late onset intractability (Engel J Jr et al, 1997), a condition not addressed in this study. Despite these criticisms and cautionary comments, the criteria for IE identified by Berg and associates will be valuable in counseling parents on the increased necessity for adequate treatment and careful and frequent monitoring of seizures.

DEMYELINATING DISORDERS

ACUTE DISSEMINATED ENCEPHALOMYELITIS

The clinical and neuroradiological features of acute disseminated encephalomyelitis (ADEM) were determined by a retrospective review of medical records and MRI findings in 31 children (mean age 5.9 yrs; range 2-16 yrs) examined at the Royal Children's Hospital, Melbourne, Australia, between 1993 and 1998. Prodromal illnesses, mainly upper respiratory or nonspecific infections, occurred in 22 (71%). Two had received hepatitis B vaccine 3 to 6 weeks prior to symptom onset. Neurologic symptoms, developing over a mean of 4 days, included ataxia (65%), cranial nerve pareses (45%), headache (45%), vomiting (35%), neck stiffness (26%), impaired consciousness (68%), seizures (13%), optic neuritis (13%), hemiparesis (23%), and language disturbance (29%). CSF protein was elevated in 50% (0.4-0.6g/L), and csf white cell counts were abnormal, mainly lymphocytes ($52x10^6$/L or less), in 62%. Oligoclonal bands in csf were present in only one. EEGs showed slow background activity in 5 of 7 tested. Serologic testing showed elevated immunoglobulin M titers to M. pneumoniae in 4, and to Epstein-Barr virus in 2. PCR was negative in csf of 23 tested. MRI showed bilateral, asymmetrical involvement of white matter of frontal and parietal lobes in all but 3 patients; the lesions resembled tumor masses in 2 with the diagnosis of demyelination confirmed by biopsy. Corpus callosum and periventricular lesions characteristic of MS were present in 29%. Deep gray matter was involved in 61% and the spinal cord in 16%. High-dose IV methylprednisolone was usually effective in treatment. Recovery was complete in 81%, and sequelae were mild in the remainder. (Hynson JL, Kornberg AJ, Coleman LT et al. Clinical and neuroradiologic features of acute disseminated encephalomyelitis in children. Neurology May (2 of 2) 2001;56:1308-1312). (Respond: Dr Andrew J Kornberg, Department of Neurology, Royal Children's Hospital, Flemington Road, Parkville, Victoria, Australia 3052).

COMMENT. Serial MRI may be necessary to distinguish ADEM from MS in some cases. In the absence of a clinical relapse, new lesions should not appear in ADEM. The diagnosis of ADEM is usually apparent at presentation. Characteristic features are the prodromal viral illness, early onset of ataxia, large lesions on MRI with involvement of deep gray matter including thalamus, and absence of oligoclonal bands.

ADEM in a patient with autoimmune hemolytic anemia. A 16-year old girl with a 3 year history of hemolytic anemia had received immunosuppressive prednisolone treatment at Chang Gung Children's Hospital, Taoyuan, Taiwan. She developed a systemic infection with Cryptococcus neoformans which triggered the encephalomyelitis. Her symptoms included ataxia, drowsiness, and coma. MRI showed multiple bilateral, asymmetrical lesions involving subcortical white matter, basal ganglia, and frontoparietal lobes. She responded to IV immunoglobulin IVIG, the ataxia and weakness resolving after 2 weeks. Follow-up MRI in 1 month was almost normal, and neurologic symptoms had completely resolved. No relapse had occurred at 1 year follow-up. (Jaing T-H, Lin K-L, Chiu C-

H et al. Pediatr Neurol April 2001;24:303-305). The authors suggest a link between the chronic steroid treatment in an immunosuppressed patient and the development of ADEM.

VASCULAR DISORDERS

PROGNOSTIC FACTORS IN ISCHEMIC ARTERIAL STROKE

The predictive value of presenting symptoms, MRI and CT findings, and etiology in the outcome of ischemic arterial childhood stroke was determined in a consecutive series of 31 patients followed at the University Hospital, Rotterdam, The Netherlands. Hemiparesis was the most common presenting symptom (74%), seizures occurred in 19%, altered level of consciousness in 16%, and ataxia in 7%. Location of infarction on neuroimaging was in the territory of the middle cerebral artery (MCA) in 27 cases, basilar artery (BA) in 4, and in the cerebellum involving the posterior inferior cerebellar artery (PICA) in 2. Three MCA and 2 MCA and ACA strokes (19%) were complete. Etiology was identified in 24 (77%), including cardiac surgery complications in 6, varicella zoster-related in 5, mitochondrial disease in 2, migraine-related in 2, and Moya-Moya, Kawasaki disease, factor V Leiden, sickle-cell disease, and hyperthyroid crisis in 1 each. Risk factors at presentation that correlated with a poor prognosis were an altered level of consciousness, seizures, and a completed stroke of the MCA. Etiology, age at presentation, or gender showed no significant correlation with outcome. (Delsing BJP, Catsman-Berrevoets CE, Appel IM. Early prognostic indicators of outcome in ischemic childhood stroke. Pediatr Neurol April 2001;24:283-289). (Respond: Dr Catsman-Berrevoets, Child Neurologist, Dept of Child Neurology, Dr Molewaterplein 60, 3015 GJ Rotterdam, The Netherlands).

COMMENT. Almost one-half the patients in this study died or had severe residual morbidity. The early risk factors for this poor outcome were an altered level of consciousness at presentation, seizures, and MRI evidence of complete or end-zone MCA infarction.

NEUROMUSCULAR DISORDERS

EXPANDED MOBIUS SYNDROME

An infant born with Mobius syndrome died at 22 days and was found at autopsy to have more widespread involvement of brainstem and cranial nerve nuclei than usual, resulting in an "expanded Mobius syndrome," as reported from the University of Iowa Hospital, Iowa City, IA. At emergency cesarean section, performed at 33 weeks gestation because of fetal distress and arm tremor, a 1672 gm male infant required continuing ventilatory support. At neurologic examination, the diagnosis was expanded Mobius syndrome with diffuse cranial nerve and brainstem involvement. CT and MRI revealed diffuse cerebral atrophy. Postmortem examination showed bilateral pneumonia secondary to aspiration. The brain was of normal weight and its surface appeared normal. Cranial nerve rootlets VI-XII were absent. Microscopic examination showed bilateral brain, basal ganglia, and brainstem gliosis and mineralization. Neurons in the nuclei of cranial nerves III-XI were absent. There was lesser involvement of the spinal cord, cerebral white matter, and cerebellum. No inflammatory cells or evidence of infection were evident. (Peleg D, Nelson GM, Williamson RA, Widness JA. Expanded Mobius syndrome. Pediatr Neurol April 2001;24:306-309). (Respond: Dr Widness, Department of Pediatrics, University of Iowa Hospital and Clinics, 200 Hawkins Drive, Iowa