diagnosis should allow more accurate parental counseling and more effective long-term treatment.

ABORTED AND REFRACTORY STATUS EPILEPTICUS COMPARED

Clinical and EEG characteristics, etiologies, treatment response, and predictors of long-term outcome were determined in 154 children with status epilepticus (SE) hospitalized at the Mayo Clinic, Rochester, MN, 1994-2004. Patients with status aborted with medication (ASE) in 69% were compared to 39% with refractory SE (RSE). SE was defined as continuous tonic-clonic or electrographic seizure activity for at least 10 min or intermittent seizure activity without recovery of consciousness for at least 30 min. (Mayer SA et al. Arch Neurol 2002;59:205-210). Etiology of SE was acute symptomatic in 26%, remote symptomatic 35%, idiopathic 20%, and febrile in 10%. RSE compared to ASE was significantly associated with a higher family history of seizures, higher number of seizures and AEDs, nonconvulsive SE, and focal or electrographic seizures on initial EEG. In-hospital mortality was significantly higher with RSE (13.3%) than ASE (2.1%). RSE patients developed more neurological deficits and more epilepsy at long-term follow-up than ASE children. More aggressive treatment resulted in better responses and outcomes. Poor outcome risk factors included long seizure duration, acute symptomatic etiology, nonconvulsive SE, and young age (<5 years) at admission. Prospective, randomized trials of different treatment protocols are advocated. (Lambrechtsen FACP, Buchhalter JR. Aborted and refractory status epilepticus in children: a comparative analysis. Epilepsia 2008;49(4):615-625). (Respond: Jeffrey R Buchhalter MD, Phoenix Children's Hospital, 1919 E Thomas Road, Phoenix, AZ 85016).

COMMENT. Status epilepticus in children is refractory in 40%, and RSE is related to family history, number of seizures and AEDs, nonconvulsive status, and initial EEG abnormalities. Etiology is an important determinant of outcome, especially acute symptomatic causes. Identification of these risk factors should lead to more aggressive therapy and better outcome.

INFECTIOUS DISORDERS

HERPES SIMPLEX VIRUS-1 AND BELL'S PALSY

The association between herpes simplex virus-1 (HSV-1) infection and Bell palsy was determined in 47 children studied at Children's Hospital at Montefiore, Bronx, NY. Swabs of saliva and conjunctiva were taken for PCR testing. To validate PCR testing, swabs were obtained from patients with oral lesions of herpes gingivostomatitis. An HSV-1 enzyme-linked immunosorbent assay was positive in 33 of 42 affected patients compared to 16 of 41 controls (P=0.003). HSV-1 polymerase chain reaction was positive in 10 of 47 affected patients compared to 4 of 45 controls (P=0.08). The findings support an association between HSV-1 infection and Bell palsy in children. (Khine H, Mayers M, Avner JR, Fox A, Herold B, Goldman DL. Association between herpes simplex virus-1 infection and idiopathic unilateral facial paralysis in children and adolescents. **Pediatr Infect Dis J** May

2008;27:468-469). (Respond: David L Goldman MD, Division of Pediatric Infectious Diseases, Children's Hospital at Montefiore, Albert Einstein College of Medicine, 1300 Morris Park Ave, Bronx, NY 10461).

COMMENT. These findings in children confirm previous reports of a probable role for HSV-1 infection in adults with Bell's palsy. Acyclovir in treatment of Bell's palsy deserves further study. Other infectious causes reported include Epstein-Barr virus, mumps, enteroviruses, and rarely, varicella zoster virus (geniculate herpes, Ramsay Hunt syndrome).

INFLUENZA-ASSOCIATED ENCEPHALITIS/ENCEPHALOPATHY

The role of influenza A and influenza B in acute childhood encephalitis and encephalopathy (ACE) was evaluated prospectively in all children admitted to the Hospital for Sick Children, Toronto, Canada, during an 11-year period from Jan 1994- Dec 2004. Influenza infection was defined by detection in the nasopharynx by immunofluorescence microscopy or viral culture and/or by a 4-fold or greater rise in complement fixation titer. In 311 children with ACE, influenza infection was detected in 22 (7%); 11 were <5 years of age. Fourteen fulfilled criteria for ACE. Influenza A was detected in 13 of 14 cases, and influenza B in 1 case. Neurologic manifestations developed within 5 days of onset of respiratory symptoms in 64%. These included seizures, cranial nerve abnormalities, focal motor deficits, gait abnormalities, meningismus, torticollis, hyperreflexia and opisthotonus. Two presented with status epilepticus, and 2 had hemiparesis, CSF pleocytosis occurred in 3 patients, and elevated protein in 4. Neuroimaging abnormalities noted in 8 of 14 tested were more common in children <2 years of age. Neurologic sequelae occurred in 8 patients (in 5 <2 years of age), and included seizures, hemiparesis, ataxia, and speech disorder. EEGs were abnormal in all 8 of those with neurologic sequelae and in 4 of 6 without sequelae. An acute rather than a postinfectious process was suggested by the briefness of the respiratory prodrome. (Amin R, Ford-Jones E, Richardson SE, et al. Acute childhood encephalitis and encephalopathy associated with influenza. A prospective 11-year review. Pediatr Infect Dis J May 2008;27:380-395). (Respond: Ari Bitnun MD, FRCPC, University of Toronto, Division of Infectious Diseases, Hospital for Sick Children, 555 University Ave, Toronto, Ontario, Canada M5G 1X8, E-mail:ari.bitnun@sickkids.ca).

COMMENT. Influenza virus infection is associated with 5% of cases of acute childhood encephalitis/encephalopathy in Canada. The younger children <5 years of age are most susceptible, and children <2 years of age are most likely to have neurologic sequelae.

A Japanese study of prognostic factors in influenza-associated encephalopathy evaluated 442 cases retrospectively. (Nagao T, Morishima T, Kimura H et al. **Pediatr Infect Dis J** 2008;27:384-389). Type A influenza was detected in 84% and type B in 9.5%. Fifty-four cases (22%) had a history of febrile convulsions. Significant factors for a poor prognosis and death in 35(19%) of 184 cases were an elevated transaminase, hyperglycemia, hematuria or proteinuria, and use of diclofenac sodium for fever during the infection. Factors showing a trend toward poor prognosis were elevated body temperature (>41C), low platelets, and low blood sugar. The occurrence of these signs should prompt admission to intensive care.