large, four-generation Utah family, significant evidence for a novel febrile seizure locus (FEB3) on chromosome 2q23-24 with linkage to the marker D2S2330. Four loci associated with a febrile seizure phenotype have now been defined, FEB1-3 and GEFS. The clinical variations observed in febrile seizure families can now be explained by genetic heterogeneity, as previously suspected, and ongoing genotype/phenotype correlations should segregate these febrile seizure genes and their specific mutations, leading to advances in their causes and treatment.

That the cause of febrile seizures and epilepsy is multifactorial has been expressed in numerous earlier reports (Millichap JG. Febrile Convulsions, Macmillan, 1968), but until recently the inheritance factor has not been supported by specific gene linkages. Evidence that febrile seizures have a strong genetic component has been derived from family and twin studies (Lennox WG and Lennox MA. Epilepsy and Related Disorders, Little, Brown, 1960), and EEG studies (Stores G. Arch Dis Child 1991;66:554-557). See Progress in Pediatric Neurology II, PNB Publ, 1994;pp20-22, for literature abstracts and commentaries.

An excess of concordant monozygotic compared to dizygotic twin pairs, observed for both febrile seizures and epilepsy, is significant, showing a factor of almost 3 to 1. The familial incidence of febrile seizures is quoted as high as 58%, with a mean of 17%; the incidence was 30% in an unselected series of patients. Siblings of affected children have a 10% risk of developing febrile seizures. If the index child and 1 parent are affected, the risks to siblings are 30-40% (50% if both parents are affected). The risk for developing afebrile seizures and epilepsy in later life is estimated at 2% to 7% of children with simple febrile seizures, but the risk is higher in those with complex febrile seizures. In the Utah febrile seizure family and FEB3 gene carriers, all having simple febrile seizures, the risk of epilepsy was 47%, and considerably higher than that reported in population studies and most febrile seizure families (Nelson KB, Ellenberg JH. Eds Febrile Seizures. New York, Raven Press, 1981). This indicates an unusually high epilepsy predisposition among the FEB3 family members, and the likelihood of an additional predisposing epilepsy gene.

MEG LOCALIZATION IN EPILEPSY SURGERY

The concordance rate between the anatomical location of interictal magnetoencephalography (MEG) spike foci with the location of ictal onset zones identified by invasive ictal intracranial electroencephalographic (EEG) recordings was determined in 11 children evaluated for epilepsy surgery at the Hospital for Sick Children and University of Toronto, Ontario, Canada. In 10 of 11 patients, the anatomical location of epileptiform discharges determined by MEG corresponded to the ictal onset zone recorded by subdural electrodes. Functional EEG mapping of the somatosensory hand area was the same as the MEG localization. Seizures were completely or more than 90% controlled after surgery in 9 patients, at a mean follow-up of 24 months. Noninvasive magnetic source imaging by MEG and MRI provides an accurate presurgical localization of epileptic foci in children with refractory nonlesional extratemporal epilepsy and may obviate the need for invasive monitoring. (Minassian BA, Otsubo H, Weiss S, Elliott I, Rutka JT, Snead III, OC. Magnetoencephalographic localization in pediatric epilepsy surgery: Comparison with invasive intracranial electroencephalography. Ann Neurol October 1999;46:627-633). (Respond: Dr O Carter Snead III, Division of Neurology, Department of Paediatrics, Hospital for Sick Children and University of Toronto, 555 University Avenue, Toronto, Ontario, Canada M5G 1X8).

COMMENT. MEG detects magnetic fields generated by intraneuronal electrical currents, and MEG spikes correlate precisely with irritative zones

adjacent to an epileptogenic lesion. Unlike EEG, MEG is restricted to interictal recordings, but the procedure is noninvasive. This study demonstrates that the accuracy of MEG localization of epileptogenic foci is equal to that of the invasive ictal EEG recordings.

LANGUAGE RECOVERY AFTER LEFT HEMISPHERECTOMY

The language proficiency of 6 right-handed children (ages 7-14 years) with Rasmussen's syndrome, who underwent left hemidecorticectomy after 5 or more years of normal language development before seizures, were investigated at the Johns Hopkins Hospital, Baltimore, MD. The ability to discriminate consonants and vowels was improved within 4 to 16 days after surgery, when compared to that before surgery, whereas other language functions remained severely impaired until 6 months. Word repetition and phrasal comprehension recovered fully within 1 year, while expressive functions and naming were delayed, and spontaneous speech was telegraphic and restricted to single words. All patients could walk unaided, but had little use of the right arm or hand. Seizures were controlled and antiepileptic drugs were withdrawn in 3.

Rapid recovery of receptive language after surgery suggests that the intact right hemisphere has an innate ability to analyse phonemes and discriminate consonants and vowels. Delayed and partial recovery of expressive language functions may be attributed to plasticity of the right hemisphere, persisting after 5 years of age, the proposed critical age for completion of language acquisition and lateralization. (Boatman D, Freeman J, Vining E et al. Language recovery after left hemispherectomy in children with late-onset seizures. Ann Neurol Oct 1999;46:579-586). (Respond: Dr Boatman, Department of Neurology, Johns Hopkins Hospital, 600 North Wolfe Street, Meyer 222, Baltimore, MD 21287).

COMMENT. Explanations offered for the immediate improvement in phoneme discrimination after left hemispherectomy in these patients included: 1) the innate receptive language capability of the right hemisphere; and 2) a bilateral representation of phoneme processing, at least until adolescence. The right hemisphere is capable of functioning when receptive, and to some extent expressive, language abilities have been undermined by seizures and damage to the left hemisphere in young children.

EARLY ONSET SEIZURES BEGET SEIZURES

The long-term effect of early-life seizures on later seizure-induced neuronal damage and behavior was investigated in the laboratory using systemic kainate to induce seizures in rats at the Massachusetts General Hospital, Boston, MA. Memory was tested using a Morris water maze, and brains were examined histologically for evidence of injury. Seizures induced during the second week of life (15 days) were not associated with brain injury or cell death, but they predisposed animals to extensive neuronal injury and impairment of learning when seizures were again induced in adulthood (45 days). (Koh S, Storey TW, Santos TC, Mian AY, Cole AJ. Early-life seizures in rats increase susceptibility to seizure-induced brain injury in adulthood. Neurology September 1999;53:915-921). (Reprints: Dr Andrew J Cole, Epilepsy Service, Massachusetts General Hospital, VBK 830, 55 Fruit Street, Boston, MA 02114).

COMMENT. These laboratory studies confirm previously reported evidence that seizures in young experimental animals can be followed by delayed brain growth, and lowered seizure thresholds. (Theodore W, Wasterlain CG. Do early seizures beget epilepsy? Editorial. Neurology Sept 1999;53:898-899). The studies