1:256 (cut off <1:64). *Ehrlichia* and Lyme serology were negative. Significant improvement in symptoms occurred within 3 weeks of starting antibiotic therapy, and a repeat CSD titer was 1:128. After 1 year follow-up and gradual tapering of antiepileptic drugs the patient remains seizure-free. Brain MRI and SPECT are now normal. (Nowakowski GS, Katz A. Epilepsia partialis continua as an atypical presentation of cat scratch disease in a young adult. Neurology December (1 of 2) 2002;59:1815-1816). (Reprints: Dr GS Nowakowski, Mayo Clinic, 200 First St, SW, Rochester, MN 55905).

COMMENT. Encephalopathy and seizures are an unusual presentation of cat scatch disease, and symptoms may be delayed for several weeks after the kitten scratch or bite. Diagnosis is dependent on the history of kitten exposure and positive serology. Spontaneous recovery usually occurs after several days without the need for antibiotics. In the present case, symptoms persisted for several weeks and recovery was prompt only after antibiotics were initiated. The differential diagnosis includes Lyme encephalitis which may occur concurrently. (See Progress in Pediatric Neurology II pp 421-423, for review of 4 previous articles on neurologic complications of cat scratch disease.

GENETICS OF SIMPLE FEBRILE SEIZURES

A clinical and genetic study of three families with simple febrile seizures (FS) and an autosomal dominant (AD) trait with high penetrance is reported from the Hopital Pitie-Salpetriere, Paris, France. Among 29 affected family members, FS ceased before 5 years of age, only one had rare afebrile seizures in addition, and none developed epilepsy. A genome-wide scan in two famiies identified a new locus on chromosome 6q22-q24. This linkage was absent in the third family, supporting genetic heterogeneity of the AD form of simple FS. (Nabbout R, Prud'homme J-F, Herman A et al. A locus for simple pure febrile seizures maps to chromosome 6q22-q24. Brain December 2002;125:2668-2680). (Respond: Rima Nabbout MD, INSERM U289, Hopital Pitie-Salpetriere, 47 boulevard de l'Hopital, 75013 Paris, France).

COMMENT. This mapping to 6q22-q24 is the first identified locus responsible for simple febrile seizures. Identification of the gene is ongoing. This phenotype differs from the known loci reported for FS and GEFS+. All modes of inheritance have been described, autosomal dominant, autosomal recessive and polygenic.

A nonsense mutation of the MASS1 gene is reported in a family with febrile and afebrile seizures, from University of Tsukuba, Ibaraki, Japan (Nakayama J et al. Ann Neurol Nov 2002;52:654-657).

ABSENCE EPILEPSY AND PAROXYSMAL DYSKINESIA

Six patients aged 6 to 27 years (mean, 14 years) with childhood absence epilepsy and paroxysmal dyskinesia (PD), identified at five European centers participating in a study group, are reported from Great Ormond Street Hospital, London, UK. The onset of absence seizures was early, at a mean age of 16 months (range, 3 months to 3 years 6 months), and seizures remitted between age 8 and 13 years. The types of associated PD included paroxysmal kinesigenic dyskinesia (1 patient), paroxysmal exercise-induced dystonia (3 patients), and paroxysmal tonic upgaze (two siblings). Apart from the siblings with tonic upgaze which had an earlier onset, PD developed after the onset of absence seizures, and continued after seizures had remitted. PD improved with increasing age and was not severely disabling. Seizures and PD were idiopathic and were thought to be genetic. Seizures were accompanied by a characteristic 3Hz spike-and-wave EEG and they responded to ethosuximide. (Guerrini R, Sanchez-Carpintero R, Deonna T