concerning hippocampal injury with febrile seizures.

Interictal EEG spikes for seizure lateralization in mesial temporal lobe epilepsy. In a study of 21 patients at the Universitatsklinik fur Neurologie, Vienna, Austria, lateralization of clinical seizures was correct (ipsilateral to side of hippocampal sclerosis) in almost 100% of cases with unitemporal spikes and in only 50% of those showing bitemporal spikes (Serles W et al. Clinical seizure lateralization in mesial temporal lobe epilepsy. Differences between patients with unitemporal and bitemporal interictal spikes. Neurology March 1998;50:742-747).

TEMPORAL LOBE MALFORMATIONS AND EPILEPSY

Temporal lobe developmental malformations (TLDM) (focal cortical dysplasia and balloon cells) occurred with mesial temporal sclerosis as dual pathologies in 87% of 30 patients with unilateral TLDM and intractable partial epilepsy treated at the UAB Epilepsy Center, University of Alabama at Birmingham, AL. A quantitative MRI analysis with the inclusion of a normalization process was used for the detection of bilateral hippocampal formation atrophy when visual analysis, using optimal protocol including IR and FLAIR sequences, failed. The dual pathologies might be developmental or the hippocampi could be damaged secondarily by a kindling effect of repeated seizures from the TLDM. The surgical implications of the dual pathology are discussed. (Ho SS, Kuzniecky RI, Gilliam F, Faught E, Morawetz R. Temporal lobe developmental malformations and epilepsy. Dual pathology and bilateral hippocampal abnormalities. Neurology March 1998;50:748-754). (Reprints: Dr Ruben I Kuzniecky, Department of Neurology, UAB Station, Birmingham, AL 35294).

COMMENT. Mesial temporal sclerosis can occur in association with temporal lobe focal dysplasia in patients with refractory temporal lobe epilepsy. These authors have previously demonstrated that the MRI will identify lesions with moderate to severe histologic abnormalities, but may not detect mild neuronal and cortical dysplasias. Dual pathology in patients with mesial sclerosis may be underdiagnosed by MRI and may explain the occurrence of temporal lobe epilepsy as a sequel to complex febrile seizures in some cases.

Of 67 patients with medial temporal lobe seizures controlled by temporal lobectomy at Yale University and Epilepsy Center, 45 (67%) had histories of febrile seizures before 5 years of age, and of these, 33 had complex febrile seizures lasting longer than 30 minutes. The duration of the febrile convulsion was the most important predictor of temporal lobe epilepsy (TLE) in a study at the University of Western Ontario, London, Ontario. The mean duration of the FC was 100 min in patients with TLE, and 9 min in those without TLE. (See Progress in Pediatric Neurology III, PNB Publishers, 1997;pp19 and 32)).

DUAL ETIOLOGY OF RASMUSSEN'S SYNDROME

Five patients with Rasmussen's syndrome reported from the Montreal Neurological Institute had the typical findings of chronic encephalitis together with tuberous sclerosis, tumor, or vascular abnormality discovered on pathological examination of tissue removed at operation. Dual pathologies were found in 10% of the patients in the authors' series. (Hart YM, Andermann F, Robitaille Y et al. Double pathology in Rasmussen's syndrome. A window on the etiology? Neurology March 1998;50:731-735). (Reprints: Dr F Andermann, Montreal Neurological Institute, 3801 University Street, Montreal, Quebec H3A 2B4).

COMMENT. Rasmussen's syndrome of focal seizures and progressive hemiparesis begins in early childhood, is often preceded by a minor febrile and probable viral illness, and is manifested by epilepsia partialis continua and intellectual deterioration. Short-term reduction in seizure frequency has been reported following IV immunoglobulin and long-term oral prednisolone treatments at the Montreal Neurological Institute. (See Progress in Pediatric Neurology III, 1997;p127).

EARLY WITHDRAWAL OF ANTIEPILEPTIC DRUGS

Recurrence rate, risk factors for recurrence, and outcome in 161 children after early withdrawal of antiepileptic drugs (AEDs) were studied at Leiden University and other centers in The Netherlands. The probability of remaining seizure free at 24 months was 51% for 78 patients whose AEDs were discontinued after a 6 month seizure-free period, and 52% for 83 patients with a 12 month period of seizure control. Risk factors for relapse were partial epilepsy, onset of seizures at 12 years or older, known seizure etiology, and epileptiform EEG. After a mean 42 month follow-up, 129 (80%) were seizure free for at least 1 year, two thirds without AEDs. (Peters ACB, Brouwer OF, Geerts AT et al. Randomized prospective study of early discontinuation of antiepileptic drugs in children with epilepsy. Neurology March 1998;50:724-730). (Reprints: Dr ACB Peters, Department of Child Neurology, University Hospital Utrecht and Wilhelmina Children's Hospital, PO Box 85500, 3508 GA Utrecht, The Netherlands).

COMMENT. Early withdrawal of antiepileptic therapy after 6 or 12 months seizure control is followed by seizure recurrence in 50% of patients, regardless of the treatment duration. A comparison of these results of early drug withdrawal with previous reports of later withdrawal (2, 3, or 4 years) suggests an optimal seizure-free treatment period of 2 years. The shorter duration of seizure-free therapy results in a higher relapse rate. (See Progress in Pediatric Neurology III, 1997;pp115-122).

GENETIC TWIN STUDIES OF EPILEPSY SYNDROMES

The genetics of epilepsy syndromes was studied by an evaluation of 253 twin pairs at the University of Melbourne, Australia. One or both twins had seizures, Among monozygous (MZ) and dizygous (DZ) twin pairs, 44% and 10% were concordant for seizures, respectively. Both twins had the same major epilepsy syndromes in 94% of concordant MZ pairs and 71% of concordant DZ pairs. The concordance rates for generalized epilepsies, both idiopathic and symptomatic, were greater than those for partial epilepsies. Febrile seizures and unclassified epilepsies had intermediate rates. Genetic factors play a role in all epilepsy syndromes but especially in generalized epilepsies. (Berkovic SF, Howell RA, Hay DA, Hopper JL. Epilepsies in twins: Genetics of the major epilepsy syndromes. Ann Neurol April 1998;43:435-445). (Respond: Dr Samuel F Berkovic, Department of Neurology, Austin and Repatriation Medical Centre, Heidelberg (Melbourne), Victoria 3084, Australia).

COMMENT. The authors conclude that genetics of epilepsy is syndrome-specific rather than a broad genetic predisposition. Their findings support those of Lennox WG who pioneered the research on genetics of epilepsy in twins in the early 1950s. I was privileged to be a Fellow in his "Seizure Clinic" at Harvard when he and a psychologist co-worker reported a series of 173 twin pairs (Lennox WG, Jolly DH. Seizures, brain waves and intelligence tests of epileptic twins. A Res