selective amnestic effect on episodic memory in children without significantly impairing long-term memory or attention.

Lorazepam has become increasingly popular for the treatment of status epilepticus in children; the usual median dose is 0.1 mg/kg. Midazolam, a benzodiazepine used primarily for induction of anesthesia, in contrast to lorazepam and diazepam, is water soluble and its injection is neither painful nor irritating by the intramuscular route. Midazolam 15 mg IM was as effective in abolishing spikes as 20 mg of diazepam IV five minutes after administration in a study in adults with epilepsy. (Jawad S et al. J Neurol Neurosurg Psychiat 1986; 49:1050). The half life of lorazepam is longer than that of diazepam or midazolam, however, and its duration of action is more prolonged.

CARBAMAZEPINE THERAPY AND LONG-TERM PROGNOSIS OF EPILEPTIC CHILDREN

The long term prognosis in 90 children with partial or generalized tonic-clonic seizures treated with CBZ has been evaluated in the Department of Pediatrics, Kyoto University, Kyoto, Japan. Sixty-seven (74%) treated with CBZ monotherapy were seizure free for more than three years. Fifty (56%) had no epileptiform discharge on the follow-up EEG. Patients with mental retardation and a genetic predisposition were more likely to have an abnormal EEG. The incidence of mental retardation was significantly higher in those treated with polytherapy. The prognosis of patients with partial seizures secondarily generalized was less favorable than that of the other patients. Patients without mental retardation more often received CBZ monotherapy and patients with seizures of undetermined etiology more often received polytherapy. The lowest blood level of CBZ for maintenance was 4 meg/ml and maximum blood levels ranged from 6-12 meg/ml. Side effects were observed in 20 patients who had drowsiness, 4 ataxia, 2 a rash and 1 had anorexia. The SGOT, SGPT, or both were elevated in 16 patients. Leukopenia between 2,000 and 4,000 occurred in 32 patients. (Okuno T et al. Carbamazepine therapy and long-term prognosis in epilepsy of childhood. Epilepsia Jan/Feb 1989; 30:57-61).

COMMENT. There was no correlation between the type of seizure and the prognosis of the patients in this study. All patients with simple partial seizures and benign epilepsy of children with centrottemporal foci were seizure free for more than one year and the majority were seizure free for more than three years. There was no correlation between a history of febrile convulsions and the prognosis of children with partial or generalized tonic-clonic seizures. Patients with partial seizures secondarily generalized had a less favorable prognosis than that of other patients.

SEIZURES IN OFFSPRING OF EPILEPTIC PARENTS

The risks of unprovoked seizures in the offspring of parents with generalized versus partial epilepsies among 687 patients born in Rochester, MN between 1922 and 1985 and followed for the occurrence of seizures through 1986 are reported from the Division of Epidemiology and Department of Neurology, Columbia University, New York, the University of Texas Health
The incidence of recurrent unprovoked seizures in the total group was 3.3% and three times that expected. The incidence was approximately the same in the offspring of parents with either generalized or partial seizures. For parents with absence generalized seizures the incidence of epilepsy among offspring was substantially higher than that for offspring of parents with other types of generalized onset seizures and was three times as high as for partial cases. The early age at onset and idiopathic nature of the epilepsy explained only in part the higher incidence in offspring of absence cases. These offspring had a higher risk not only for absence seizures but for other seizure types as well, suggesting that absence epilepsy is not genetically distinct from other seizure types of epilepsy. For offspring of parents in the largest subset of generalized seizures (primary generalized tonic-clonic convulsions) there was no evidence of higher risk than for offspring of parents with partial seizures. (Ottman R et al. Seizure risk in offspring of parents with generalized versus partial epilepsy. Epilepsia March-April 1989; 30:157-161).

COMMENT: These findings contrast with the widely held assumption that partial epilepsies are less likely than generalized epilepsies to be genetic. The dramatically elevated risks in offspring of probands with absence seizures agreed with the findings of Metrakos and Metrakos (1961). However, in the present study, the increased risk in offspring of absence cases was not restricted to absence seizures, but was observed in all seizure types. The authors suggest that the data are more consistent with a common genetic basis for all seizure types, with absence cases having a higher genetic liability than other cases, leading to a higher risk for all seizure types in their relatives. This study did not take into account the etiology of seizures and febrile convulsions were not included.

ATAXIA

THYROTROPIN-RELEASING HORMONE FOR CEREBELLAR ATAXIA

A nine year old girl with cerebellar ataxia that responded to thyrotropin-releasing hormone is reported from the Department of Pediatrics, Kyoto Prefectero University of Medicine, Kyoto, Japan. Clinical improvement occurred 18 months after the onset of cerebellar ataxia and neurological deficits which included speech impairment, gait disturbance, ataxia of the extremities and positional nystagmus. CSF examination demonstrated that the concentrations of 5-HIAA and HVA increased and that the 5-HIAA/HVA ratio rose from 0.243 to 0.358 during TRH treatment. The levels of monoamine metabolites in the CSF reflect CNS biogenic amine turnover. The changes observed suggested that TRH influenced serotonin neurons rather than catecholamine neurons. The preparation of TRH was protireline tartrate: Takeda Co. Ltd., Japan and the dose injected intravenously was 1 mg per day for 20 days. Improvement in gait began to improve immediately after the treatment was begun. (Takeuchi Y et al. Efficacy of thyrotropin-releasing hormone in the treatment of cerebellar ataxia. Pediatr Neurol Mar-Apr 1989; 5:107-110).