onset of FS was <12 months, if family history was positive for unprovoked seizures, and if the initial FS was focal or partial. (Offringa M et al. Risk factors for seizure recurrence in children with febrile seizures: A pooled analysis of individual patient data from five studies. J Pediatr April 1994;124:574-84). (Reprints: Martin Offringa MD, Room EE 2091, Erasmus University, PO Box 1738, 3000 DR, Rotterdam, The Netherlands).

COMMENT. In a previous report of a follow-up study of 155 Dutch children the principal author had concluded that the predictive value of combined risk factors (age at onset, family history, height of fever) was superior to that of single variables (see Ped Neur Briefs March 1992;6:17). Similar risk factors have been identified previously by a metaanalysis study (Berg AT et al. J Pediatr 1990;116:329-37) and a prospective study (Berg AT et al. N Engl J Med 1992;327:1122-7). A threshold to febrile seizures based on the height of body temperature was first established in animals with seizures induced by microwave diathermy (Millichap JG. Pediatrics Jan 1959;23:76-85), and has been confirmed clinically (Febrile Convulsions, New York, Macmillan, 1968).

None of the patients in the pooled analysis study had received monitored prophylactic treatment, continuous or intermittent. Having established that 54% of children had one or more recurrences of febrile seizures, the authors may be encouraged to conduct trials of intermittent oral diazepam in their patient population at increased risk, especially in those between the ages of 12 and 24 months, with a positive family history, and whose first FS occurred with a temperature <40°C.

BENIGN FAMILIAL CONVULSIONS

Results of linkage analysis between benign infantile familial convulsions (BIFC) and two linked DNA markers, D20S19 and D20S20, in 52 members from eight BIFC pedigrees are reported from centers in Montpellier and Paris, France, and Rome and Treviso, Italy. The gene responsible for benign familial neonatal convulsions (BFNC) has been mapped to chromosome 20q in the close vicinity of these two DNA markers. Several recombinants were observed betweeen the BIFC locus and D20S19-D20S20 markers, whereas none appeared between the BFNC locus and the markers in 11 BFNC families. The gene responsible for BFNC is not implicated in BIFC. (Malafosse A et al. Benign infantile familial convulsions are not an allelic form of the benign familial neonatal convulsions gene. Ann Neurol April 1994;35:479-482). (Respond: Dr Malafosse, Laboratoire de Medicine Experimentale, CNRS UPR 9008-INSERM U249, Institut de Biologie, Bvd Henri IV, 34060 Montpellier, France).

COMMENT. The authors distinguish BFNC and BIFC by clinical and genetic markers, as follows: 1) Onset of BFNC is before 3 months and BIFC, after 3 months of age; 2) Seizures, generalized in BFNC and partial in BIFC; and 3) genetic heterogeneity.

Seizure patterns cannot be used to differentiate these benign familial convulsions without documentation by ictal EEG recordings. Ictal EEGs demonstrated a seizure of right frontal onset with secondary generalization and one of right frontal onset which remained focal in a neonate with BFNC presenting with seizures at 50 hours of age, and reported from the Prince of Wales Children's Hospital, Randwick,

Australia (Bye AME. Neonate with benign familial neonatal convulsions: Recorded generalized and focal seizures. Pediatr Neurol March 1994;10:164-165). BFNC is heterogeneous in clinical and EEG features and cannot be distinguished from BIFC on the basis of clinical seizure patterns.

JUVENILE MYOCLONIC EPILEPSY

Video-polygraphic analyses of 302 myoclonic seizures (MS) in 5 patients with juvenile myoclonic epilepsy (JME) are reported from the Department of Pediatrics, Tokyo Women's Medical College, Japan. MS occurred singly or repetitively and corresponded to generalized bilaterally synchronous single or multispike-and-wave complexes at 3-5 Hz. Either distal or proximal muscles were involved, and facial jerks were infrequent. MS were asymmetrical in 4 of 5 patients and 9 to 38% of all seizures. Contraction and postmyoclonic inhibition of proximal muscles with atonia alternated with a flapping tremor during analysis of EMG in outstretched arms; myoclonic EMG potentials were suddenly disrupted when the arms dropped. Four patients fell when MS were intense. (Oguni H, Fukuyama Y et al. Video-polygraphic analysis of myoclonic seizures in juvenile myoclonic epilepsy. Epilepsia March/April 1994;35:307-316). (Reprints: Dr H Oguni, Dept Pediatrics, Tokyo Women's Medical College, 8-1 Kawada-cho, Shinjuku-ku, Tokyo 162, Japan).

COMMENT. A total of eight articles on juvenile myoclonic epilepsy were published in the March/April 1994 issue of Epilepsia. Panayiotopoulos CP et al reported a 5-year prospective study of 66 patents with JME seen at the King Khalid University Hospital, Riyadh, Saudi Arabia (Epilepsia 1994;35:285-296). Prevalence was 10.2% among 672 patients with epilepsies. Inheritance was autosomal recessive with siblings involved in 13 of 41 families.. Diagnosis had been missed before referral in 63 and even after the initial visit in one-third. Age at onset was 10 years (range 5 - 16 years). Absence seizures (in 33%) predated myoclonic jerks (in 97%) by 4 years, and generalized tonic-clonic seizures (in 79%) by 4.4 years. Myoclonic and GTC seizures occurred mainly on awakening. One-third had an essential type tremor. A combination of valproate and clonazepam was the most effective treatment. Relapse occurred in 9 of 11 patients after drug withrawal.

Clinical and EEG asymmetries were reported in 26 of 85 (31%) patients with JME seen at the Department of Neurology, Bowman Gray School of Medicine, Winston-Salem, NC. Fourteen (54%) were initially misdiagnosed as having partial seizures. (Lancman ME et al. Epilepsia 1994;35:302-306).

FELBAMATE IN INTRACTABLE CHILDHOOD EPILEPSY

Of 51 children with intractable seizures treated for two months with add on felbamate (50-75 mg/kg/day) at the Scottish Rite Children's Hospital, Atlanta, GA, 51% responded with improved seizure control, 22% were unchanged, and 28% had increased seizure frequency. Significant insomnia limited the usefulness of felbamate in 39% of children. Other adverse effects included anorexia, hyperactivity, and choreoathetosis. (Trevathan E et al. Febamate: Short-term efficiacy and side effects in 51 children with intractable