for Rehabilitative Services, University of Arizona Health Sciences Center, Tucson, AZ. Seventeen (21%) had seizures during follow-up ranging from 1.3 to 16 years. Fourteen (17%) had epilepsy and 5 had seizures controlled by anticonvulsant drugs. CNS pathology in addition to the shunted hydrocephalus included encephalomalacia in 7, cerebral malformations in 2, and calcifications in 1. (Talwar D et al. Epilepsy in children with meningomyelocele. Pediatr Neurol July/August 1995;13:29-32). (Respond: Dr Talwar, Department of Pediatrics, University of Arizona Health Sciences Center, 1501 North Campbell Avenue, Tucson AZ 85724).

COMMENT. Although epilepsy in children with meningomyelocele occurs mainly in those with shunted hydrocephalus, structural cerebral abnormalities other than the shunt may be important causes.

## INFECTIOUS DISORDERS

### HERPESVIRUS-6 INFECTION AND FIRST FEBRILE SEIZURES

The association between acute human herpesvirus-6 (HHV-6) infection and first febrile convulsions was investigated prospectively in 42 children evaluated by virologic and serologic methods at the North Shore University Hospital-Cornell University Medical College, Manhasset, New York. Primary HHV-6 infection was documented by viral culture in 8 (19%), and fourfold rises in HHV-6 titer were present in 9 (26%) of 34 children whose blood was analyzed for acute and convalescent HHV-6 titers. The majority (10 of 11) HHV-6 cases were less than 24 months of age, and 3/11 had roseola. Viral isolation in CSF, attempted in 29, including 7 with evidence of HHV-6 illness, was negative. (Barone SR et al. Human herpesvirus-6 infection in children with first febrile seizures. J Pediatr July 1995;127:95-97). (Reprints: Stephen R Barone MD, North Shore University Hospital, 300 Community Drive, Manhasset, NY 11030).

COMMENT. Acute HHV-6 infection is a significant factor in the etiology of fever and convulsions in young children. Seizures associated with exanthem subitum and HHV-6 infection are not always simple in type, however. They are occasionally prolonged and complex and a manifestation of encephalitis or encephalopathy. See Progress in Pediatric Neurology II, 1994, Chicago, PNB Publishers, for a report and comment on HHV-6 infection, exanthem subitum, and encephalitis/encephalopathy. HHV-6 virus DNA was detected in the cerebrospinal fluid of 6 infants with exanthem subitum, 3 having a pleocytosis and elevated protein in the CSF. (Suga S et al. Ann Neurol 1993;33:597-603).

### OPSOCLONUS-MYOCLONUS OUTCOME

The developmental outcome of 11 patients with opsoclonus-myoclonus, 8 having occult neuroblastoma, is reported from the Division of Pediatric Neurology, Children's Memorial Hospital, Chicago. Nine were treated with ACTH and 3 received prednisone. Symptoms recurred in 9 when ACTH was withdrawn. The response to predisone was minimal. Symptoms were not improved by removal of a neuroblastoma. The median age at presentation was 17 months. Follow-up ranged from 12 to 115 months. Delayed development with motor incoordination and speech delay occurred in 8 children and 3 had behavioral problems. IQs ranged from 56 to 75 in 7 children and one had a

borderline IQ. Development was normal in 2 of 3 patients without neuroblastoma and in only 1 of 8 whose opsoclonus-myoclonus was associated with neuroblastoma. (Hammer MS, Larsen MB, Stack CV. Outcome of children with opsoclonus-myoclonus regardless of etiology. Pediatr Neurol July 1995;13:21-24). (Respond: Dr Hammer, Division of Pediatric Neurology, Children's Memorial Hospital, 2300 Children's Plaza, #51, Chicago, IL 60614).

COMMENT. Other terms for this syndrome include myoclonic encephalopathy of infancy (MEI), dancing eyes syndrome, and infantile polymyoclonia. The majority of children with opsoclonus-myoclonus in this study were found to have significant developmental delay. Others report that about 50% are left with intellectual deficits. (Boltshauser E et al. Helv Pediatr Acta 1979;34:119). The criteria for diagnosis were 1) marked motor incapacity from myoclonic jerking and/or cerebellar ataxia, 2) opsoclonus, 3) acute or subacute onset, and 4) absence of central nervous system infection. All 3 children with MEI without neuroblastoma had a viral illness 1-2 weeks before symptoms began. The pathogenesis is multiple and is usually viral in origin, notably poliovirus, Coxsackie virus B3, and St Louis encephalitis virus. An autoimmune mechanism and DDT intoxication have also been invoked. (Menkes JH. Textbook of Child Neurology. 3rd ed. Philadelphia, Lea & Febiger, 1985). In treatment, some advocate ACTH for the acute stage followed by predisone for several months. (see Progress in Pediatric Neurology I, 1991, Chicago, PNB Publishers, p 486).

## HIV INFECTION AND NEURODEVELOPMENT

The mental and motor development of 24 children with vertically transmitted human immunodeficiency virus (HIV) infection in the first 30 months of life was compared to 27 HIV exposed but uninfected children at the Boston City Hospital and Boston University Medical Center, MA. Bayley Scales of Infant Development, assessed at 4-16 months and at 17-30 months of age, showed that motor development in the infected group was delayed compared to the uninfected seroreverter group in both age periods. Mental development was similar in the two groups at 4-17 months, but was delayed in the HIV infected children at 17-30 months of age. (Chase C et al. Early neurodevelopmental growth in children with vertically transmitted human immunodeficiency virus infection. Arch Pediatr Adolesc Med August 1995;149:850-855). (Reprints: Dr Chase, Department of Pediatrics, D4S, Boston City Hospital, 818 Harrison Ave, Boston, MA 02118).

COMMENT. Neurodevelopmental outcome in children with HIV infection is variable, but early delay in motor development and late infantile deceleration in mental development can be expected in HIV infected children.

# TOXIC DISORDERS

## LEAD EXPOSURE IN DAY CARE CENTERS

The risk of lead poisoning among 155 of 234 eligible children (mean age, 4.8 years) enrolled in university affiliated day care centers with elevated environmental lead sources was determined at the Department of Pediatrics and University Hygienic Laboratory, The University of Iowa, Iowa City.