

and vigilance and standardized for American children, is evaluated for use among Swedish children. (El-Sayed E, van't Hooft I, Fried I et al. Acta Paediatr Nov 1999;88:1262-1268). Statistically significant differences are found between ADHD children and age-matched control children.

TRIAL OF CHOLINERGIC AGONIST IN ADULTS WITH ADHD

A novel cholinergic activating agent, ABT-418, was used to treat 29 adults with ADHD, in a double-blind, placebo-controlled, randomized, crossover trial at the Pediatric Psychopharmacology Clinic, Massachusetts General Hospital, Boston. A significantly higher proportion of patients was benefited by ABT-418 in a transdermal patch (75 mg/day) compared to placebo (40% versus 13%). ADHD symptom checklist scores were also significantly reduced (28% versus 15%). Inattentiveness scores and patients with less severe ADHD responded more frequently. Dizziness, skin irritation, nausea, and headaches were the most common adverse effects, and necessitated dose reduction in 6 patients and withdrawal in 1. (Wilens TE, Biederman J, Spencer TJ et al. A pilot controlled clinical trial of ABT-418, a cholinergic agonist, in the treatment of adults with attention deficit hyperactivity disorder. Am J Psychiatry Dec 1999;156:1931-1937). (Respond: Dr Wilens, Pediatric Psychopharmacology Clinic, ACC 725, Massachusetts General Hospital, Boston, MA 02114).

COMMENT. ABT-418, a nicotinic analog, may be a useful new agent in the treatment of ADHD, and especially in patients with the inattentiveness subtype (ADD) and those with less severe symptoms. The results of this trial provide further evidence of a link between nicotinic-cholinergic agents, catecholamine function, and improvement in ADHD.

EEG operant conditioning (neurotherapy) to treat ADHD is reviewed by Nash JK (Clin Electroencephalogr January 2000;31:30-37). Studies suggest that clinical improvement in ADHD is related to measurable improvements in the EEG, with a decline in theta/beta ratios over the frontal/central cortex and/or reduced theta/alpha band amplitudes.

SEIZURE DISORDERS

KETOGENIC DIET IN TREATMENT OF EPILEPTIC APHASIA

Three children, ages 9, 12 and 14 years, with acquired epileptic aphasia refractory to treatment with antiepileptic drugs, ACTH and prednisone, responded to the ketogenic diet for 12 to 26 months, in a trial at the Children's Hospital of Philadelphia, and University of Pennsylvania School of Medicine, PA. Improvements in language, and lessening of seizures and EEG seizure discharges occurred in all patients. Social interactions and behavior also improved significantly. (Bergqvist AGC, Chee CM, Lutchka LM, Brooks-Kayal AR. Treatment of acquired epileptic aphasia with the ketogenic diet. J Child Neurol Nov 1999;14:696-701). (Respond: Dr AG Christina Bergqvist, Division of Neurology, The Children's Hospital of Philadelphia, Wood Building 6th Floor, 34th and Civic Center Blvd, Philadelphia, PA 19104).

COMMENT. The ketogenic diet is presented as a new therapeutic alternative for patients with Landau-Kleffner syndrome. The results are promising and the method deserves further study in this frequently resistant epileptic disorder.

Language dominance in epilepsy patients is studied using functional MRI at the Medical College of Wisconsin, Milwaukee, WI. (Springer JA, Binder JR, Hammeke TA et al. Brain Nov 1999;122:2033-2045). Compared to normal subjects considered left hemisphere dominant in 94%, there was greater variability of language dominance in the epilepsy group, 78% showing left hemisphere dominance, 16% a symmetric pattern and 6% right hemisphere dominance. Atypical language dominance in the epilepsy group was correlated with an earlier age of brain injury and with weaker hand dominance.

BEHAVIORAL EFFECTS OF CARBAMAZEPINE

The cognitive and behavioral effects of carbamazepine (CBZ) were evaluated in 10 children (ages 6 to 12 years) with benign rolandic epilepsy at Columbia Presbyterian Medical Center, New York, and Children's Hospital Los Angeles, CA. Controls were 14 unmedicated children with migraine. The mean CBZ serum level was 6.4 mcg/ml (range, 3.7-11.6). Higher CBZ blood levels were associated with slower performances on Trail Making Test A, a visual-search task, but the difference was not significant. Significant group differences on psychometric tests were limited to poorer performance on Wide Range Assessment of Memory and Learning Story Memory and Trail Making Test A. Two subjects had particularly poor scores while treated with CBZ. Significant practice effects in repeat testing emphasized the need for controls. (Seidel WT, Mitchell WG. Cognitive and behavioral effects of carbamazepine in children: data from benign rolandic epilepsy. J Child Neurol Nov 1999;14:716-723). (Respond: Dr William T Seidel, Babies and Children's Hospital, 11 North Room 8, 3959 Broadway, New York, NY 10039).

COMMENT. Children treated with carbamazepine may suffer some adverse effects on memory, and the effects on behavior and cognition should be monitored closely.

See Progress in Pediatric Neurology II, PNB Publ, 1994;pp187-189, and III, 1997;pp146-151, for further articles on the cognitive and behavioral effects of carbamazepine. In one study involving 64 children with epilepsy, serum levels of CBZ showed a significant negative correlation with scores on 5 memory tests, after 6 months treatment and at 1 year (Forsythe I et al. Dev Med Child Neurol 1991;33:524-534).

STRUCTURAL BRAIN ABNORMALITY IN JME

An automated and objective technique of statistical parametric mapping was employed in the analysis of structural MRI from 20 patients with juvenile myoclonic epilepsy (JME) and 30 control subjects studied at the Institute of Neurology, London, UK. An increase in cortical grey matter was observed in the mesial frontal lobes of JME patients. Significant structural abnormalities in cortical grey matter were present in 5 of 20 JME patients studied individually; 2 had bilateral areas of increased grey matter volume in temporoposterior and mesioparietal regions, while 3 had decreased grey matter volume in frontal areas. (Woermann FG, Free SL, Koeppe MJ, Sisodiya SM, Duncan JS. Abnormal cerebral structure in juvenile myoclonic epilepsy demonstrated with voxel-based analysis of MRI. Brain Nov 1999;122:2101-2107). (Respond: Professor JS Duncan, National Society for Epilepsy, National Hospital for Neurology and Neurosurgery, Chalfont St Peter, Gerrards Cross, Bucks SL9 0RJ, UK).

COMMENT. Juvenile myoclonic epilepsy, a syndrome of idiopathic generalized epilepsy, is shown to have a structural cause in some patients.