Arch Pediatr Adolesc Med Feb 1994;148:174-179). (Reprints: Dr Stone, Child Development Center, Vanderbilt University Medical Center S, Room 426, 2100 Pierce Ave, Nashville, TN 37232).

COMMENT. Improved awareness of early signs of autism should help physicians recognize and refer patients for specialized intervention. Parents are better judges of a child's imaginative play and peer friendships, whereas physicians may be more objective about a child's social awareness, interactive play, imitation skills, and nonverbal communication.

Decreased plasma concentrations of the C4B complement protein are reported in a group of 42 autistic subjects examined at the Center for Persons with Disabilities and Department of Biology, Utah State University, Logan, UT. (Warren RP et al. Arch Pediatr Adolesc Med Feb 1994;148:180-183).

ABNORMAL EEG IN AUTISM: VALPROATE RESPONSE

Three children, ages 3, 4, and 5 years, with autism and epileptiform EEG discharges showed clinical improvement with valproic acid therapy at Mercy Hospital and Medical Center, Chicago, IL. None had a history of seizures. Within one month of VPA 125 mg tid treatment, language and social skills improved and the DSM-III-R criteria for autism no longer applied. Improvement had been maintained at follow-up 7 to 11 months later. (Plioplys AV. Autism: electroencephalogram abnormalities and clinical improvement with valproic acid. Arch Pediatr Adolesc Med Feb 1994;148:220-222). (Reprints: Dr Plioplys, Division of Neurology, Mercy Hospital and Medical Center, Stevenson Expressway at King Drive, Chicago, IL 60616).

COMMENT. The author stresses the importance of sleep EEGs to uncover epileptiform discharges in young autistic patients without history of clinical seizures. Further trials of antiepileptic drugs in autistic children seem justified.

POSTERIOR FOSSA ABNORMALITIES IN INFANTILE AUTISM

Previously published cerebellar vermis measures of 78 autistic patients from 4 separate MRI studies have been reanalysed at the Neurosciences Department, School of Medicine, University of California at San Diego, La Jolla, CA. Abnormalities were in 2 groups: vermal hypoplasia in 80-90% and vermal hyperplasia in 8-16% patients. These subgroups also differed significantly from normal controls. Failure to recognize these variations in vermal structure among patients may have lead to disparate reports of cerebellar maldevelopment in infantile autism. (Courchesne E et al. The brain in infantile autism: Posterior fossa structures are abnormal. Neurology Feb 1994;44:214-223). (Reprints: Dr Eric Courchesne, Neuropsychology Research Laboratory, Children's Hospital, 3020 Children's Way, San Diego, CA 92123).

COMMENT. Cerebellar pathology and hypoplasia have been reported in Rett and Down syndromes as well as autism. Attentional asynergia and dysfunction following cerebellar damage are linked to impaired social communication skills. Cerebellar mutism and personality changes have followed surgical removal of medulloblastoma.(Ped Neur Briefs Feb

1992;6:15-16). The finding of subtypes of neuroanatomic changes supports the belief that autism is a heterogeneous disorder.

TOURETTE SYNDROME

FREQUENCY, FAMILY AND SOCIAL ASPECTS OF TS

Results of a prospective, longitudinal study of 21 children recruited at age 2 1/2 to 3 1/2 years without tics but with a first-degree relative with Tourette syndrome (TS) are reported from the Department of Psychology and Child Study Center, Yale University, New Haven, CT. All subjects were evaluated annually and for 2 to 4 years. Among these high risk children, 24% had developed TS, 9% chronic tics, and 9% transient tics. Obsessive-compulsive symptoms occurred in 19%, and obsessive-compulsive disorder in 5%. Other diagnoses included attention deficit disorder, speech problems, and anxiety disorder in 24%. Children in this sample demonstrated an increased risk for tic disorders as well as other psychiatric disorders. Family functioning, independent of parental psychopathology, was associated with attention-deficit and anxiety disorders, decreased adaptive and increased maladaptive behaviors, and lower self-esteem but not tics or learning disorders. (CarterAS et al. A prospective longitudinal study of Gilles de la Tourette's syndrome. J Am Acad Child Adolesc Psychiatry March/April 1994;33:377-385). (Reprints: Dr David L Pauls, Child Study Center, Yale University School of Medicine, 230 S Frontage Road, New Haven, CT 06510).

COMMENT. An autosomal dominant mode of transmission for TS is suggested by the rates of tic disorders observed. Stressors in family functioning play a role in comorbid disorders such as anxiety and attentional difficulties. The authors advise family, cognitive-behavioral, and interpersonal therapies to address the social-emotional difficulties that often accompany TS.

Clonazepam was a useful adjunctive treatment for tics in children with comorbid ADHD studied at the Children's Hospital, Boston (Steingard RJ et al. J Am Acad Child Adolesc Psychiatry March/April 1994;33:394).

CONGENITAL DEVELOPMENTAL DISORDERS

SEPTO-OPTIC DYSPLASIA AND DIABETES INSIPIDUS

The clinical and endocrinological findings in 24 children with septo-optic dysplasia and/or agenesis of the corpus callosum are described with reference to posterior pituitary function in a report from the Institute of Child Health and The Hospital for Sick Children, London, UK. Congenital optic nerve hypoplasia, absent septum pellucidum, and pituitary deficiency, characteristic of the complete syndrome of septo-optic dysplasia, were present in 8 children, and 13 had incomplete forms. Five had agenesis of the corpus callosum. Growth hormone insufficiency was found in 20 (83%). Nine (38%) had diabetes insipidus, often complicated by hypernatremia. Management of fluid balance was difficult, even with vasopressin treatments, because of blindness, developmental delay, impairment of the sense of thirst, and dependence on the parents for food and water intake. (Masera N, Grant DB et al. Diabetes insipidus with impaired osmotic regulation in septo-optic dysplasia and agenesis of the corpus callosum. Arch Dis Child Jan 1994;70:51-53). (Respond: Dr DB Grant, The Hospital for Sick Children, Great Ormond Street, London WC1N 3JH, England).