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. Maturational 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, Kurlemann 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 fur Kinderund 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