problems in children with epilepsy are sufficiently prevalent to warrant routine screening. The screening program in the Epilepsy Center at the Lurie Children's Hospital of Chicago has been expanded to include patients up to 18 years of age. The American Academy of Pediatrics recommends routine developmental and autism screening for all infants and toddlers. (AAP. **Pediatrics** 2001 Jul;108(1):192-6) (Filipek PA et al. **Neurology** 2000 Aug 22;55(4):468-79).

GAIT DETERIORATION IN ADOLESCENTS WITH DRAVET SYNDROME

Researchers at the University of Melbourne, Australia studied prospectively changes in gait by age in patients with Dravet syndrome. Of 26 patients aged 2 to 34 years, 15 were male and 11 female, mean age 11.6 years; 23 had *SCN1A* mutations. Children aged 0 to 5 years (N=7) had a normal gait, whereas 5 of 10 aged 6 to 12 years and 8 of 9 children aged 13 years or older had crouch gait. Ataxia was rarely observed in this cohort. Crouch gait is characterized by increased hip and knee flexion and ankle dorsiflexion, weakness in antigravity lower limb muscles, and without hypertonia or spastic cerebral palsy. Physical examination showed with increasing age, a decrease in passive knee extension and hip extension, and an increase in external tibial torsion and pes planovalgus. Increase in hip internal rotation showed no age-related change. The Functional Mobility Scale showed universal independent walking over 5 and 50 m, whereas adolescents and adults showed wide variation in ratings over 500 m, wheelchair use to independent walking. (Rodda JM, Scheffer IE, McMahon JM, Berrkovic SF, Graham HK. Progressive gait deterioration in adolescents with Dravet syndrome. **Arch Neurol** 2012 Jul;69(7):873-8). (Respond: Ingrid E Scheffer PhD, MBBS, Florey Neuroscience Institutes, Melbourne Brain Centre, 245 Burgundy Rd, Heidelberg, Melbourne, Victoria 3084, Australia. E-mail: scheffer@unimelb.edu.au).

COMMENT. Some reports estimate that 50% to 60% of Dravet syndrome patients have an ataxic gait (Korff C, Laux L, Kelley K, Goldstein J, Koh S, Nordli D Jr. **J Child Neurol** 2007 Feb;22(2):185-94). The cause of crouch gait in adolescents with Dravet syndrome in the present study is unclear, and the typical gait abnormality usually referred to as ataxic or spastic was absent. The authors speculate a possible relation to joint problems and planus foot deformity as precursors, and discuss the pros and cons of orthotic and surgical management. The impact of seizures, type of mutation, and anticonvulsants on the development of crouch gait are also suspect. Development of some connective tissue disorders (Dupuytren's contracture, general joint pain) is linked to treatment with anticonvulsant drugs, especially phenobarbital. (Mattson RH, Cramer JA, McCutchen CB. **Arch Intern Med** 1989 Apr;149(4):911-4).

MRI ABNORMALITIES WITH FEBRILE STATUS EPILEPTICUS: THE FEBSTAT STUDY

Researchers in the FEBSTAT Study Team at centers in New York, Durham, Virginia, and Chicago, IL report MRI findings in their prospective study of the acute effects of febrile status epilepticus (FSE) in 199 children age 1 month to 5 years. Patients

presenting with FSE (>30 minutes) were enrolled in FEBSTAT within 72 hours of the FSE episode. Mean duration of FSE was 71.7 minutes; 129 (68%) were focal, and 37 (19%) had prior FS. A group of 96 children with first simple FS were imaged as controls, using a similar protocol. MRI was performed within 3 days of FSE in 129 (67.5%) cases, and within 1 week in 165 (86%) cases.

Twenty-two (11.5%) children had definitely abnormal (n=17) or equivocal (n=5) T2 signal increase in the hippocampus following FSE compared to none in the control group. ($p<0.0001$). Developmental abnormalities in the hippocampus were revealed in 20 (10.5%) of FSE patients compared to 2 (2.1%) of the controls ($p=0.0097$); hippocampal malrotation was the most common abnormality (15 FSE cases and 2 controls). Extrahippocampal imaging abnormalities occurred with equal frequency in the FSE group and controls (15.7% and 15.6%, respectively); temporal lobe abnormalities, including amygdala and insula, were more common in the FSE group (7.9%) than in controls (1.0%)($p=0.015$). Ongoing follow-up of cases should determine whether the presence of a concurrent developmental anomaly might increase risk of hippocampal sclerosis following FSE. (Shinnar S, Bello JA, Chan S, et al. for the FEBSTAT Study Team. MRI abnormalities following febrile status epilepticus in children. The FEBSTAT study. **Neurology** 2012 Aug 28;79(9):871-7).(Response and reprints: Dr Shlomo Shinnar MD, PhD. E-mail: sshinnar@aol.com).

COMMENT. The FEBSTAT study shows that FSE in young children is associated with a risk of acute hippocampal injury, and 10% of cases also have a developmental anomaly of the hippocampus that may increase susceptibility to complex seizures. Earlier reports of an association between prolonged febrile convulsions and mesial temporal sclerosis were confirmed at surgical excision in adults with refractory temporal lobe epilepsy (Falconer MA. **Epilepsia** 1971 Mar;12(1):13-31). However, Falconer may have used a liberal definition of a febrile convulsion that included patients with acute encephalopathy (Millichap JG. Febrile and later complex seizures. Discussion. In **Febrile Seizures.** Eds Nelson KB, Ellenberg JH. New York, Raven Press. 1981;75-79). A provocative editorial comment (Berg MJ, Abou-Khalil B. Childhood febrile status epilepticus: Chicken or egg? Does it matter? **Neurology** 2012 Aug 28;79(9):840-1) draws attention to results of viral studies reported by FEBSTAT (Epstein LG et al. Human herpesvirus 6 and 7 in febrile status epilepticus: The FEBSTAT study. **Epilepsia** Epub 2012 June 14). Viremia with HHV-6 was identified in 32% cases of FSE and with HHV-7 in 7%. The findings support the role of viruses in the etiology of FSE and mesial temporal lobe epilepsy and the use of antiviral agents and vaccines in prevention. The FEBSTAT study findings rekindle the debate on the mechanism and definition of the febrile seizure. Is the febrile seizure triggered solely by fever and a threshold convulsive temperature or by a viremia and a transitory encephalitis or encephalopathy?

A case report of a 21-month-old boy who presented with fever and right-sided focal seizure lasting 10 hours and followed by persistent hemiparesis illustrates a possible outcome of a complex febrile seizure and febrile status epilepticus. (Tenney JR, Schapiro MB. Child Neurology: Hemiconvulsion-hemiplegia-epilepsy syndrome. **Neurology** 2012 Jul 3;79(1):e1-4). A pre-existing structural lesion is not ruled out in this case, but more effective therapies for febrile status epilepticus might prevent the development of cerebral edema, brain atrophy, and persistent hemiplegia.