizure were similar. Children with FCs in the first year of life required special schooling more often than those with late-onset FCs (7.5%

cf 1.5%). (Verity CM, Greenwood R, Golding J. Long-term intellectual and behavioral outcomes of children with febrile convulsions. N Engl J Med June 11, 1998;338:1723-1728). (Reprints: Dr Verity, Child Development Centre, Box 107, Addenbrooke's Hospital, Cambridge CB2 2QQ, UK).

COMMENT. Febrile convulsions do not impair academic progress, intellect, and behavior in patients followed and tested at 10 years of age. Children with complex febrile seizures have the same cognitive and behavioral outcome as those with simple febrile seizures. These UK results are similar to those of the prospective US National Collaborative Perinatal Project that found similar IQ levels in FC and seizure-free siblings at 7 year follow-up. They differ from studies showing associations between FCs and attention deficit hyperactivity disorders and learning disabilities.

ATYPICAL BENIGN EPILEPSY WITH CENTROTEMPORAL SPIKES

Six children treated with carbamazepine or valproate for benign epilepsy with centrotemporal spikes (BECT), and followed at the Hospital de Cruces, Vizcaya, Spain, had an atypical and deteriorating course during therapy and a rapid remission when antiepileptic drugs were discontinued. Patient 1 had a first nocturnal, partial oromotor seizure at age 6 years, The father had BECT in childhood. EEG showed the typical bilateral midtemporal spikes. After 2 weeks of carbamazepine therapy, seizures recurred several times a day and the EEG abnormality became generalized, with continuous spike-and-wave during slow wave sleep (CSWS). Clinically, he developed a receptive language disorder, dysarthria, drooling, and myoclonic jerks of perioral muscles. Carbamazepine was discontinued and phenytoin and clobazam substituted. Within one week, the language disorder and seizures subsided, and subsequent EEGs were either normal or showed only occasional midtemporal spikes. AEDs were discontinued at 10 years of age without seizure recurrence, and school performance was normal at age 15 year follow-up. Patient 2 developed Landau-Kleffner syndrome and behavior disturbance during treatment with CBZ, with recovery after drug withdrawal. Patient 3 suffered seizure recurrences and language disorder with CBZ, and recovered when primidone was substituted. Patient 4 had severe language deterioration, seizures and CSWS on CBZ, and normalized when clobazam was substituted. Patient 5 had seizure recurrences and CSWS during valproate therapy, worsening with increased dosage, and recovery with drug withdrawal. Patient 6 developed dementia and CSWS after 6 months therapy with CBZ and VPA, and recovered when clobazam was substituted. A combination of AED effects and the syndrome itself could be responsible for the reversible epileptic and neuropsychological deterioration observed in these cases. (Prats JM, Garaizar C, Psy MLG-N, Madoz P. Antiepileptic drugs and atypical evolution of idiopathic partial epilepsy. Pediatr Neurol May 1998;18:402-406). (Respond: Dr Jose M Prats, Division of Pediatric Neurology, Department of Pediatrics, Hospital de Cruces, 48903 Baracaldo, Vizcaya, Spain).

COMMENT. Carbamazepine-induced seizure exacerbation and valproate-induced dementia in children with BECT have been reported previously. The AED-associated worsening of symptoms and course of this epileptic syndrome is correlated with a diffuse spread of the EEG sleep abnormality, termed "continuous spike-and-wave during slow wave sleep" (CSWS). The above report emphasizes the risks involved in treating benign idiopathic partial epilepsies with carbamazepine or sodium valproate. The possible neuropsychological deterioration, with severe language disorders, including Landau-Kleffner