

OUTCOME AFTER TEMPORAL LOBECTOMY

Thirty seven patients, ages 7 to 18 years, who underwent temporal lobectomy for intractable seizures at the Mayo Clinic, Rochester, MN, between 1970 and 1983, were followed for a mean of 19 years (range 4 - 27 years). Mean age at seizure onset was 7.2 yrs (range 1-16), and mean age at surgery was 14.4 yrs (range 7-18). An excellent outcome (0-4 seizure frequency score based on Engel classification) was obtained in 31 (88%) patients at 5 years, and in 19 (60%) at 15 years. Of those followed at least 15 years, 40% (13/32) were seizure free at 5 years but had recurrence of seizures at 15-year follow-up; 9.5% (3/32) improved significantly; and 50% (16/32) were unchanged. Of 13 with long-term seizure recurrence (9 on medication), 6 had abnormal pathology including oligodendroglioma or ganglioglioma (4), ectopic neurons (1), and mesial temporal sclerosis in 1. Of 19 patients who improved or did not relapse, only 2 had abnormal pathology (astrocytoma and arachnoid cyst) ($p=0.03$). Long-term seizure recurrence was independent of a positive family history of epilepsy, history of head trauma, encephalitis, or febrile convulsions, or abnormal postoperative EEG. Only 3 of 32 followed were unemployed; 6 had no driver's license. (Jarrar RG, Buchhalter JR, Meyer FB, Sharbrough FW, Laws E. Long-term follow-up of temporal lobectomy in children. *Neurology* November (2 of 2) 2002;59:1635-1637). (Reprints: Dr Jeffery Buchhalter, Child and Adolescent Neurology, Mayo Clinic, 200 First Street SW, Rochester, MN 55905).

COMMENT. In this long-term study of children following temporal lobectomy for intractable seizures, of 88% with an initial excellent outcome by 5 years, half of the patients had recurrence of seizures by 15 years. The incidence of mesial temporal sclerosis in patients with long-term recurrence was only 2.7% in this series, compared to 14% in previous reported series. The poorer outcome with long-term follow-up should be considered in decisions to operate early in children with temporal lobe seizures.

The unpredictable nature of postoperative seizure relapse was stressed in a report of 282 temporal resections, mean age 26 years (range 4-59 years), from Kings College Hospital, London, UK (Hennessy MJ et al. *Brain* 2000;123:2445-2466; see *Ped Neur Briefs* 2001;15:5-6). The relapse rate was approximately 20%, either immediately or after a 12 month seizure-free interval.

Previous studies of the natural history of recurrent seizures after resective surgery for epilepsy have shown that the outlook is better if seizure recurrence is delayed until after the first postoperative year. Also, changes in antiepileptic medication, dosages, or poor compliance may be important factors in recurrence of seizures. (see Wingkun EC, Awad IA et al. *Epilepsia* 1991; see *Progress in Pediatric Neurology II*, PNB Publishers, 1994;pp136-7).

CAT SCRATCH DISEASE AND EPILEPSIA PARTIALIS CONTINUA

An 18-year-old female young adult with seizures associated with cat scratch disease is reported from Norwalk Hospital, Yale University School of Medicine, CT. She presented with difficulties in speaking followed by generalized tonic-clonic seizures and continuous right facial motor seizures refractory to antiepileptic drugs. Audio-video EEG monitoring revealed the epilepsy partialis involving the right lower face and occasionally, the tongue and larynx, without surface EEG abnormalities. MRIs were initially normal, and later showed an increased signal on T2-weighted images in the left frontoparietal region. SPECT also showed a focus of increased activity in the same region. The patient had a new kitten at home and cat scratch disease (CSD) serology for *Bartonella henselae* was positive at a titer of

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 scratch 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 Hôpital 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 families 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, Hôpital Pitie-Salpetriere, 47 boulevard de l'Hôpital, 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