syndrome (TS) patients (mean age, 41 years), with 10 normal volunteers as controls, in a study at North Shore University Hospital, Manhasset, New York. Quantitative fluorodeoxyglucose (FDG) and PET were used to calculate global and regional rates of glucose metabolism. While global glucose metabolic rates were normal in TS patients, SSM analysis identified bilateral metabolic increases in lateral premotor and supplementary motor cortex and midbrain, and decreases in caudate and thalamic metabolism. TS patients have a nonspecific pattern of increased motor cortical activity common to other hyperkinetic disorders, and a specific brain network involving reduced activity of limbic basal ganglia-thalamocortical projections. (Eidelberg D, Moeller JR, Antonini A et al. The metabolic anatomy of Tourette's syndrome. Neurology April 1997;48:927-934). (Reprints: Dr D Eidelberg, Movement Disorders Center, North Shore University Hospital, 444 Community Drive, Manhasset, New York, 11030).

COMMENT. Two independent brain networks are identified in patients with Tourette syndrome: 1) increased metabolic activity of motor cortical regions involved in execution of movement; and 2) decreased metabolic activity in basal ganglia-thalamic areas governing TS global scale severity.

GENETICS OF TOURETTE SYNDROME

Age at onset, age at diagnosis, and phenotypic expressions of TS were compared in the offspring of affected males and females among 437 first degree relatives of 57 probands studied at Academic Department of Psychiatry, University College London Medical School, London, UK. Maternally transmitted cases had an earlier age at onset, suggesting a genomic imprinting effect on the expression of TS. (Eapen V, O'Neill J, Gurling HMD, Robertson MM. Sex of parent transmission effect in Tourette's syndrome: Evidence for earlier age at onset in maternally transmitted cases suggests a genomic imprinting effect. Neurology April 1997;48:934-937). (Reprints: Dr V Eapen, Faculty of Medicine & Health Sciences, United Arab Emirates University, PO Box 17666, Al Ain, United Arab Emirates).

COMMENT. Family history data on Tourette syndrome cases must be evaluated for maternal or paternal transmission. Mothers affected with TS are likely to have affected offspring with an earlier age at onset. Environmental factors, such as exposure to stress or cocaine, may be alternative explanations for this finding.

Developmental Basal Ganglia Syndrome. Failure to distinguish TS from other causes of DBGS may explain confusion in localizing the genetic defect in TS, determining prevalence, and assessing therapy, according to a report from the University of Rochester, NY. (Palumbo D, Maughan A, Kurlan R. Hypothesis III. Tourette syndrome is only one of several causes of developmental basal ganglia syndrome. Arch Neurol April 1997;54:475-483). Causes of the DBGS include primary (hereditary) tic disorders, obsessive-compulsive disorder, ADHD, Huntington disease, and secondary (symptomatic) autism, mental retardation, intrauterine drug exposure, perinatal asphyxia, encephalitis, head trauma, etc.

STIMULANT THERAPY OF ADHD AND TOURETTE SYNDROME

The effects of methylphenidate (MPH) and dextroamphetamine (DEX) on tic severity in 20 boys with ADHD and comorbid Tourette syndrome were investigated in a 9-week, placebo-controlled, double-blind crossover study at

the Child Psychiatry Branch, NIMH, Bethesda, MD. Tic severity was significantly greater during treatment with relatively high doses of both MPH (20-25 mg bid) and DEX (12.5-22.5 mg bid). Both stimulants significantly decreased hyperactivity and treatment was continued for 1 to 3 years in 14 of 20 patients. Tic exacerbations were reversible, and MPH was better tolerated than DEX. (Castellanos FX, Giedd JN, Elia J, Rapoport JL et al. Controlled stimulant treatment of ADHD and comorbid Tourette's syndrome: effects of stimulant and dose. J Am Acad Child Adolesc Psychiatry May 1997;36:589-596). (Reprints: Dr Castellanos, Bldg 10/Room 6N240, 10 Center Drive, Bethesda, MD 20892).

COMMENT. In treatment of ADHD and TS, the lowest effective dose of stimulant should be used, and increases in dosage should be made slowly. MPH appears less likely to induce persistent tic exacerbation than DEX.

Conventional doses of methylphenidate (0.1-0.3 mg/kg) produced dramatic improvement in behavior in children with ADHD and tic disorders, but complete normalization of behavior was not attained even with larger doses of 0.5 mg/kg, in a study at the Department of Psychiatry, SUNY at Stony Brook, NY. (Nolan EE, Gadow KD. Children with ADHD and tic disorder and their classmates: behavioral normalization with methylphenidate. J Am Acad Child Adolesc Psychiatry May 1997;36:597-604).

Weinberg WA et al, University of Texas Southwestern Medical Center, Dallas, TX, refer to the depressive reaction induced by methylphenidate, particularly with larger doses and in children with a genetic vulnerability. A stimulant-induced exacerbation of the neurochemical abnormality underlying depression is proposed as an explanation for seizures complicating MPH treatment of ADHD. (J Pediatr April 1997;130:665-669).

NEUROPATHIES

DIABETIC NEUROPATHY IN CHILDREN

Nerve conduction studies and renal function were evaluated prospectively in 144 diabetic children followed from the time of diagnosis and at 2, 5 and 10 years in the Department of Clinical Neurophysiology, Huddinge University Hospital, Sweden. At diagnosis and before complete remission of the diabetes, abnormal values were found in 25% of patients for motor conduction velocity and sensory nerve action potentials (SNAP) in the median nerve, for sensory conduction velocity and SNAP in the sural nerve, for parasympathetic autonomic function assessed by R-R intervals in the ECG, and for renal function evaluated by glomerular filtration rate. During long-term follow-up, an initial improved sensory nerve conduction was followed after 2 years by deteriorations in sensory and motor nerve conduction and autonomic nerve function. Poor glycemic control correlated with abnormal prolonged nerve conduction. Improved glycemic control by intense insulin treatment reduced the risk of diabetic neuropathy. (Solders G, Thalme B, Aguirre-Aquino M, et al. Nerve conduction and autonomic nerve function in diabetic children. A 10-year follow-up study. Acta Paediatr April 1997;86:361-366). (Respond: Dr G Solders, Dept of Clinical Neurophysiology, Huddinge University Hospital, S-141 86 Huddinge, Sweden).

COMMENT. Optimal glycemic control of diabetes in children may prevent or delay abnormalities in nerve conduction and autonomic dysfunction and the development of diabetic neuropathy.