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SEIZURE DISORDERS

STATUS EPILEPTICUS

The mortality and incidence of sequelae of status epilepticus of varying causes was studied in 193 children, ages 1 month to 18 years (mean, 5 years), followed for a mean period of 13.2 months in the Division of Pediatric Neurology, Montefiore Medical Center, 111 E. 210th St, Bronx, NY. The cause of the status epilepticus was idiopathic in 46 cases, symptomatic of a previous CNS insult (stroke, head trauma, meningitis, or static encephalopathy) in 45 cases, febrile in 46, acute symptomatic with neurologic insult or systemic metabolic dysfunction in 45, and progressive neurologic with neurodegenerative, malignant, and neurocutaneous syndromes in 11. The mortality and incidence of sequelae following status epilepticus was low and primarily a function of etiology. Seven children (3.6%) died within three months of the episode of status epilepticus. All seven deaths occurred among the 56 children with status epilepticus associated with an acute CNS insult or progressive encephalopathy. The mortality in this group was 12.5%. Only two of the 137 children with unprovoked (idiopathic), remote symptomatic, or febrile status epilepticus sustained neurologic deficit attributable to the status epilepticus. None of the 67 children in the unprovoked or febrile groups studied prospectively had any residual motor or cognitive disability. The difference in the rate of neurologic sequelae between different causes was highly significant ($P < .001$). A total of 17 (9.1%) children sustained new motor or cognitive deficits following status epilepticus. The incidence of significant sequelae was a function of age. It declined from 29% among infants less than 1 year of age to 11% in children between 1-3 years of age and 6% for children older than 3 years of age ($P < .001$). The majority of the patients with sequelae were in the acute symptomatic or progressive encephalopathy groups and these consisted of extremely young patients. Within each etiologic group, age did not significantly affect the outcome. The incidence of severe sequelae in the younger age group was related to the more frequent occurrence of acute symptomatic status epilepticus and progressive encephalopathy in that age group. The risk of unprovoked seizures following status epilepticus in 125 surviving children with no history of

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prior seizures was 30%. The authors concluded that the morbidity of aggressively treated status epilepticus in children in the absence of an acute neurologic insult or progressive neurologic disorder was low. (Maytal J et al. Low morbidity and mortality of status epilepticus in children. Pediatrics March 1989; 83:323-331).

COMMENT. Dr. John M. Freeman of Johns Hopkins Hospital, Baltimore, commented that status epilepticus is "not what we've thought or taught." He asks the question: "Does the morbidity of the treatment of seizures in the emergency room to prevent status epilepticus now exceed the morbidity of the status epilepticus itself? He states that "just as it is not necessary to administer long term anticonvulsant medication to a child after a first seizure, it seems also not necessary to initiate long term therapy when a child's first seizure is status epilepticus."

Many will not agree with this advice, however. An incidence of 30% of unprovoked seizures in children surviving status epilepticus is sufficiently high to warrant long term preventive anticonvulsant therapy. Furthermore, status epilepticus if not treated aggressively is a serious and potentially fatal complication of convulsive disorders. The conclusions to this study and commentary should not permit a diminished respect for the hazards of status epilepticus nor change the accepted methods of treatment with maximally tolerated intravenously administered anticonvulsant therapy. The use of rectal anticonvulsant therapy (see Ped. Neur. Briefs Jan 1988; 2:7) by parents in selected patients may prevent prolongation of seizure recurrences and avoid the necessity for toxic levels of anticonvulsant drugs for refractory cases.

TREATMENT OF NEONATAL SEIZURES

The rapid sequential phenobarbital treatment of neonatal seizures was examined in 120 newborns and the efficacy of high dose monotherapy was compared with the addition of a second anticonvulsant for persistent seizure activity. Patients were examined in three participating neonatal intensive care units: Comprehensive Epilepsy Center, Pharmacokinetics Laboratory, Miami; Greensboro Area Health Education Center; and Department of Neonatal Medicine, Moses H. Cone Memorial Hospital, Greensboro, North Carolina. A single loading dose of phenobarbital 15-20 mg/kg was administered initially and nonresponders received sequential bolus doses of 5-10 mg/kg until seizures ceased or a serum concentration of 40 mg/mL was obtained. Infants with refractory seizures received additional phenobarbital to a maximum serum concentration of 100 mg/mL. The majority of neonates with recurrent seizure activity (77%) responded to phenobarbital monotherapy administered in a rapid sequential dosing schedule that achieved a serum concentration of 40 mg/mL. In 40%, seizures were controlled with a single 15-20 mg/kg initial loading dose and a serum concentration in the range of 10-30 mg/mL. Of 28 subjects refractory to phenobarbital, 13 (46%) were controlled by a second anticonvulsant (phenytoin or lorazepam) and four were controlled by three or more agents. Eleven were resistant to medication and ten died. There was no significant difference in drug responsiveness among patients with different seizure patterns and seizure etiology was not a significant determinant of