

pediatric autoimmune neuropsychiatric disorders associated with streptococcal infection (PANDAS) and their limitations (Neurology June 1998;50:1530-1534). Criteria for diagnosis of TS-PANDAS include childhood onset of tics, and sudden onset or exacerbations related to streptococcal infection.

TOURETTE SYNDROME PREVALENCE

The prevalence of Tourette syndrome in a mainstream school population (ages 13 to 14 years) in West Essex, England has been determined by parent, teacher, and pupil questionnaires, class observations to identify tics, and subject and parent face-to-face interviews, and reported from the Section of Epidemiology and General Practice, Institute of Psychiatry, Denmark Hill, London, UK. From data available on 166 pupils in one school year, 5 children had TS, based on DSM-III-R criteria. Hyperactivity was an associated disorder in 4 and ADHD was present in 1. The prevalence estimate for TS in this age group was 299 per 10,000 pupils, or 3%. In comparison, the clinical records of the West Essex child and adolescent psychiatry service revealed 18 referred cases of TS out of a population of 37,500 children, ages 4 to 16 years, or a prevalence rate of 4.8 per 10,000 (0.05%). (Mason A, Banerjee S, Eapen V, Zeitlin H, Robertson MM. The prevalence of Tourette syndrome in a mainstream school population. Dev Med Child Neurol June 1998;40:292-296). (Respond: Dr Sube Banerjee, Section of Epidemiology and General Practice, The Institute of Psychiatry, De Crespigny Park, Denmark Hill, London SE5 8AF, UK).

COMMENT. TS among children in a community as a whole is more common and milder than that diagnosed in a health-care psychiatry service. The relative prevalence rates are 3% in a school population compared to 0.05% in a child psychiatry service in the UK. Prior studies involving school children from Monroe County, NY and a California school district found estimated TS prevalence rates of 3 per 10,000 (0.03%) and 76 per 10,000 (0.8%), respectively. (see Progress in Pediatric Neurology I, PNB Publ, 1991;pp228-9). The prevalence of TS in the UK study is four times greater than the highest US estimate in similar populations. The authors attribute the higher TS prevalence to be related to more thorough case ascertainment methods.

TOURETTE SYNDROME MATURATIONAL CHANGES

Tourette syndrome is considered a model neuropsychiatric disorder of childhood, reflecting an interaction between genetic and environmental factors, in a review of clinical characteristics, heredity and vulnerability, and neuroanatomy and neurochemistry, from the Child Study Center, Yale University, New Haven, CT. Referring to Israeli studies, the prevalence of TS is cited at 0.1% in boys and 0.01% in girls, or 1 in 1500 children; milder forms or chronic tic disorders have much higher prevalence rates of 4-6%. Most children developing tics have a history of prior hyperactivity or ADHD, and 30-60% develop OCD in preadolescence. Improvement during adulthood can be expected. TS is often familial, about 8% of relatives having TS and 17% exhibiting chronic tic disorders. Degree of severity is broad, mostly mild and only a minority showing serious functional impairment. An autosomal dominant transmission of inheritance is suggested by studies of large, multigenerational pedigrees, with higher penetrance for males than females. The search for the gene locus has been elusive, impeding research in the relative importance of genetic and environmental factors. Anatomically, symptoms of TS are related to abnormalities in cortico-striatal-thalamic-cortico pathways. Chemically, dopaminergic and serotonergic systems may be involved. (Cohen DJ, Leckman JF, Pauls D. Neuropsychiatric disorders of childhood: Tourette's syndrome as a model. Acta

Paediatr Suppl July 1997;422:106-111). (Respond: Dr DJ Cohen, Child Study Center, Yale University School of Medicine, 230 South Frontage Road, New Haven, CT 06510).

COMMENT. This review of the history of our understanding of Tourette syndrome serves as a model of changing concepts of neuropsychiatric disorders. Initially considered among neuroses and hysterias, TS is now treated as an example of genetic, developmental disorders, with a neuroanatomical and neurochemical basis, and more recently, an autoimmune disorder. Maturation changes in symptomatology are also complicated by comorbid OCD and ADHD, and the adverse effects of pharmacotherapy, especially stimulants. Methylphenidate is perhaps the primary environmental trigger for the onset or exacerbation of TS, and may also explain the apparent increased incidence and awareness of TS during the past 30 years.

Previous reports from the Yale group of TS investigators are reviewed in Progress in Pediatric Neurology III, PNB Publishers, 1997;pp314-5.

INFECTIOUS DISORDERS

ECHOVIRUS INFECTION AND BASAL GANGLIA EDEMA

The case of a 4-year-old girl with bilateral edema of the basal ganglia in association with echo type 21 viral infection is reported from the University Hospitals of Munster and Hamburg, Germany. Following an acute upper respiratory infection, the child developed viral meningitis, complicated by muscle hypotonia, ataxia, resting tremor, drowsiness, hyperesthesia, and speech dysarthria. MRI T2-weighted images showed hyperintense lesions of caudate nucleus, putamen, pallidum, and cerebellar peduncles, consistent with edema. Recovery began after 9 weeks, with a normal MRI and CSF at 3 months follow-up. (Freund A, Zass R, Kurlmann G, Schuierer G, Ullrich K. Bilateral oedema of the basal ganglia in an echovirus type 21 infection: complete clinical and radiological normalization. Dev Med Child Neurol June 1998;40:421-423). (Respond: A Freund MD, Abteilung für Kinder und Jugendmedizin, St Franziskus-Hospital, Hohenzollernring 72, 48145 Munster, Germany).

COMMENT. The authors cite 15 similar reports of pediatric postinfectious acute encephalopathies with striatal lesions, mostly with unspecified respiratory infection and more severe course, 4 diagnosed at autopsy. Other acute causes of bilateral striatal lesions include trauma, hemolytic-uremic syndrome, carbon monoxide, methylmalonic acidemia, glutaric aciduria type 1, sulfite oxidase deficiency, MELAS, hypoxia-ischemia, and vasculitis (after Roig M et al. Bilateral striatal lesions in childhood. Pediatr Neurol 1993;9:349-358).

HEADACHE

HEADACHE TRIGGERS AND PREVALENCE

The prevalence and triggers of various headache types in Finnish children at school entry and age 6 years were investigated at the University of Turku, Finland. Questionnaires sent to 1132 families with 6-year-old children revealed 96 children with headache disturbing their daily activities. Migraine was diagnosed in 55% and tension-type headache in 36%. The headache group and an asymptomatic control group were interviewed and examined. Compared to controls, those with headache had significantly more bruxism, occipital and temporomandibular joint tenderness, and more travel sickness. Triggers of