the risk of a second seizure but does not alter the prognosis for long-term seizure remission. The majority of patients will have few or no recurrences, and only 10% have more than 10 recurrences regardless of therapy. Cognitive and behavioral side effects of AEDs may occur, particularly with phenobarbital. Treatment is not indicated for the purpose of preventing epilepsy; it may be considered when the benefits of reducing the risk of a second seizure outweigh the risks of educational and psychosocial side effects. Decision to treat should be individualized and based on patient and family preferences as well as medical issues. Future research should focus on prevention and cure of the underlying disorder. (Hirtz D, Berg A, Bettis D et al. Practice parameter: treatment of the child with a first unprovoked seizure. Neurology January (2 of 2) 2003;60:166-175). (Reprints: QSS, American Academy of Neurology, 1080 Montreal Ave, St Paul, MN 55116).

COMMENT. The early identification of patients likely to develop epilepsy after a first seizure, the underlying causes for the poor prognosis, and the development of more specific treatments without side effects are the aims of future epilepsy research. Virological and immunological factors have been invoked in some epilepsies, notably Rasmussen’s encephalitis.

PARTIAL SEIZURES AS AN EARLY SYMPTOM OF RASMUSSEN’S ENCEPHALITIS

Early manifestations of Rasmussen’s encephalitis (RE) were studied in 12 patients with clinical and neuropathological diagnosis followed from disease onset by members of a study group in Milan and other centers in Italy. Disease onset was marked by partial seizures in 11 patients (epilepsia partialis continua (EPC) in 1) and by hemiparesis and partial status epilepticus in 1. EPC developed in 10 patients 3 weeks to 31 months after onset of isolated partial seizures, and focal motor deficits developed in all patients 15 days to 24 months (mean 6.8 +/- 6.8 months) after the first seizure. Age at onset ranged from 14 months to 11 years (mean 5.2 +/- 2.8 years). Five had minor viral infection in the 6 months before onset. Signs of cognitive impairment, memory, attention and learning deficits, occurred 4 to 36 months after onset (mean 11.1 +/- 9.4 months). Initial EEG abnormalities included delta waves over the affected hemisphere, mainly central and temporal, in all patients. Epileptiform discharges were present with the delta activity in first recordings in 9, and appeared after 2 to 6 months in 3. Initial MRIs showed focal cortical atrophy involving the insular cortex and extending to frontal, temporal and parietal areas. The caudate head was atrophied in 4 early studies and in 9 at follow-up. White matter hyperintensity occurred beneath the cortical atrophy. Anti-GluR3 A and B antibodies were detected in 4 of 7 patients tested before surgery, and csf oligoclonal bands were present in 4 of 6 tested. Other laboratory tests were negative, including serum antibodies for EBV, CMV, and HSV. Epilepsy was refractory to medication within a few months of onset. Status epilepticus recurred several times in all but 1 patient. All developed severe hemiparesis. Serial EEGs showed progressive flattening of background activity and persistent multifocal slow epileptiform abnormalities over the affected hemisphere. Significant but transient improvement was obtained medically only with high-dose steroids and selective immunoabsorption. All patients were treated surgically (7 months to 14 years...
after disease onset). EPC and partial seizures ceased post-surgically in all patients; they recurred within 6 months in 2. (Granata T, Gobbi G, Spreafico R et al. Rasmussen’s encephalitis. Early characteristics allow diagnosis. Neurology February (1 of 2) 2003;60:422-425). (Reprints: Dr Tiziana Granata, Division of Neuropsychiatria Infantile, Instituto Nazionale Neurologico C Besta, Via Celoria 11, 20133 Milan, Italy).

COMMENT. A tentative diagnosis of Rasmussen’s encephalitis is possible within 4 to 6 months after onset of partial seizures, especially when epilepsy partialis continua occurs at presentation. The focal EEG findings and MRI changes are usually characteristic. Proposed mechanisms are virus-induced inflammation and an immune-mediated process. The use of antiviral and immunosuppressive therapies in the early stages of the disease may be more effective in slowing progression.

Virological and immunological aspects of seizure disorders are reviewed by Eeg-Olofsson O, Uppsala University, Sweden (Brain Dev Jan 2003;25:9-13). The etiology of Rasmussen’s encephalitis (RE) is thought to be a viral infection coupled with immunodysfunction. HSV-1 DNA has been recovered from the cortex of 3 children with RE, and CMV DNA in brain biopsies in 2 reports. Significantly decreased IgA levels have been reported in 7 patients with RE. Glutamate receptor type 3 (GluR3) antibodies in serum of RE patients, documented in the above study, have been reported previously.

HHV-6 and -7, the causative agents for exanthem subitum, are linked to febrile seizures, but the association is debated. Recurrences of febrile seizures may be associated with reactivation of HHV-6 that has invaded the brain during the acute phase of exanthem subitum.

Defects in human leukocyte antigens (HLA) A1,B8, T4 and T8 lymphocytes, and immunoglobulins in relation to various epilepsies suggest a genetic predisposition to virus persistence that results in neuronal membrane defect and seizures. Virology and immunologic research and trials of antiviral agents may be a promising approach to the cause and treatment of epilepsy in the future.

RECURRENT SEIZURES AND BEHAVIOR PROBLEMS

The association of seizures and behavior problems in children with new onset seizures was investigated in a prospective study of 224 children (aged 4-14 years) and 159 siblings (4-18 years) at Indiana University and Children’s Hospital Medical Center, Cincinnati, OH. Caregiver’s ratings of the behavior, based on the Child Behavior Checklist (CBCL) administered by telephone, were collected at baseline, and at 6 months, 12, and 24 months. During the 2-year period of follow-up, 163 (73%) children had at least one seizure recurrence, and 61 (27%) had none. In children with recurrent seizures, CBCL Total and Internalizing Behavior Problem scores on average were higher during seizure recurrence periods than when not experiencing recurrent seizures (p=0.041). Siblings had significantly lower Total and Internalizing Problem scores compared to patients either experiencing or not experiencing recurrent seizures. Externalizing Problems scores were not significantly different among children with or without recurring seizures, and siblings. Demographics and antiepileptic medication effects were controlled in these analyses. Both seizures and behavior problems may be caused by the underlying neurological disorder,