

30-40% (50% if both parents are affected). (See Bird TD. Epilepsia 1987;28 (Suppl 1):S71-81, reviewed in Progress in Pediatric Neurology I, PNB Publ, 1991;pp15-16). In my own series of FS patients, a family history of FS is found in 17% of cases. The incidence is similar in those complicated by nonfebrile seizures, and an inherited tendency to nonfebrile seizures is equally prevalent. Recurrent nonfebrile seizures, especially generalized tonic-clonic epilepsy, among children with FS are reported in 20% (average) of 4500 patients in 35 publications between 1929 and 1964 . The average familial incidence of epilepsy in the total was 15%. Analyses of these case reports suggest that the genetically determined factors that may result in a low threshold to FS are associated with an equally prevalent genetic predisposition to nonfebrile seizures (Millichap JG. Febrile Convulsions. New York, Macmillan, 1968). These previous reports are in agreement with the concept of the GEFS+ syndrome, and families identified warrant genetic analysis.

### TEMPERATURE, AGE, AND FEBRILE SEIZURE RECURRENCE

Factors that predict recurrence of a seizure during a subsequent fever episode were studied in 230 children included in a randomized placebo controlled trial of ibuprofen prophylaxis treatment at the Sophia and Juliana Children's Hospitals, Rotterdam, the Netherlands. There were 67 febrile seizure (FS) recurrences in a total of 509 fever episodes; 35 (52%) occurred within 2 hours of fever onset and 32 (48%) after more than 2 hours of fever. The median temperature with recurrences early in the fever episode was lower (39.3°C) than that registered during delayed FS recurrences (40.0°C). The risk of FS recurrence increases with temperature at fever of onset and during the febrile episode and decreases with age. A child aged 1 year with a 30% recurrence risk has a 5 to 10% risk at 4 years. (van Stuijvenberg M, Steyerberg EW, Derksen-Lubsen G, Moll HA. Temperature, age, and recurrence of febrile seizure. Arch Pediatr Adolesc Med Dec 1998;152:1170-1175). (Respond: Henriette A Moll MD, PhD, Sophia Children's Hospital, Rm Sp 1543, Dr Molewaterplein 60, 3015 GJ Rotterdam, the Netherlands).

COMMENT. Recurrent febrile seizures occur within 2 hours of onset of a fever episode in 50% of cases. Seizures recurring after 2 hours of fever are associated with higher temperature levels than those within the first 2 hours of fever. The risk of FS recurrence with subsequent fever decreases with age and increases with temperature at fever onset and during fever episodes.

A *threshold convulsive temperature* , suggested by this report, was first demonstrated in laboratory and clinical studies in Jan 1959, exactly forty years ago (Millichap JG. Studies in febrile seizures. I. Height of body temperature as a measure of the febrile seizure threshold. Pediatrics 1959;23:76-85. *Idem*. Febrile Convulsions. New York, Macmillan, 1968). The rapidity of rise of body temperature, previously proposed as an essential mechanism for febrile seizures, was non-contributory. Factors found to modify the threshold convulsive temperature include age, brain maturation, electrolyte imbalance, and various medications. A postictal elevation in seizure threshold requires a relatively higher body temperature to induce a second seizure.

### GENETICS OF BENIGN ROLANDIC EPILEPSY

Twenty-two nuclear families with benign epilepsy of childhood with centrotemporal spikes (BECTS, rolandic epilepsy) were analyzed by DNA linkage studies at the Department of Neuropediatrics, University of Kiel, Germany. Screening of all chromosomal regions known to harbor neuronal nicotinic acetylcholine receptor (AChR) subunit genes found evidence for linkage of BECTS

to chromosome 15q14, similar to that involved in families with juvenile myoclonic epilepsy. An autosomal recessive mode of inheritance with heterogeneity was suggested. (Neubauer BA, Fiedler B, Himmelhein B, et al. Centrottemporal spikes in families with rolandic epilepsy. Linkage to chromosome 15q14. Neurology Dec 1998,51:1608-1612). (Reprints: Dr Bernd A Neubauer, Dept of Neuropediatrics, University of Kiel, Schwanenweg 20, 24105 Kiel, Germany).

COMMENT. Both benign rolandic epilepsy, a common partial, idiopathic epilepsy syndrome, and juvenile myoclonic epilepsy, a generalized idiopathic syndrome, have been linked genetically to chromosome 15q14.

## NEONATAL DISORDERS

### OUTCOME OF NEONATAL CEREBRAL INFARCTION

Antenatal and perinatal factors, early clinical signs, electroencephalograms (EEG), and magnetic resonance imaging (MRI) findings were compared with neurodevelopmental outcome in 24 infants with neonatal cerebral infarction followed at the Dept of Paediatrics, Hammersmith Hospital, London, UK. Infarcts defined by MRI involved a major cerebral vessel in 19 and borderzones in 5. Duration of follow-up ranged from 15 months to 5 years. Of 7 (29%) infants with abnormal neuromotor outcome, 5 were hemiplegic and 2 showed asymmetry of tone or function. None developed seizures. Adverse antenatal factors, present in 11 (46%), perinatal continuous decelerations below 90 with slow recovery in 14, meconium staining in 11, cord blood pH<7.1 in 2, and Apgar <5/1min in 5 were not related to outcome. Abnormal signs on neonatal neurologic exam, chiefly hypotonia, were poor prognostic indicators. Both EEG and MRI were predictors of abnormal outcome. Abnormal neonatal EEG background was associated with later hemiplegia whereas epileptic discharges were not predictive. MRI showing involvement of hemispheres, basal ganglia, and internal capsule, but not one or two of these regions, tended to develop hemiplegia or asymmetry of tone. Concomitant thalamic involvement did not increase the risk of poor outcome. (Mercuri E, Rutherford M, Cowan F et al. Early prognostic indicators of outcome in infants with neonatal cerebral infarction: a clinical, electroencephalogram, and magnetic resonance imaging study. Pediatrics Jan 1999;103:39-46). (Reprints: Dr Eugenio Mercuri, Dept of Paediatrics, Hammersmith Hospital, Du Cane Rd, London W12 0HN, UK).

COMMENT. Neonatal EEG and MRI findings may be predictive of later outcome and development of hemiplegia in infants born with cerebral infarcts. Abnormal antenatal and perinatal factors and neonatal hypotonia fail to identify infants with a poor prognosis.

### ANTE/INTRAPARTUM RISKS OF NEONATAL ENCEPHALOPATHY

The role of antepartum and intrapartum factors in the etiology of neonatal encephalopathy (NE) in 164 term infants was investigated in a Western Australian case-control study, with 400 randomly selected controls. The prevalence of NE was 3.8/1000 term live births, with a 9.1% case fatality. The features of NE included seizures, abnormal tone, apneas, feeding difficulties, abnormal consciousness, and ventilatory support. Independent risk factors before conception and in the antepartum period included lower socioeconomic status, family history of seizures or other neurologic disease, conception after infertility treatment, maternal thyroid disease, severe pre-eclampsia, bleeding during pregnancy, viral illness,