Wellcome Department of Cognitive Neurology, Queen Square, London, six right-handed male subjects with KS showed activation in the motor cortex both contralateral and ipsilateral to the voluntarily moved hand, although the contralateral activation was significantly stronger. Activation of the ipsilateral motor cortex in KS may be caused by sensory feedback from the involuntarily mirroring hand. (Krams M, Quinton R, Mayston MJ et al. Mirror movements in X-linked Kallmann's syndrome. II A PET study. Brain July 1997;120:1217-1228).

HEADACHE DISORDERS

PSEUDOMIGRAINE PLEOCYTOSIS SYNDROME

A series of 50 patients with a total of 164 episodes of pseudomigraine with temporary neurologic symptoms and CSF lymphocytic pleocytosis (PMP syndrome) is reported from the University Hospital Marques de Valdecilla, Santander, Spain. Onset was between 14 and 39 years, 68% in males. About one third had a past history of migraine, and one-quarter had a viral-like illness within 3 weeks of onset. A throbbing, bilateral headache lasting an average of 19 hours was associated with unilateral sensory symptoms (78% of episodes) of mean duration 5 hours, aphasic (66%) and motor (56%) symptoms, and visual symptoms in only 12% of episodes. Lymphocytic pleocytosis was 10 to 760 cells/mm3 (mean, 199). CSF protein was increased. Viral studies were negative. EEG showed focal slowing. Angiography in 12 patients was normal except one showing localized vasculitis. An aseptic inflammation of leptomeningeal vasculature is suggested as a possible cause. (Gomez-Aranda F, Canadillas F, Marti-Masso JF, et al. Pseudomigraine with temporary neurological symptoms and lymphocytic pleocytosis. A report of 50 cases. Brain July 1997;120:1105-1113). (Respond: Dr Julio Pascual, Service of Neurology, University Hospital Marques de Valdecilla, 39008 Santander, Spain).

COMMENT. This appears to be the largest series of patients reported with the syndrome of transient headache and CSF lymphocytosis. A previous report of 7 patients and review of 33 cases in the literature, 13 in children and adolescents, were included in Ped Neur Briefs, Oct 1995 (see Progress in Pediatric Neurology III, 1997;p178). The differential diagnoses listed by the authors (Berg MJ, Williams LS) included Lyme neuroborreliosis, neurosyphilis, neurobrucellosis, neoplastic meningitis, HIV meningitis, hemiplegic migraine, seizures, Mollaret's meningitis, and a side effect of angiography. The syndrome is self limited, and a viral etiology appears plausible.

ATTENTION DEFICIT AND LEARNING DISORDERS

LARGE CAUDATE NUCLEUS ON MRI IN ADHD

MRI measurements of the head of the caudate nucleus correlated with neuropsychological deficits and behavioral problems in 11 adolescents with ADHD in a study at the University of Barcelona, Spain. The ADHD group had a larger right caudate nucleus and a trend toward a larger left caudate than a control group of 19 healthy subjects. Larger caudate nuclei in controls were associated with poorer performance on tests of attention and higher ratings on the Conners Teachers Rating Scale. A L>R pattern of caudate asymmetry was present in the control group and a reverse pattern (R>L) for the ADHD subjects. A bilateral dysfunction is suggested for ADHD, more pronounced on

the right side. (Mataro M, Garcia-Sanchez C, Junque C, Estevez-Gonzalez A, Pujol J. Magnetic resonance imaging measurement of the caudate nucleus in adolescents with attention-deficit hyperactivity disorder and its relationship with neuropsychological and behavioral measures. Arch Neurol Aug 1997;54:963-968). (Reprints: Carme Junque PhD, Departament de Psiquiatria i Psicobiologia Clinica, Facultat de Psicologia, Universitat de Barcelona, Passeig de la Vall d'Hebron, 171 08035 Barcelona, Spain).

COMMENT. This study provides further evidence of a neuropathological or developmental structural defect underlying behavioral and cognitive abnormalities in adolescents with ADHD. Caudate volume normally decreases with increasing age, but in children with ADHD this maturational process is delayed or absent. These findings support the hypothesis of a frontal-striatal dysfunction in the mechanism of ADHD. (Progress in Ped Neurology III, 1997;p198, 212). Structural cerebral anomalies in ADHD reported previously have involved the corpus callosum (Semrud-Clikeman M et al. 1994), caudate nucleus and other regions (Castellanos FX et al. 1996), and the left temporal lobe (Millichap JG, 1997).

EFFECT OF METHYLPHENIDATE ON THE IMMUNE SYSTEM

The effects of methylphenidate (MPH) on the immune system was studied in laboratory mice and in 6 healthy boys treated for ADHD with 30-45 mg/day MPH at the Kings County Hospital, Brooklyn, NY. In mice, MPH (1, 5, or 10 mg/kg) reduced by up to 63% numbers of T-helper/inducer cells and also IgG+ cells in the spleen and increased up to 400-fold the serum levels of IgG (ELISA), both in a dose-dependent pattern. Three of 6 boys had twofold increases in IgE levels (188-285 IU/mL). MPH induced a marked hypersensitivity to mitogen-induced proliferation of lymphocytes, a hypergammaglobulinemia, and increased IgE levels. (Auci DL, Fikrig S, Rodriquez J. Methylphenidate and the immune system. J Am Acad Child Adolesc Psychiatry Aug 1997;36:1015-1016 (Letter to Editor). (Respond: Dr Auci, State Univ NY, Health Sci Ctr, Brooklyn, or Dr Rodriquez, Kings Cty Hosp, Brooklyn, NY).

COMMENT. The apparent immunological effects of methylphenidate suggested by these studies is a disturbing finding which should discourage the use of larger and more toxic doses of MPH in the treatment of ADHD. Further investigations of this MPH-induced immune system hyperactivity are indicated, especially in children with IgE-mediated asthma, allergic rhinitis, and other atopic diseases, in HIV infected children, and its possible interference with immunizations and the normal maturation of the immune system in young children. Drugs used in asthma have been implicated in causation of ADHD. We are now concerned with the possible effects of stimulant treatment of ADHD on the outcome of asthmatic and other allergic disorders.

THE CANTWELL MODEL OF ADHD SCIENTIFIC RESEARCH

Dr Dennis Cantwell, a world renown expert on ADHD, died April 14, 1997. In an article submitted from the UCLA Neuropsychiatric Institute in March 1997, as guest editor of a special section on ADHD, Dr Cantwell outlines his model in 8 phases of scientific study as follows: clinical diagnostic criteria, demographic, psychosocial, biological, family genetic, and family environmental factors, natural history, and management with psychostimulant medication and psychosocial methods of intervention.