Australia. (Levy F et al. J Am Acad Child Adolesc Psychiatry June 1997;36:737-744). ADHD may be explained as an inherited trait with liability and expression throughout the population, a deviance from an acceptable norm, and not restricted to an arbitrary number of symptoms or DSM criteria. The need for treatment including medication is relative, and dependent on multiple factors.

METHYLPHENIDATE TREATMENT AND HOME BEHAVIOR

Behavioral, situational, and temporal effects of treatment of ADHD with methylphenidate are reported in a study of 91 children receiving MPH (titrated to 0.7 mg/kg twice a day) or placebo for 4 months at the Hospital for Sick Children, Toronto, Canada. Symptoms of ADHD and comorbid oppositional behavior improved while at school, but not on returning home. Side effects were observed by the parents, not by teachers, and necessitated MPH withdrawal in 10%; these included sadness, behavioral deterioration, irritability, withdrawal, lethargy, violent behavior, and mild mania. Anorexia, loss of weight gain (without effect on growth), and stomachache were the most common physiological side effects, and withdrawal, sadness, and crying, the most common affective side effects during MPH treatment. (Schachar RJ, Tannock R, Cunningham C, Corkum PV. Behavioral, situational, and temporal effects of treatment of ADHD with methylphenidate. J Am Acad Child Adolesc Psychiatry June 1997;36:754-763). (Reprints: Dr Schachar, Child Psychiatry Research Unit, The Hospital for Sick Children, 555 University Ave, Toronto, Ontario, Canada M5G 1X8).

COMMENT. Methylphenidate administered twice daily benefits behavior and attention of ADHD children in the classroom but not in the home. Three times daily schedule of doses might facilitate completion of home work assignments and lead to improved parent-child relations. (see Ped Neur Briefs Nov 1996;10:82, for review of MPH dosing schedules and a report by Stein MA, Roizen NJ et al).

DEGENERATIVE DISORDERS

GENETICS OF JUVENILE SPINAL MUSCULAR ATROPHY

A 20-year-old female with difficulties in running and climbing stairs since age 10 and suspected of having spinal muscular atrophy (SMA) type III (Kugelberg-Welander disease) was diagnosed with GM2 gangliosidosis at the Department of Human Genetics, Sackler Faculty of Medicine, Tel Aviv University, and Sapir Medical Center, Kfar-Sava, Israel. Amyotrophy extended to the middle of the thighs in the lower limbs and had a distal glove distribution in the upper limbs. Deep tendon reflexes were present except for the ankle jerks. The survival motor neuron (*SMN)* gene, lacking in SMA, showed no deletion. Biochemical studies showed increased accumulation of GM2 ganglioside and deficiency of hexosaminidase A (Hex A) activity in fibroblasts. In the *HEXA* gene, two mutations occurred, and the patient was a compound heterozygote, with each allele containing a different mutation. (Navon R, Khosravi R, Melki J et al. Juvenile-onset spinal muscular atrophy caused by compound heterozygosity for mutations in the *HEXA* gene. Ann Neurol May 1997;41:631-638). Respond: Prof Navon, Molecular Genetics, Sapir Medical Center, Kfar-Sava 44281, Israel).

COMMENT. Progressive spinal muscular atrophy (SMA) type III