and Adolescent Psychiatry, Goteborg University, Annedals Clinics, S-413 45 Goteborg, Sweden).

COMMENT. A simplified school entry examination involving four motor tests (standing on one foot, Fog test for associated movements, design copying and diadochokinesis), combined with clinical observation and parent interview, identified 80% of children with DAMP. In Scandinavia, DAMP replaced MBD in the 1980s, and overlaps with ADHD. One third of children with DAMP meet criteria for ADHD, and the remainder have ADD. Diagnosis of DAMP requires the presence of both attentional and motor/perceptual difficulties and a normal IQ. Testing by a neuropsychologist is recommended in suspected cases. The inclusion of neuromotor and perceptual criteria in the diagnosis of ADHD would add some objective data to the subjective behavioral criteria of the DSM-IV diagnosis.

DAMP and MBD versus AD/HD and hyperkinetic disorders is discussed in an invited commentary from the Children's Hospital, Karolinska Hospital, Stockholm, Sweden (Rydelius P-A. Acta Paediatr March 2000;89:266-268). The author refers to Rutter's publications (1970, 1994), stating that "brain disease with localizing neurologic signs is uncommon in children with ADDH....by contrast, the more severe hyperkinetic disorder is disproportionately common in children with damaged brains." In Sweden, "the concept of DAMP is in need of revision," as are the diagnostic criteria for ADHD in the USA.

ANTIEPILEPTIC DRUGS

VIGABATRIN-ASSOCIATED "ACUTE ENCEPHALOPATHY"

A 6-month-old girl with Alexander disease and hydrocephalus, treated at Children's Hospital, Tubingen, Germany, developed acute encephalopathy within 3 days of starting vigabatrin (VGB). She had been admitted because of seizures refractory to phenobarbital (plasma level 20 mg/L). VGB was added in an initial dose of 150 mg daily, and doubled the following day (45 mg/kg body weight). On the third day, the child became somnolent and soporous. Levels of phenobarbital were unchanged. A pre-VGB EEG showing 5 HZ background activity changed to generalized high voltage delta waves. Causes other than VGB, including shunt dysfunction, encephalitis, metabolic disorder and renal failure, were excluded. VGB was discontinued and symptoms subsided within two days. The EEG returned to the pre-VGB 5 HZ background activity. (Haas-Lude K, Wolff M, Riethmuller J, Niemann G, Krageloh-Mann I. Acute encephalopathy associated with vigabatrin in a six-month-old girl. Epilepsia May 2000;41:628-630). (Reprints: Dr K Haas-Lude, Children's Hospital, University of Tubingen, Hoppe-Seyler-Strassee 1, D-72076 Tubingen, Germany).

COMMENT. Acute encephalopathy associated with VGB monotherapy has been reported in 7 adults and a child aged 14 years, but not previously in an infant. The infant was profoundly sleepy (soporous), not stuporous, and perhaps the diagnosis of "encephalopathy" could be revised to "VGB-associated excessive somnolence." A previous case of an infant with VGB-induced somnolence, necessitating drug withdrawal, was reported by Dimova PS, Korinthenberg R (Pediatr Neurol 1999;21:802-807). In this report, the initial dose of VGB was 5 to 63 mg/kg, increasing to 15 to 180 mg/kg (median 63). A smaller initial dose of VGB in the above case may have averted the adverse effect and the need to discontinue therapy. A similar phenomenon was common with primidone, when this antiepileptic drug was first introduced.