Sciences Center, Houston, and the Section of Clinical Epidemiology, Mayo Clinic, Rochester, MN. 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).

COMMENT: Thyrotropin-releasing hormone (TRH) therapy has been used in several neurologic disorders, including spinocerebellar degeneration, amyotrophic lateral sclerosis, and infantile spasms with hypsarrhythmia (see Ped. Neur. Briefs June 1987; 1:3). The present patient had an acute cerebellar ataxia following an infection of unknown origin and persisting for 18 months before treatment with TRH was begun.

CEREBELLAR ATAXIA BENEFITTED BY 5-HYDROXYTRYPTOPHAN

Levorotatory 5-hydroxytryptophan (10 mg/kg/day) was found to benefit patients with various inherited or acquired cerebellar ataxias in a long-term randomized, double-blind study at the Hopital Neurologique, Alexis Carrel Faculty of Medicine, Lyon, France. Of 30 patients in test and placebo groups, 2 had Friedreich's ataxia, 8 had postsurgical ataxia, 6 multiple sclerosis, 2 brain stem infarction, and 12 cerebellar cortical atrophy. The majority were adults, and the degree of ataxia was measured by four semiquantitative subtests. The treatment continued initially for four months, was extended in five patients without controls for a further eight months. Levo-5-hydroxytryptophan significantly improved the ataxia score and modified the time of standing upright, the speed of walking, speaking, and writing. The process appears to be serotonin-dependent and provides benefit particularly in static cerebellar disturbances and speech dysarthria caused by lesions of the anterior vermis. (Trouillas P et al. Improvement of cerebellar ataxia with levorotatory form of 5-hydroxytryptophan. A double-blind study with quantified data processing. Arch Neurol Nov 1988; 45:1217-1222).

COMMENT. The rationale for this treatment was the discovery of serotoninergic nerve terminals in the cortex of the cerebellum, and the induction of cerebellar tremor by the experimental depletion of serotonin. The treatment was well tolerated and should be considered for trial in children with Friedreich's ataxia and in static, postsurgical or post-viral cerebellar syndromes.

HEADACHE

EEG AND DIET RELATED MIGRAINE

Thirty-eight patients with a history of diet induced migraine were studied with recording of clinical responses and electroencephalography at the Departments of Neurology and Biometry, Kansas University Medical Center, Kansas City, Kansas. The subjects consisted of 30 females and 8 males aged from 17 to 38 years, all having a history of migraine attacks consistently provoked by either chocolate, cheese, or alcohol. With the exception of one patient with a febrile seizure at age 2, none had a seizure history. There was a family history of migraine in first degree relatives in 22 patients (58%). Tests were carried out on an initial baseline day and on a second day, after challenge with chocolate, red wine, cheese, and fasting. Migraine headache occurred in 16 (42%), four with scintillating scotomata. Electroencephalograms were abnormal in 12 subjects (32%) most abnormalities being nonspecific slow waves. In three cases there were paroxysmal