remission rate of 0.71 after AED withdrawal, predictors of relapse were adolescent age at onset, symptomatic epilepsies, and an abnormal interictal EEG (Berg AT, Shinnar S. Relapse following discontinuation of antiepileptic drugs: a meta-analysis. Neurology 1994;44:601-608). An identical remission rate was reported by Camfield and colleagues in a study in which patients with absence and minor motor seizures were excluded and seizure type was not of predictive value. (see Progress in Pediatric Neurology II, PNB Publ, 1994, for further discussion of outcome studies in childhood epilepsies). Greater attention to EEG characteristics, especially form of spike-wave complexes and duration of paroxysms, at the time of diagnosis and later at AED withdrawal could be more revealing in future outcome studies.

Accidental injuries, especially bicycle accidents, pose a 27% risk during absence seizures in children, according to a study of 59 patients at the IWK-Grace Health Centre and Dalhousie University, Halifax, Nova Scotia (Wirrell EC, Camfield PR, Camfield CS, Dooley JM, Gordon KE. Accidental injury is a serious risk in children with typical absence epilepsy. Arch Neurol 1996;53:929-932).

NOCTURNAL FRONTAL LOBE EPILEPSY

The electroclinical pattern of 33 patients with familial, autosomal dominant, nocturnal frontal lobe epilepsy was studied, including video-polysomnographic monitoring in 12, at the University of Milano, School of Medicine, Italy. The syndrome is characterized by clusters of brief nocturnal motor seizures during sleep, beginning in childhood and persisting throughout adult life. The motor seizures during sleep varied from thrashing hyperkinetic activity to tonic extension with clonic movements. The most frequently repeated patterns included pelvic thrusting, facial grimacing and moaning, and dystonic posturing. Some had sudden elevation of the head and an expression of fear. Misdiagnoses included benign nocturnal parasomnias, including nightmares, night terrors, and somnambulism. Diurnal episodes in 58% included generalized shivering followed by loss of consciousness, and complaints of tingling and daytime sleepiness. Interictal and ictal EEGs showed nonspecific patterns (atypical K-complexes), or epileptiform abnormalities (in 58% of patients), consisting of bilateral or right frontal spikes, during stage 2 non-REM sleep. Normal EEGs were recorded during wakefulness. Both nocturnal and diurnal attacks were controlled by carbamazepine or clonazepam. (Oldani A, Zucconi M, Ferini-Strambi L, Bizzozero D, Smirne S. Autosomal dominant nocturnal frontal lobe epilepsy: electroclinical picture. Epilepsia October 1996;37:964-976). (Reprints: Dr A Oldani, Sleep Disorders Center, IRCCS H San Raffaele, via Prinetti 29, 20127 Milano, Italy).

COMMENT. Nocturnal frontal lobe epilepsy is often misdiagnosed as nightmares, night terrors, or somnambulism. Nocturnal paroxysmal dystonia is also considered in the differential diagnosis. EEGs are frequently nonspecific, and video-polysomnographic monitoring is often essential. If the diagnosis is suspected but unconfirmed by EEG, a trial of antiepileptic drugs may still be warranted.

RISK OF SEIZURE RECURRENCE AFTER FIRST SEIZURE

The long-term recurrence risk after a first unprovoked seizure was determined in a prospective study of 407 children, followed for a mean of 6.3 years, at the Montefiore Medical Center, Albert Einstein College of Medicine, Bronx, New York. Seizures recurred in 42%; the cumulative risk of seizure recurrence at 1, 2, 5, and 8 year follow-up was 29%, 37%, 42%, and 44%,