ataxia (23%), and sensory signs (23%). Cranial nerve paralyses involved the VIIth in 35%, and extraocular muscles in 16%. Autonomic dysfunction was present in 26%, and 28% required assisted ventilation. All except 2 recovered completely. Motor and sensory nerve conduction studies showed signs of demyelination during the first week in 90%, and the diagnosis of AIDP was confirmed in the 2nd week in all patients. A reduced compound muscle action potential was the earliest abnormality (83%), and motor conduction was abnormal in only 50%, during week 1. The outcome was better in children than in adults. (Delanoe C, Sebire G, Landrieu P, Huault G, Metral S. Acute inflammatory demyelinating polyradiculopathy in children: Clinical and electrodiagnostic studies. Ann Neurol Sept 1998;44:350-356). (Respond: Dr Metral, Service d'Explorations Fonctionnelles du Systeme Nerveux, 78 Rue du General Leclerc, 94275 Kremlin, Bicetre Cedex, France).

COMMENT. Based on electrophysiologic criteria, the diagnosis of AIDP may be suspected in the first week and confirmed in the second week of the illness. The clinical manifestations in the acute phase are similar in children and adults, but the outcome of AIDP in children is better. More than one-third of children affected are under 3 years of age. Severe limb or back pain, weakness, and ataxia are the most prominent initial symptoms.

In contrast to the acute form of inflammatory demyelinating polyneuropathy (IDP), the chronic form presents with lower extremity weakness and difficulty in walking in 85%. The majority have a poor long-term prognosis, with frequent relapses and residual weakness (Progress in Pediatric Neurology III, PNB Publ, 1997;pp360-362).

Correlation between cytomegalovirus infection and IgM anti-MAG/SGPG antibody-associated neuropathy (Yuki N, Yamamoto T, Hirata K. Ann Neurol Sept 1998;44:408-410) is reported from Dokkyo University, Tochigi, Japan. CMV infection may induce the IgM anti-myelin-associated glycoprotein antibody found in some patients with chronic polyneuropathy.

ATTENTION DEFICIT AND LEARNING DISORDERS

EFFECT OF TRAUMATIC BRAIN INJURY ON ADHD SYMPTOMS

The course of attention-deficit hyperactivity (ADH) symptomatology in 50 children and adolescents after traumatic brain injury (TBI) at 6 to 14 years of age was studied prospectively at the University of Iowa, Iowa City, IA. Changes in the ADH symptomatology and early onset of hyperactivity in the first 2 years after TBI were significantly related to the severity of the injury and degree of brain damage, and to a measure of family dysfunction, but a correlation with MRI findings was not documented. (Max JE, Arndt S, Castillo CS et al. Attention-deficit hyperactivity symptomatology after traumatic brain injury: a prospective study. J Am Acad Child Adolesc Psychiatry Aug 1998;37:841-847). (Reprints: Dr Jeffrey E Max, Department of Psychiatry, University of Iowa, 1876 JPP, Iowa City, IA 52242).

COMMENT. This study confirms previous reports of the relation between head injury and brain damage and the ADHD syndrome. It also demonstrates a positive "dose-response" relationship between severity of injury and change in ADH symptoms. It is unfortunate that the MRI findings in this series of patients could not document a neuroanatomical correlate of the post-TBI change in ADH symptoms. The literature regarding head injury and ADHD is reviewed in Attention Deficit Hyperactivity and Learning Disorders, PNB Publ, 1998;pp 14-21.