The clinical characteristics were reviewed in 34 patients, age 5-35 years (Wing L. <u>Psychol Med</u> Feb 1981: <u>11</u>:115-129). Several children walked at a normal age but were slow to talk and became increasingly odd and withdrawn in personality. A genetic factor is suspected and no specific organic pathology has been identified. The syndrome listed in the ICD-10 but not in DSM-III-R is sometimes classified as a subgroup of the autistic spectrum with motor clumsiness, higher IQ and higher verbal skills (Tuchman R F. Autism: Delineating the spectrum. Int <u>Pediatr</u> 1991: <u>6</u>:161-169). Studies involving MRI and possible genetic or environmental factors require further investigation. In the absence of a specific etiology and biological marker, diagnosis is dependent on clinical criteria, a careful history, and full psychiatric and pediatric neurology evaluation.

## ORGANIC BRAIN DYSFUNCTION AND AUTISM

A population-based neurobiological study of 35 children with autistic disorder (AD) and 17 with autistic-like conditions (ALC) is reported from the Department of Child and Adolescent Psychiatry, University of Goteborg, Sweden. Major indications of brain damage or dysfunction were found in 90%. Etiological groups included Moebius syndrome (9%), fragile X syndrome (6%), chromosomal anomalies (6%), neurocutaneous disorders (6%), congenital hydrocephalus (3%), Rett syndrome (3%), Laurence-Moon-Biedl syndrome (3%), severe perinatal distress (9%), and epilepsy or severe EEG pathology (20%). Genetic factors were implicated in 9% with fathers and a brother with Asperger's syndrome. The EEG was abnormal in 50% and 18 (40%) had epileptiform discharges, maximal in the temporal lobes. CAT scan abnormalities in 25% included dilated ventricles, porencephaly, and general atrophy. BAERs were abnormal in 33%. (Steffenburg S. Neuropsychiatric assessment of children with autism: a population-based study. Dev Med Child Neurol June 1991; 33:495-511).

**COMMENT.** Multiple biological etiologies for autism and autistic-like disorders are suggested by this comprehensive study. Despite the extra effort and patience involved, the pediatric neurologist should not dismiss the child with autistic symptoms to the care of the psychiatrist without first attempting a full neurologic evaluation including EEG and CT or MRI.

## COGNITIVE IMPAIRMENT AND CARBAMAZEPINE

The effects on cognition of carbamazepine, phenytoin and sodium valproate were compared in 64 new cases of childhood epilepsy treated at the Leeds General Infirmary, England. The children age 5-14 years had no neurological deficit and were allocated randomly and assessed prospectively over a 12 month period. They were seizure-free throughout the 12 months. Psychological tests included visual recall, auditory recall (memory), visual scanning (vigilance), Stroop test (concentration), speed of information