

placed more on the manufacturer of hazardous video games and regulatory agencies than on the consumer, however.

HOT WATER EPILEPSY

The clinical and electroencephalographic features of 21 patients with hot water epilepsy (HWE) have been analysed at the University of Istanbul, Turkey. The male:female ratio was 3:1. Age at onset of seizures ranged from 19 months to 27 years (mean, 12 years). Seizures were precipitated by pouring water over the head in 14 cases or by bathing in hot water. Nine expressed a pleasurable feeling during the seizures. Seizures were partial in 20, with secondarily generalized in 8, and generalized in 1. Spontaneous seizures also occurred in 62%. EEGs were abnormal in 8, with focal temporal epileptiform discharges. Neuroimaging was normal in 12 patients studied. Seizures were controlled by avoiding hot water in 7 patients. One patient who induced seizures compulsively was treated with carbamazepine (CBZ). The remaining patients responded to CBZ or valproate. (Bebek N, Gurses C, Gokyigit A et al. Hot water epilepsy: clinical and electrophysiologic findings based on 21 cases. Epilepsia Sept 2001;42:1180-1184). (Reprints: Dr C Gurses, University of Istanbul, Istanbul Faculty of Medicine/Dept Neurology, Millet cad 34390 Capa-Istanbul, Turkey).

COMMENT. Hot water epilepsy (HWE), a rare form of reflex epilepsy, has a male preponderance, may be self induced, often for pleasure, and shows temporal lobe localization of EEG epileptiform discharges. It is generally self-limited, controlled by avoiding hot water-head baths or showers, but antiepileptic medication is sometimes required.

HWE is a regional religious custom in Southern India, where 279 patients were studied between 1980-83. Ages ranged from 8 mos to 58 years, 28% below 6 years. Only 7% had febrile convulsions. Complex partial seizures occurred in 67% and generalized tonic-clonic seizures in 33%. Spontaneous seizures also occurred in 30%. A positive family history of epilepsy was obtained in 22% and for HWE in only 7%. (Satishchandra P et al. Epilepsia 1988;29:52-56). See Progress in Pediatric Neurology, PNB Publishers, 1991;pp43-44.

VIGABATRIN-ASSOCIATED VISUAL FIELD DEFECTS

The prevalence, risk factors, and long-term outcome of vigabatrin-associated visual field defects were examined in 60 adult patients with partial epilepsy treated with vigabatrin for 7 months to 14 years at the University of Kuopio Hospital, Finland. At first examination, bilateral concentric defects occurred in 24 (40%) of 60 patients; they were severe in 8 (13%), and a mild constriction in 16 (27%). Repeated kinetic Goldmann perimetries and follow-up examination performed after 4 to 38 months (mean, 15 mos) in 55 patients (29 having discontinued therapy) revealed no reversion or progression in visual field constriction. (Nousiainen I, Mantjarvi M, Kalviainen R. No reversion in vigabatrin-associated visual field defects. Neurology Nov (2 of 2) 2001;57:1916-1917). (Reprints: Dr Iiris Nousiainen, Department of Ophthalmology, Kuopio University Hospital, POB 1777, 70211 Kuopio, Finland).

COMMENT. The age of the patients and high cumulative doses of vigabatrin employed could be risk factors for the poor outcome and severe limiting effect on daily activities in adults. In a young patient, recovery of visual fields has been reported after early withdrawal of vigabatrin (Giordano L et al. Neurol Sci 2000;21:185-186). Duration of vigabatrin treatment (but not the dose or age of child) was correlated with visual field constriction in 65% of 17 children

examined by perimetry at Shaare Zedek Medical Center, Jerusalem (Gross-Tsur V et al. 2000, reviewed in *Ped Neur Briefs* July 2000;14:49). VEPs and ERGs can be useful indicators of visual function in young or retarded patients when perimetry is inappropriate. Unless the effect on visual fields can be prevented, this significant risk seems to outweigh the benefits of using vigabatrin for the treatment of infantile spasms.

MOVEMENT DISORDERS AND ADHD

EYE MOVEMENTS IN TOURETTE SYNDROME & COMORBID ADHD

The effect of comorbid ADHD on oculomotor abnormalities in boys with Tourette syndrome (TS) was determined using three saccade tasks to examine the planning and execution of eye movements in a study at the Kennedy Krieger Institute and Department of Neurology, Johns Hopkins School of Medicine, Baltimore, MD. Subjects in 3 groups, ages ranging from 7.8-14.6 years (means 10-11 yrs) included 14 with TS-only, 11 with TS+ADHD, and 10 controls. Eye movements were recorded by electro-oculography, and maximum saccadic velocities, amplitudes, and latencies were computer analysed.

Latency of prosaccades (measuring ability to initiate saccades to an unpredictable peripheral visual stimulus) was prolonged in both patient groups (TS-only and TS+ADHD) compared to controls, which indicates that TS is associated with delay in initiation of oculomotor responses. Response inhibition errors on antisaccade (ability to inhibit a prosaccade) tasks (directional errors) and memory-guided saccade task (anticipatory errors) were increased in boys with TS+ADHD compared to the TS-only group, suggesting that comorbid ADHD is associated with deficits in response inhibition and excessive variability in motor response to a visual stimulus. Accuracy of memory guided saccades was not significantly different in the three group. (Mostofsky SH, Lasker AG, Singer HS, Denckla MB, Zee DS. Oculomotor abnormalities in boys with Tourette syndrome with or without ADHD. *J Am Acad Child Adolesc Psychiatry* December 2001;40:1464-1472). (Respond: Dr Stewart H Mostofsky, Developmental Cognitive Neurology, Kennedy Krieger Institute, 707 North Broadway, Baltimore, MD 21205).

COMMENT. This is the first report of the impact of comorbid ADHD on the execution of eye movements. Whereas TS results in slowed oculomotor responses to a visual stimulus, ADHD is associated with an increased variability of response. This finding is consistent with excessive variability in reaction time demonstrated by continuous performance tasks in children with ADHD. The authors emphasize the importance of treating comorbid conditions, especially ADHD, in children with tic disorders. ADHD is reported to occur in more than 50% of patients with TS. See *Ped Neur Briefs* Nov 2001 for article on increased risk of cognitive deficits and behavioral disturbance in children with tic disorders complicated by ADHD.

In a previous report of the control of volitional and reflexive saccades in 10 subjects with Tourette's syndrome and 10 controls (LeVasseur AL et al. *Brain* 2001;124:2045-2058) from Queen's University, Kingston, Ontario, Canada, saccadic reaction times were longer, saccadic amplitudes were smaller, but the occurrence of direction errors was normal in the immediate antisaccade task. The ability to inhibit reflexive saccades towards novel stimuli was not impaired in TS. Timing errors were significantly greater in TS compared to controls, indicating that the ability to inhibit planned motor programs is significantly impaired. Altered cortical-basal ganglia circuitry leading to reduced cortical inhibition may explain the inability of TS subjects to delay execution of motor responses. Four of