CT scan, the history and neurologic examination are abnormal, pointing to a neurologic cause with symptoms and signs of raised intracranial pressure. The value of a thorough neurologic history and examination outweighs that of a CT scan. In patients with primary headaches, the family history is frequently positive for migraine and provides reassurance in a decision to defer neuroimaging of young children with headache in the ED.

**Brain imaging in children referred to a pediatric neurology clinic for headache** was also found of very limited value, in a retrospective study of 133 patients ages 3 to 18 years at Schneider Children’s Hospital, NY. (Maytal J et al. *Pediatrics* 1995;96:413-416). The indications for brain imaging in 78 patients were atypical headache pattern in 12, parental concern in 12, physician concern about cerebral tumor in 11, systemic symptoms of fatigue and weight loss in 11, focal symptoms or signs during headaches in 7, neurologic or ocular abnormalities in 6, increasing severity or frequency of headache in 5, and unspecified in 17. Abnormal scans in 11 (14%) patients included evidence of chronic sinusitis in 7, neuroepithelial cyst near the foramen of Monroe in 1, temporal lobe arachnoid cyst in 1, cerebral hemiatrophy in 1, and Dandy-Walker malformation in 1. None of the scans showed lesions requiring neurosurgical intervention. MRI indications proposed by the authors include 1) atypical recurrent headaches, 2) recent change in character of headache, 3) persistent vomiting, 4) abnormal neurologic findings, and 5) occurrence in younger age groups. Headache associated with abnormal EEG should also be considered for MRI. In a young child with recurrent headache seen by a neurologist in a single consultation, without prospect of follow-up, deferral of imaging may not be practical or judicious.

**SEIZURE DISORDERS**

**SINGLE-PULSE ELECTRICAL STIMULATION IN IDENTIFICATION OF EPILEPTOGENIC CORTEX**

Single-pulse electrical stimulation (SPES) was evaluated in 35 children who underwent intracranial subdural electroencephalographic (EEG) monitoring at Great Ormond Street Hospital for Children and King’s College Hospital, London, UK. Median age was 14 yrs 2 mos (range 9 mos to 17 yrs 7 mos). Using a series of 10 or more single, brief (1 ms) electrical stimuli, the cortical responses were examined for associations between response type, ictal onset zone, lesion boundary, and seizure outcome. Studies were conducted during interictal periods, while the patient was awake, and in parallel with video-EEG. Subdural grids (in 25 patients), subdural strips (in 30 patients), or depth electrodes (9 patients) covered a number of areas in each patient. The median number of electrodes in each patient was 54 (range 17-78). Abnormal responses to SPES indicative of epileptic cortical excitability were present in 54% of cases, and were “delayed”(DR) or “repetitive”(RR) in type. The DR is a sharp wave or spike, occurring later than 100 ms after stimulus, and corresponding with the area of seizure onset. The RR has the form of a successive repetition of an early response (ER), a sharp wave followed by a slow wave, typically lasting for a second or longer. Removal of the entire area responsible for abnormal responses to SPES was associated with good outcomes. (Flanagan D, Valentin A, Seoane JLG, Alarcon G, Boyd SG. Single-pulse electrical stimulation helps to identify epileptogenic cortex in children. *Epilepsia* July
COMMENT. Cortical responses to SPES in children are similar to those observed in adults. Abnormal SPES responses (DRs and RRs) correlate with epileptogenic cortex, and are useful in the presurgical evaluation and positioning of electrodes. The method may be used with or without anesthesia (Valentin et al, King’s College Hospital, unpublished observation).

KETOGENIC DIET AND HORMONAL THERAPY IN PREVENTION OF EVOLUTION OF WEST SYNDROME TO LENNOX-GASTAUT

Medical records of 98 patients diagnosed with West syndrome and monitored at Sanggye Paik Hospital, Seoul, Korea, for at least 3 years were retrospectively reviewed to assess etiology, age at onset, value of various therapies, and the rate of evolution from West syndrome to Lennox-Gastaut syndrome. During follow-up, West syndrome evolved to Lennox-Gastaut syndrome in 48 of the 98 (49%) patients. Etiology of West syndrome was cryptogenic in 36 (36.7%) and symptomatic in 62 (63.3%). Patients with normal psychological development before seizure onset were excluded. All patients had hypsarrhythmia and infantile spasms. Treatment of West syndrome varied as follows: antiepileptic drugs in 31 patients, ketogenic diet in 33, prednisolone in 45, ACTH in 15, surgery in 3, and herbal medication or no treatment in 4 patients. Age at onset of seizures (mean, 5.8 +/- 2.4 mos) or disease etiology was not related to development of Lennox-Gastaut syndrome. Risk of evolving to Lennox-Gastaut syndrome in patients treated with anticonvulsant drugs was 17 in 31 (55%). Risk was significantly lower in patients treated with ketogenic diets (10/33, 30%) or hormonal therapy (26/60, 43%) or a combination of both (7/27, 26%), (P<0.05). (You SJ, Kim HD, Kang H-C. Factors influencing the evolution of West syndrome to Lennox-Gastaut syndrome. Pediatr Neurol Aug 2009;41:111-113). (Respond: Dr Kang, 134 Shinchon Dong, Seodaemun Gu, Seoul 120-752, Korea. E-mail: hipo0207@yuhs.ac).

COMMENT. The ketogenic diet, prednisolone or ACTH or a combination of both diet and hormonal therapy may prevent the development of encephalopathy and Lennox-Gastaut syndrome in patients with West syndrome. It is estimated that 20-50% of West syndrome cases evolve to Lennox-Gastaut syndrome; 70-80% of Lennox-Gastaut cases have no history of West syndrome. The relatively higher risk of development of Lennox-Gastaut after West syndrome in the present study may be related to the exclusion of West syndrome patients with normal neuropsychological development prior to onset of infantile spasms. That the evolution to Lennox–Gastaut syndrome is significantly reduced by treatment with ketogenic diet, hormonal therapy or both suggests that these therapies may modify the underlying encephalopathic process in West and Lennox-Gastaut syndromes. Age and brain maturation are not the primary factors in the development of Lennox-Gastaut syndrome following West syndrome.