(Lombroso CT, Lerman P. Pediatrics 1967;39:563-581). However, the absence of cyanosis in BHS is disputed by some authorities (Livingston S. Comprehensive Management of Epilepsy in Infancy, Childhood and Adolescence. Springfield, IL, Charles C Thomas, 1972). So-called *pallid BHS* are precipitated by trauma rather than anger, and are associated with cardiac asystole and EEG slowing. The pathophysiology of the pallid BHS is vagal cardiac inhibition and cerebral anoxia, and treatment with atropine may be beneficial. Under carefully controlled EEG and cardiac monitoring, the diagnosis has been confirmed by ocular compression (Breningstall GN. Pediatr Neurol 1996;14:91-97). A fall in arterial oxygen saturation has been demonstrated during an attack (Gauk EW et al. N Engl J Med 1963;268:1436-1441). An iron deficiency anemia found in 23% of cases of BHS is suggested as a possible contributing causative factor (Holowach J, Thurston DL. N Engl J Med 1963;268:21-23; Ped Neur Briefs May 1997;11:33). Autonomic dysregulation is a common mediating mechanism for both pallid and cyanotic BHS, resulting in loss of consciousness (DiMario FJ Jr, Burleson JA. Pediatr Neurol 1993;9:268-274).

As demonstrated in the above study and in many previous clinical reports, although a BHS is a frightening disorder for parents, the prognosis is invariably benign, attacks usually ending by age 5 years. The differential diagnosis includes a cardiac pathology with prolonged QT interval or an epilepsy. An EKG is recommended in children with pallid BHS, and an EEG in those with prolonged convulsive movements. In BHS the EEG is normal and treatment with conventional antiepileptic drugs is ineffective. Treatment requires parental counseling: 1) frequent reassurance about the benign nature of the spells, 2) an explanation of the strong genetic factor, with an autosomal dominant inheritance and equal male to female ratio, and 3) the risks of a tendency to syncopal attacks and behavior disturbance in later childhood. A trial of iron therapy in patients with a lowered hemoglobin level may be beneficial. Of 33 children treated with ferrous sulfate orally (5 mg/kg/day for 16 weeks) 88% had a complete or partial control of BHS, whereas in 34 receiving placebo only 6% resolved (Daoud AS et al. J Pediatr 1997;130:547-550; Ped Neur Briefs 1997;11:33).

SEIZURE DISORDERS

RISK OF EPILEPSY AFTER COMPLEX FEBRILE SEIZURES

The risk of epilepsy developing after complex febrile seizures (CFS) was studied in 477 children admitted between 1991 and 1998 with febrile convulsions at Tel Aviv Medical Center, Israel. Among 57 (12%) diagnosed with CFS and an otherwise normal neurologic exam, 48 were available for follow-up, and 13 (27%) developed epilepsy. Thirty percent had a family history of febrile seizures, but none had a positive family history of epilepsy. The follow-up period was 8-78 months (mean 43 +/- 24 months). Among patients developing epilepsy, the mean age of CFS onset was 11 months compared to 17 months in those without epilepsy. CFS of the partial seizure type had a higher risk of epilepsy (45%) than those with multiple febrile seizures (21%). None of 3 patients with the prolonged type of CFS developed epilepsy at follow-up. Neuroimaging studies performed in 15 patients were all normal, and 10 had partial CFS. Two of the 13 patients with epilepsy treated with anticonvulsants were refractory. (Sapir D, Leitner Y, Harel S, Kramer U. Unprovoked seizures after complex febrile convulsions. Brain Dev Dec 2000;22:484-486). (Respond: Dr Uri Kramer, Child Development Center, Beit Habriut Strauss, 14 Balfour Street, Tel Aviv 65211, Israel).

COMMENT. In this study, 27% of children with complex febrile seizures (CFS) developed epilepsy at follow-up. Of the 3 characteristics that define a CFS (prolonged duration >15 minutes, focal partial pattern, or multiple recurrence with a single fever episode), the partial CFS patient carried a higher risk of developing epilepsy (45%) than patients with multiple seizures (21%). Only 3 of the 57 patients with CFS had prolonged FS and none developed epilepsy. In some previous studies, the risk of epilepsy following CFS has been lower, reported at 4-15% (Nelson & Ellenberg,1976; Verity & Golding, 1991). Berg and Shinnar (1996) found a strong correlation between prolonged CFS and focal features. In my own prospective studies and reports (Millichap, 1960-1968), prolonged FS, an abnormal EEG, and frequent recurrence of FS were associated with an increased risk of epilepsy. Focal EEG abnormalities were more predictive of epilepsy than focal FS patterns. Spontaneous seizures and epilepsy developed in 29% of my patients with CFS over a 2 year follow-up, a figure similar to that reported above by Sapir et al, but the prolonged FS duration was more predictive of epilepsy than the focal pattern. For further reference to FS and epilepsy, see Millichap JG, Progress in Pediatric Neurology III, PNB Publ, 1997;pp19-36; and Febrile Convulsions, MacMillan, 1968).

SEIZURE PRECIPITANTS AND EPILEPSY SYNDROMES

The prevalence of various seizure precipitants in relation to different epilepsy syndromes and patient age and gender was determined by a questionnaire survey of 400 patients attending (Dec 1998-May 1999) the Comprehensive Epilepsy Program, University of Virginia, Charlottesville. The mean age of the pediatric and adult patients was 25 +/- 16 years; 200 were male and 200 female. Specific ILAE epilepsy syndromes were defined in 309 (77%). At least one specific seizure precipitant was identified by 247 (62%) patients, the most frequent being *stress*, in 30%. *Sleep deprivation* was invoked by 18%, *sleep* in 14%, *fever or illness* (excluding simple febrile convulsions) in 14%, and *fatigue* in 13%. Other less frequent precipitants included heat and humidity (9%), flashing lights (4%), caffeine (2%), fasting (2%), and alcohol in only 0.5%. Rare precipitants were physical discomfort or pain in 3 patients, exertion in 2, awakening (2), light (2), odors (1), laughing (1), noise (1), and dietary triggers (1). No precipitant was recognized by 38% of patients.

Stress, fatigue, and sleep deprivation were positively correlated, and these precipitants were reported especially in patients with symptomatic localization-related epilepsies; 46% of patients with temporal lobe epilepsy (TLE) identified stress compared to 15% of those with cryptogenic generalized epilepsy (CGE). Sleep was negatively correlated with other precipitants, and was invoked in 66% of patients with idiopathic partial, extratemporal epilepsy compared to 4% of those with TLE. Extralimbic seizures (autosomal dominant frontal lobe epilepsy) are nocturnal and occur especially during sleep whereas limbic seizures (TLE) are diurnal and occur in wakefulness. Menstrual effects were particularly prominent in women with TLE (28%), more than twice the percentage in women with other epilepsy syndromes. (Frucht MM, Quigg M, Schwaner C, Fountain NB. Distribution of seizure precipitants among epilepsy syndromes. Epilepsia December 2000;41:1534-1539). (Reprints: Dr Mark S Quigg, Department of Neurology, Box 394, Charlottesville, VA 22908).

COMMENT. The recognition and avoidance of specific seizure precipitants is an important aspect of the management of epilepsy. Stress, sleep deprivation, and fatigue are endogenous seizure precipitants that appear to act through common