Ahtiluoto S, Linnavuori K et al. Human herpesvirus-6 associated encephalitis with subsequent infantile spasms and cerebellar astrocytoma. Dev Med Child Neurol June 2000;42:418-421). (Respond: Dr Heikki Rantala, Department of Paediatrics, University of Oulu, 90220 Oulu, Finland).

COMMENT. This is the first reported case of HHV-6 association with subsequent infantile spasms and pilocytic cerebellar astrocytoma. HHV-6 DNA found in the tumor cells may indicate a causative role for the virus or simply an invasion of the tumor by the virus. The absence of MRI evidence for a tumor at the onset of infection is in favor of the former explanation. Laboratory studies have demonstrated oncogenic properties of HHV-6 virus.

MOVEMENT DISORDERS

VOLUMETRIC MRI IN TOURETTE SYNDROME: A GENDER EFFECT

Subcortical volumetric MRIs were compared in 19 girls (aged 7 to 15 years) with Tourette syndrome (TS) and 21 controls. Eleven of the patients had TS only and 8 had TS with attention deficit hyperactivity disorder (TS + ADHD). TS-only patients had significantly smaller lateral ventricles compared with TS + ADHD patients and the control subjects. Putamen asymmetry index as a marker for TS, previously demonstrated in boys with TS, was present in girls with TS and also in control girls with no TS. (Zimmerman AM, Abrams MT, Giuliano JD, Denckla MB, Singer HS. Subcortical volumes in girls with Tourette syndrome. Support for a gender effect. Neurology June (2 of 2);54:2224-2229). (Reprints: Michael T Abrams, Kennedy Krieger Institute, Neuroimaging Laboratory, Suite 522, 707 North Broadway Street, Baltimore, MD 21205).

COMMENT. In contrast to previous studies involving mainly boys, abnormal lenticular asymmetry does not occur in girls with Tourette syndrome. Girls, with or without TS, exhibit the putamen asymmetry seen in boys. TS-only girls differed from TS + ADHD girls and controls in having significantly smaller lateral ventricles. Future investigations of TS should make distinctions of gender and patients with and without ADHD.

TICS IN ASPERGER'S SYNDROME AND AUTISTIC DISORDER

Twelve patients (8 male and 4 female; ages 3 to 32 years; 9 less than 16 years) with autistic spectrum disorder were evaluated in a Movement Disorders Clinic for tics, at the University of California, Irvine, CA. Six had Tourette syndrome, and eight were also diagnosed with Asperger's syndrome. Three TS patients had severe congenital sensory deficits; 2 with Leber's amaurosis and 1 with deafness. All had stereotypic motor behavior, including rocking, head banging, and hand waving. Sensory deprivation was thought to contribute to the stereotypic movement disorder. (Ringman JM, Jankovic J. Occurrence of tics in Asperger's syndrome and autistic disorder. J Child Neurol June 2000;15:394-400). (Dr Joseph Jankovic, Baylor College of Medicine, 6550 Fannin #1801, Houston, TX 77030).

COMMENT. Asperger's syndrome should be considered in children of high verbal intelligence who do poorly in school, both academically and socially, and who exhibit speech and language disorders, tics, motor clumsiness, and stereotyped movements such as repetitive hand flapping. Asperger's syndrome may overlap or occur concurrently with Tourette syndrome (Nass R, Gutman R, 1997), pervasive developmental disorder, and ADHD (see Millichap JG. Attention

Deficit Hyperactivity and Learning Disorders. Chicago, PNB Publ, 1998). Multiple biological causes for autism and autistic-like disorders have been described. The EEG may be abnormal in 50% of cases and neuroimaging may show structural abnormalities in the brain in 25%.

Autism associated with mitochondrial mutations is reported in a family with heterogeneous neurologic disorders. (Graf WD, Marin-Garcia J, Gao HG et al. J Child Neurol June 2000;15:357-361).

Failure of Secretin treatment for autism is reported in 56 patients (49 boys, 7 girls; mean age 6 years) treated at Lake Forest Hospital, Illinois (Chez MG, Buchanan CP, Bagan BT et al. J Autism and Dev Disorders April 2000;30:87-94).

DEVELOPMENTAL DISORDERS

FAMILIAL PERISYLVIAN POLYMICROGYRIA

The clinical presentation and possible mode of inheritance of familial perisylvian polymicrogyria (FPP) are described in twelve affected kindreds presenting at 10 medical centers. Among 42 patients, clinical and radiological findings were variable in families and within members of the same family, except for abnormal tongue movements and/or dysarthria correlating with bilateral MRI findings. The main clinical features, pseudobulbar palsy, cognitive deficits, epilepsy, and perisylvian abnormalities, were not present uniformly, and varied in severity. The syndrome showed an X-linked transmission, except for 2 families with autosomal dominant inheritance and decreased penetrance. (Guerreiro MM, Andermann E, Guerrini R et al. Familial perisylvian polymicrogyria: a new familial syndrome of cortical maldevelopment. Ann Neurol July 2000;48:39-48). (Respond: Dr E Andermann, Montreal Neurological Institute, 3801 University Street, Montreal, Quebec, Canada H3A 2B4).

COMMENT. A new X-linked syndrome, named familial perisylvian polymicrogyria, is characterized by pseudobulbar palsy, cognitive deficits, epilepsy, and cortical maldevelopment. The syndrome is genetically heterogeneous, and the clinical presentation is variable.

ETIOLOGY OF AGENESIS OF CORPUS CALLOSUM

Agenesis of the corpus callosum was found in 7 of 135 children (aged 3 months to 15 years) with structural cerebral defects on MRI. All 7 showed dysmorphic features, psychomotor retardation, and neurologic abnormalities, and 6 had epilepsy (infantile spasms in 3). Causal factors in 4 children were partial trisomy of chromosome 13, partial duplication on the long arm of chromosome 10, Aicardi's syndrome, and intracranial bleeding in the fetus due to injury. Dandy-Walker malformation was also present in one other patient. (Marszal E, Jamroz E, Pilch J et al. Agenesis of corpus callosum: clinical description and etiology. J Child Neurol June 2000;15:401-405). (Respond: Prof Elzbieta Marszal, Pediatric Neurology Clinic, Silesian School of Medicine, ul Medykow 16, 40-752 Katowice, Poland).

COMMENT. Agenesis of the corpus callosum occurs as part of chromosomal syndromes, trisomy 8, 13, 18, or 21, and with X-linked syndromes, especially Aicardi's syndrome. It may be associated with other brain malformations, and it sometimes accompanies various inborn errors of metabolism, Exogenous factors such as intracranial bleeding and infection may also be involved in etiology.