($p<0.0001$). By comparing the prevalence of neuropsychiatric disorders between children with and without tics, it was determined that these associations were not due to ascertainment bias or reactive asssociation but more likely, a shared neurobiologic mechanism, sometimes genetically determined. (Kurlan R, Como PG, Miller B et al. The behavioral spectrum of tic disorders. A community-based study. Neurology August (1 of 2) 2002;59:414-420). (Reprints: Roger Kurlan MD, University of Rochester School of Medicine and Dentistry, Department of Neurology, 601 Elmwood Avenue, Rochester, NY 14642).

COMMENT. The common association of tic disorders, including Tourette syndrome, and ADHD and OCD is most probably due to a shared neurobiologic mechanism.

Fluctuations in frequency and intensity of tic and associated behavioral disorders were determined in 553 children (kindergarten through 6th grade) observed monthly from November 1999 to June 2000 at the National Institute of Mental Health, Bethesda, MD. (Snider LA, Seligman LD, Ketchen BR et al. Tics and problem behaviors in schoolchildren: Prevalence, characterization, and associations. Pediatrics August 2002;110:331-336). Monthly point prevalence of motor tics ranged from 3.2% to 9.6% (overall frequency 24.4%). Monthly point prevalence of behavioral problems ranged from 2.6% to 11.0% (overall frequency 25.7%). Incidence of motor tics and problem behaviors was 3 times higher during winter months. Tics in most children were transient, and observed on only one occasion. These usually involved eye blinks and facial tics. Behavioral comorbidity was associated with more persistent and involved tic symptoms.

INFECTIOUS DISORDERS

DIAGNOSTIC CRITERIA OF ACUTE TRANSVERSE MYELITIS

Inclusion and exclusion criteria for the diagnosis of acute transverse myelitis (ATM), as a basis for multicenter clinical trials, are proposed by a Transverse Myelitis Consortium Working Group. Idiopathic ATM is distinguished from ATM secondary to known underlying disease. *Inclusion diagnostic criteria* are the following: bilateral signs and/or symptoms of spinal sensory, motor, or autonomic (sphincter) dysfunction; defined sensory level; MRI negative for extra-axial compression; CSF pleocytosis, elevated IgG index, or abnormal gadolinium enhancement indicative of spinal inflammation; progressive symptoms with nadir at 4 h to 21 d following onset. *Exclusion criteria* include : prior spinal irradiation; anterior spinal artery thrombosis; AV malformation; connective tissue disease (sarcoidosis, Behcet's, SLE etc); CNS syphilis, Lyme disease, HIV, HSV, EBV, CMV, or other viral disease; MRI evidence of MS or ADEM; history of optic neuritis and Devic's disease. A potential work-up for suspected ATM is outlined. Identification of known etiologies can lead to specific treatments whereas idiopathic ATM, that constitutes about 16% of cases, has no established therapy. (Transverse Myelitis Consortium Working Group. Proposed diagnostic criteria and nosology of acute transverse myelitis. Neurology August (2 of 2) 2002;59:499-505). (Reprints: Dr Douglas Kerr, Department of Neurology, Johns Hopkins Hospital, Pathology 627C, 600 N Wolfe St, Baltimore, MD 21287).

COMMENT. With the current interest in revival of smallpox vaccination, this proposal for diagnosis of acute transverse myelitis is timely. More than 200 cases of postvaccinial encephalomyelitis were reported in England in 1922-3, a complication of smallpox and rabies vaccination (Rivers TM, 1929). The present

proposal to distinguish idiopathic and secondary etiologies for ATV should clarify methods and results of therapy and prognosis.

Textbook descriptions of ATM mention pain in the back or extremities and sensory loss as the earliest symptoms. Two thirds have a history of recent infection (herpes, EBV, hepatitis B, influenza, measles, mumps, varicella), vaccinations, especially rabies, and with SLE. Pain is followed by progressive paraparesis, loss of sphincter tone, fever in 50%, and neck stiffness in one third. Flaccid weakness gradually changes to pyramidal tract signs, with hyperreflexia, clonus, and extensor responses. A sensory level (between T5-T10) affects primarily pain and temperature, and posterior columns are usually spared. CSF shows pleocytosis and an elevated protein in 50%, and neuroimaging is usually unremarkable. Prognosis is usually good, only 15% failing to show improvement. Therapy including steroids is of unproven benefit. (Menkes JH, 1981).

NATURAL HISTORY OF RASMUSSEN'S SYNDROME

Seizure frequency, degree of hemiparesis and cerebral hemiatrophy are analysed in 13 patients with histopathologically proven Rasmussen's encephalitis (RE) examined at the University of Bonn, Germany. The age at first seizure was in childhood in 10 cases (range, 1.6 - 15.7 years) and in adulthood in 3 (22.1 - 40.9 years). An initial prodromal stage (Montreal Neurological Institute Stage 1), characterized by low seizure frequency, had a median duration of 7.1 months (range 0 months to 8.1 years), shorter in children than adults. An acute (MNI stage 2) phase was recognized by a rapid increase in seizure frequency (to >10 simple partial motor seizures per day) accompanied by development or deterioration of hemiparesis and loss of hemispheric volume. Initial MRI scans showed inflammatory lesions, with monofocal onset between Rolandic and temporomedial areas, which spread across the ipsilateral hemisphere. The median duration of stage 2 was 8 months (range 4-8 months). Residual phase (MNI stage 3) was marked by a relatively stable permanent hemiparesis and diminished seizure frequency.

Patients were divided into two groups, according to age, duration of prodromal stage and outcome. Type 1 patients (n=7), all children, had a more rapid and severe disease than type 2 (adolescents and adults). Type 1 cases had a median age of 5.3 years (range 1.6-6.1 years) at onset of seizures, the prodromal stage was absent or short, and the acute phase lasted 8 months (range 4-8 months). Type 2 patients (n=6) had a median age of 18.9 years (range 6.4-40.9 years) at onset, a long prodromal stage (median duration 3.2 years, range 1.3-8.1 years), focal epilepsy with complex partial or secondarily generalized tonic-clonic seizures, rare occurrence of hemiparesis, of lesser frequency and degree than type 1 patients, only 2 requiring hemispherectomy, and an acute phase lasting 7.5 months (range 7-8 months). Histopathologically, the 2 types were similar, with chronic inflammatory changes in all patients. Most of the brain damage had occurred in the first 8-12 months. (Bien CG, Widman G, Urbach H et al. The natural history of Rasmussen's encephalitis. Brain July 2002;125:1751-1759). (Respond: Dr CG Bien, Department of Epileptology, University of Bonn, Sigmund-Freud-Strasse 25, D-53105 Bonn, Germany).

COMMENT. The authors recommend that future therapeutic interventions should focus on the 8 month period of the acute disease phase, when the most extensive brain damage occurs. In children, this acute phase with increased frequency of seizures and development of hemiparesis occurs early and soon after the onset of seizures, whereas in adolescents and adults, the acute phase is delayed and follows a long prodromal stage. In the later residual stage, the