patients in the one open study providing EEG data. Seizures were mentioned in 4 of the studies, affecting 13 plus patients, but the total number of patients affected was not given. The frequency of abnormal EEGs in these patients is considerably higher than that usually reported for ADHD. It seems that CBZ might be indicated for the treatment of ADHD symptoms in some patients with abnormal EEGs and/or a history of seizures. On a negative note, see Progress in Pediatric Neurology II, 1994, pp188-190, for references to cognitive impairment and impulsivity caused by CBZ treatment of epilepsy.

The cognitive effects of carbamazepine, phenobarbital, and valproate were compared in 73 children with newly diagnosed epilepsy studied at the National Cheng Kung University; Chi Nei Hospital; and Tainan Municipal Hospital, Tainan, Taiwan, ROC. (Chen Y-J, Kang W-M, Chin-Min So W, Epilepsia 1996;37:81-86). Only children treated with phenobarbital showed increased P300 latencies on auditory event-related potentials, which was inversely related to IQ scores after treatment for 6 to 12 months. WISC-R IQs and Bender-Gestalt scores were not significantly different in any of the groups before or after treatment. P300 latency was a more sensitive indicator of AED effects on cognitive function than the WISC-R and Bender-Gestalt.

Behavioral side effects of Gabapentin are reported in 7 children with base-line ADHD and developmental delays who were followed in the epilepsy program and in the Department of Child Psychiatry, Emory University, Atlanta, GA. (Lee DO et al. Epilepsia 1996;37:87-90). Tantrums, aggression, hyperactivity, and defiance were the most troublesome symptoms. The majority (64%) of intensified behaviors were similar to baseline ADHD symptoms; 21% were ODD and 8% were CD symptoms. New behaviors, not exhibited before gabapentin therapy, were ODD or CD. Behavioral side effects resolved after decrease or withdrawal of gabapentin.

BRAIN INJURY IN INFANCY AND LEARNING DISABILITIES

Dyscalculia and dyslexia in a 17-year-old boy after right hemisphere injury in infancy is reported from the Division of Neurosurgery and Department of Pediatrics, University of Maryland, Baltimore; and Cognitive Neuroscience Section, National Institutes of Health, Bethesda, MD. Social behavior was normal, but math and spelling abilities were impaired and his attention span was short. A functional MRI showed predominantly left hemisphere activation involving frontal and posterior parietal regions while the patient performed calculations. In normal subjects this test produced bilateral activation of the supramarginal gyrus. These MRI findings were consistent with early interhemisperic transfer of right parietal visuospatial skills to the left parietal region. Dyscalculia and dyslexia with normal IQ suggest an acquired left parietal dysfunction caused by competition for left hemisphere representation between verbal and visuospatial functions. (Levin HS et al. Dyscalculia and dyslexia after right hemisphere injury in infancy. Arch Neurol Jan 1996;53:88-96). (Reprints: Dr Grafman, Cognitive Neuroscience Section, NIH/NINDS/MNB, Bldg 10, Room 5S 209, 10 Center Dr, MSC 1440, Bethesda, MD 20892).

COMMENT. The authors conclude that interhemispheric reorganization of function and language may be bidirectional and not only a left hemisphere feature of language development. The MRI showed an intact left hemisphere following the injury. Visuospatial functions normally subserved by the right parietal area were probably transferred to the left parietal region, causing a crowding effect and disproportionate impairment of reading and math skills

in relation to his other cognitive abilities.

MOVEMENT DISORDERS

MRI CHANGES IN SYDENHAM'S CHOREA

Cerebral MRIs of 24 children with Sydenham's chorea and 48 matched controls were compared at the National Institutes of Health, Bethesda, MD. The caudate, putamen, and globus pallidus in the chorea group were all significantly greater in volume, whereas the total hemispheres, prefrontal, midfrontal, or thalamus areas were not increased. (Giedd JN et al. Sydenham's chorea: Magnetic resonance imaging of the basal ganglia. Neurology Dec 1995;45:2199-2202). (Reprints: Dr Jay N Giedd, National Institutes of Health, NIMH, Child Psychiatry Branch, 9000 Rockville Pike, Building 10, Room 6N240, Bethesda, MD 20892).

COMMENT. A cross-reactive antibody-mediated inflammation of the basal ganglia is suggested as the pathophysiology of Sydenham's chorea. The authors admit that volumetric MRI is of limited diagnostic value because of large variability and overlap in basal ganglia size between chorea and control subjects.

Chorea in an infant with holoprosencephaly is reported from the College of Physicians and Surgeons, New York. (Louis ED et al. Pediatr Neurol 1995;13:355-357). MRI showed small, fused frontal lobes with hypoplastic caudate nuclei. This example of chorea associated with a congenital structural anomaly and undersized basal ganglia contrasts with the inflammatory hyperplasia of the caudate in Sydenham's chorea.

NEUROLOGIC SEQUELAE OF DANCING EYE SYNDROME

A persisting disability was found at long-term follow-up in 88% of 54 patients with dancing eye syndrome (DES) reported from the Hospital for Sick Children, Great Ormond Street, London. The disability was severe in 30 (62%), 34 (69%) had a motor disability, 29 (59%) had learning disabilities, and 23 (47%) had a combined motor and learning disability. Neurologic sequelae were independent of the severity of symptoms of the illness and age at onset. A malignancy was diagnosed in only 4: neuroblastoma in 3 and acute lymphoblastic leukemia in 1. An intercurrent illness, usually respiratory, preceded onset of DES in one half the cases. Presenting symptoms included ataxia, abnormal head and limb movements, and opsoclonus. Emotional outbursts of temper and affection were later features. A favorable initial response to corticotrophin or predisolone, observed in all patients, was not predictive of a good neurological prognosis. (Pohl KRE, Pritchard J, Wilson J. Neurological sequelae of the dancing eye syndrome. Eur J Pediatr March 1996;155:237-234). (Respond: Dr KRE Pohl, Newcomen Centre, Guys Hospital, St Thomas's Street, London SE1 9RT, UK).

COMMENT. Dancing eye syndrome (opsoclonus-myoclonus, infantile myoclonic encephalopathy) presents in infancy or early childhood (93% under 3 years) and neurologic sequelae may persist into adult life. Speech deficits, described as occasional in the above series, were more prominent in patient series reported from the Children's Memorial Hospital, Chicago, and the Children's National Medical Center, Washington, DC. (see Ped Neur Briefs Jan 1996;10:2).