acceleration (152% +/-14%), but no increase was observed after levetiracetam (101%+/-14%). (Sohn YH, Jung HY, Kaelin-Lang A, Hallett M. Effect of levetiracetam on rapid motor learning in humans. Arch Neurol Dec 2002;59:1909-1912). (Respond: Mark Hallett MD, National Institute of Neurological Disorders and Stroke, National Institutes of Health, Bldg 10, Room 5N226, 10 Center Dr, MSC 1428, Bethesda, MD 20892).

COMMENT. Levetiracetam, a suppressant of motor cortex excitability, may interfere with rapid motor learning when administered to normal volunteers in a single daily dose. Previous studies (cited by the authors) have shown that this adverse effect on practice-related motor cortex plasticity is not observed with the AED, lamotrigine, although it too suppresses motor cortex excitability. These results may not apply to patients receiving long-term AED therapy.

In a clinical study of the effectiveness of levetiracetam in partial epilepsies, patients who were older at the onset of seizures did better than those with younger onset (51 vs 27 years). Also, patients with temporal lobe seizures were better controlled than those with frontal lobe localization (Bazil CW, Rose A, Resor S et al. Levetiracetam may be more effective for late-onset partial epilepsy. Arch Neurol Dec 2002;59:1905-1908).

SEIZURE DISORDERS

LEARNING AND MEMORY IN CHILDREN WITH EPILEPSY

The relation between learning and memory and epilepsy in school children with recently diagnosed idiopathic and/or cryptogenic seizures was evaluated at Wilhelmina Children's Hospital, the Netherlands. Word span (imageable nouns) and location learning of colored pictures were assessed within 48 hours after diagnosis of epilepsy and 3 and 12 months later, in 69 school children with epilepsy (aged 9.1 years, SD 2.7) and 66 classmates. Patients and controls performed similarly in registration, recall, and retention. Under conditions of increased demand on working memory (reproducing words in reverse order), patients recalled slightly less than controls; 54% of the epilepsy group, cf 26% of healthy classmates, under-performed in one or other aspects of tasks. Emotional reactions of parent and child to the onset and poor control of epilepsy contributed to impaired memory. Children with a favorable response to therapy did not differ from controls, while those with 6 months of refractory seizures had worse memory spans backwards than controls. School children with new onset idiopathic or cryptogenic epilepsy are vulnerable when processing memory tasks, particularly in tasks of increased demand and when seizures are poorly controlled. (Schouten A, Oostrom KJ, Pestman WR et al. Learning and memory of school children with epilepsy: a prospective controlled longitudinal study. Dev Med Child Neurol Nov 2002;44:803-811). (Respond: Jennekens-Schinkel A. PhD, University Medical Centre, Wilhelmina Children's Hospital, Hp KG 013271, PO Box 85090, 3508 AB Utrecht, the Netherlands).

COMMENT. School children with newly diagnosed epilepsy can retain normal learning and memory, but when task difficulty is increased or material reversed, memory may be impaired. This vulnerability to memory impairment is enhanced when patients and parents cannot adapt to the diagnosis of epilepsy or when seizures are poorly controlled. In

individual cases, memory under-performance is neither consistent nor persistent, but it is twice as frequent as among controls.

The recognition of these memory impairments should prompt academic accommodations and a more structured classroom environment. Previous investigators have reported that ongoing seizures may impair school performance and memory and learning. Memory deficits and underachievement may be transient (Deonna et al. 2000), or persistent (Austin et al. 1999).

EPILEPSY AND FRAGILE X SYNDROME

The seizure history of 136 patients with fragile X syndrome (FXS), (age range 2 to 51 years; 113 males and 23 females), were reviewed at RUSH-Presbyterian-St Luke's Medical Center, Chicago, IL. Seizures occurred in 13.3% of males with FXS and 4.3% of females; and the majority were partial seizures. Epileptiform EEGs were found in 77% individuals with seizures and 23% of those without, most commonly centrotemporal spikes. Seizures were easily controlled in 14 of 16 treated. Benign rolandic epilepsy was the most common epilepsy syndrome in FXS patients, all of whom showed remission. A specific relation between absence of fragile X mental retardation protein and benign rolandic epilepsy with centrotemporal spikes is suggested. (Berry-Kravis E. Epilepsy in fragile X syndrome. Dev Med Child Neurol Nov 2002;44:724-728). (Respond: Elizabeth Berry-Kravis MD PhD, Department of Pediatrics, Neurology, and Biochemistry, RUSH-Presbyterian-St Luke's Medical Center, 1725 West Harrison, Suite 718, Chicago, IL 60612).

COMMENT. Complex partial seizures are the most common seizure type and benign rolandic epilepsy with centrotemporal spikes in the EEG the most common epilepsy syndrome in patients with fragile X syndrome. Seizures in FXS are easily controlled with anticonvulsants and are usually limited to childhood. Seizures are less frequent in girls with FXS than in boys, attesting to the milder phenotypic manifestations of FXS in girls.

Fragile X carrier state may underlie a diagnosis of essential tremor in older male patients, according to a report of 2 fragile X carriers (ages 68 and 63 years) seen at the University of Colorado, Denver, and University of Toronto, Ontario (Leehey MA, Munhoz RP, Lang AE et al. Arch Neurol January 2003;60:117-121). In addition to a disabling intention tremor, the patients had impaired tandem gait, generalized brain atrophy with frontal psychological deficits, and T2 middle cerebellar hyperintensities on MRI. Both had elevated FXMR gene 1 messenger RNA and reduced FXMR 1 protein.

GENETICS OF ABSENCE EPILEPSY AND FEBRILE SEIZURES

In a large family with epilepsy studied at the University of Melbourne, Australia, FS in 18 children were inherited as autosomal dominant with 75% penetrance (GABA receptor subunit mutation on chromosome 5), and absence epilepsy in 8 required the GABA gene on chromosome 5 interacting with a possible further gene on chromosomes 10, 13, 14 and 15. (Marini C, Harkin LA, Wallace RH et al. Brain Jan 2003;126:230-240).