

percentage of children in this large, retrospective study. Neuroimaging should be considered for children who meet high-risk criteria. Those without predisposing conditions or focal seizures, at low risk for abnormal imaging, may be discharged from the ED without neuroimaging, provided that the neurologic exam and mental status are normal and follow-up is assured. (Sharma S, Riviello JJ, Harper MB, Baskin MN. The role of emergent neuroimaging in children with new-onset afebrile seizures. *Pediatrics* January 2003;111:1-5). (Respond: Sujit Sharma MD, Department of Emergency Medicine, Children's Healthcare of Atlanta at Scottish Rite, 1001 Johnson Ferry Rd, Atlanta, GA 30342).

COMMENT. An editorial commentary affirms that less testing is needed in the emergency room after a first afebrile seizure, that the evaluation of a child is often erroneously extrapolated from the adult in whom tumors and vascular events are more common causes of first seizures, and the cost of routine tests, especially neuroimaging, outweighs the benefit. A careful history and physical examination would have identified the majority of patients in the high risk category found to have clinically significant neuroimaging abnormalities. Unless there are special circumstances, blood work, lumbar puncture, EEG, and neuroimaging are not needed in the ED evaluation of a first afebrile seizure. A thorough discussion with the parents and older patients concerning the nature and cause of a seizure, precautions, and prognosis is the most important aspect of management of the seizure. (Freeman JM. *Pediatrics* 2003;111:194-196).

The Quality Standards Subcommittee of the American Academy of Neurology, Child Neurology Society, and American Epilepsy Society (*Neurology* 2000;55:616-623), after reviewing published literature, also advise against routine lumbar puncture, laboratory studies, and routine neuroimaging after a first unprovoked nonfebrile seizure. Emergency imaging should be reserved for patients with new-onset neurologic deficit or persisting changes in mental status. MRI may be indicated on a nonemergency basis for diagnosis of specific epilepsy syndromes. Contrary to the opinion proposed in the editorial commentary, the Quality Standards Subcommittee recommends an EEG after a first nonfebrile seizure. An EEG is useful in predicting risk of seizure recurrence, differentiation and diagnosis of the paroxysmal event, diagnosis of epileptic syndromes, and prognosis, and may influence the need for subsequent neuroimaging. It may not influence the decision to treat. Treatment with anti-epileptic drugs (AED) should be individualized and is not recommended routinely. It may decrease the risk of a second seizure, but not the long-term risk of subsequent epilepsy. In most cases, AEDs are not recommended after a first afebrile seizure. In a recently published practice parameter report of the Quality Standards Subcommittee, treatment with AED may be considered when the benefits of reducing the risk of a second seizure outweigh the risks of AED side effects (*Neurology* Jan 2003;60:166-175).

TREATMENT OF A FIRST UNPROVOKED SEIZURE

The Quality Standards Subcommittee of the AAN and Practice Committee of the CNS have reviewed published literature to determine when to begin treatment after a first unprovoked seizure, risk of recurrence, prevention of recurrence, development of chronic epilepsy, and side effects of AEDs. Treatment after a first unprovoked seizure decreases

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 +/- 9.4months). 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 immunoadsorption. All patients were treated surgically (7 months to 14 years