flaccid paralysis (4 {10%}), and brain-stem encephalitis or rhombencephalitis (37 {90%}).

In 37 patients with rhombencephalitis, 86% had myoclonus, and tremor, ataxia, or both were present in 62%. Ocular disturbance occurred in 9 and bulbar palsy in 1. Cardiorespiratory failure developed in 7, and 5 (14% of total) died despite ventilatory support. EEGs showed bilateral slow waves in 5, and MRI had high signal intensity lesions in the brain stem on T2 images of 71%, the frequency of MRI abnormalities increasing with severity of the rhombencephalitis. At follow-up, 14% had neurologic sequelae, including myoclonus, abducens palsy, facial diplegia, ataxia, internuclear ophthalmoplegia, and ventilator-dependent apnea. (Huang C-C, Liu C-C, Chang Y-C, Chen C-Y, Wang S-T, Yeh T-F. Neurologic complications in children with enterovirus 71 infection. N Engl J Med Sept 23, 1999;341:936-942). (Reprints: Dr Huang, Department of Pediatrics, College of Medicine, National Cheng Kung University, 138 Sheng-Li Rd, Tainan, 704. Taiwan).

COMMENT. Rhombencephalitis is the chief neurologic complication of enterovirus 71 infection in children affected during an epidemic in Taiwan. MRIs show lesions in the brain stem, depending on the severity of the illness, and 14% are fatal. Except for enterovirus 71, enteroviral meningoencephalitis generally has a good prognosis. The infection is characterized by self-limiting fever, vomiting, ulceration of mouth and palate, and vesicular lesions on the hands and feet, but may be followed by aseptic meningitis, meningoencephalitis, or acute flaccid paralysis resembling polio.

"INFANTILE" BOTULISM IN AN ADULT

A diagnosis of adult-onset "infant" botulism was confirmed by identification of botulinum toxins A and E in stool and serum of a 32-year-old woman with cystic fibrosis treated for a pseudomonas pneumonia and having a percutaneous gastrostomy tube placed for poor intake and weight loss, at the Department of Neurology, University of Minnesota, Minneapolis. Wound infection was not established, but 2 weeks after gastrostomy the patient developed nausea and vomiting, with ileus, progressive dysphonia, neck and proximal weakness, and respiratory failure. Partial ptosis and slowly reacting pupils were noted 2 days later, but extraocular movements, tendon reflexes, and sensation were normal. EMG studies were suggestive of presynaptic neuromuscular junction defect. Motor unit potentials were of small amplitude and brief duration with increased polyphasia. There was early recruitment with interference pattern and reduced peak-to-peak amplitude. Increments in CMAP amplitude with 50-Hz stimulation, and single-fiber EMG of extensor digitorum were abnormal. Sensory and motor conductions in all limbs were normal. Stool culture grew *Clostridium botulinum*, but the source of the infection was not identified. (Li LYJ, Kelkar P, Exconde RE, Day J, Parry GJ. Adult-onset "infant" botulism: An unusual cause of weakness in the intensive care unit. Neurology Sept (1 of 2) 1999;53:891). (Reprints: Dr Praful Kelkar, Department of Neurology, University of Minnesota, Box 295, 516 Delaware St, Minneapolis, MN 55455).

COMMENT. Adult-onset infantile botulism is rare, but this case report is of interest because the patient was suffering from cystic fibrosis, a childhood disease, which affects intestinal motility and, along with prolonged antibiotic therapy, predisposes to colonization of the gut by *C botulinum*. The diagnosis should be considered in a child admitted to the intensive care unit in respiratory distress, and presenting with nausea, vomiting, and fever that precede the onset of ptosis, nonreactive pupils, dysphagia, dysphonia, weakness of facial and neck

muscles, followed by flaccid paralysis. Sluggish pupils and paralytic ileus were clinical clues in the differential diagnosis.

TRAUMATIC DISORDERS

CLINICAL SIGNS OF BRAIN INJURY IN INFANTS

The predictive and diagnostic value of clinical signs of intracranial injury (ICI) in head-injured infants was studied at Children's Hospital, Harvard Medical School, Boston, MA. Of 608 infants presenting at the emergency department with head injury, 30 (5%) had ICI; 13% 0-2 months of age had ICI, compared to 6% of infants 3 to 11 months, and 2% of infants 12 months or older. Only 52% of infants with ICI had one or more of the following clinical signs of brain injury: loss of consciousness, seizures, vomiting, irritability, bulging fontanel, focal neurologic signs, or signs of increased intracranial pressure. Of 14 asymptomatic infants with ICI, 93% had scalp hematoma. Scalp hematoma was strongly associated with ICI in infants having CT scans. (Greenes DS, Schutzman SA. Clinical indicators of intracranial injury in head-injured infants. Pediatrics October 1999;104:861-867). (Reprints: Dr David S Greenes, Division of Emergency Medicine, Children's Hospital, 300 Longwood Ave, Boston, MA 02115).

COMMENT. Clinical symptoms and signs of brain injury are of limited value in the diagnosis of intracranial injury in infants. Radiographic imaging is important in the work-up of infants with head injury even when asymptomatic, especially in those with scalp hematoma and in infants younger than 3 months of age. The younger the infant, the greater the risk of ICI. Asymptomatic infants older than 3 months of age who present with a history of head injury and who have no scalp hematoma are not likely to have sustained intracranial injury.

MOVEMENT DISORDERS

PATTERN OF INHERITANCE OF TOURETTE SYNDROME

The frequency and pattern of bilineal transmission in families of 153 consecutive patients with Tourette syndrome (TS) (TS in both parents in 51 family sets), compared with 60 normal control subjects selected from public schools (20 family sets), were evaluated by interview and questionnaire for evidence of TS and associated OCD and ADD at Baylor College of Medicine, Houston, TX.

Evidence for bilineal transmission (both parents of patients with TS with tics, OCD, ADD, or a combination of these features) was found in 25% of patients with TS and 0% of controls. Unilineal transmission (one parent with tics, OCD, ADD) occurred in 57% of patients with TS and 5% of normal controls. More than 80% of patients with TS had at least one parent with tics, OCD, or ADD; and 38% of TS parents had two or more of the TS spectrum traits of tics, OCD, or ADD. In normal control subjects, the prevalence rate of observed tics was 0.4% and TS by history was 0.7% of the school population. Bilineal transmission of TS suggests a polygenic or recessive inheritance. (Hanna PA, Janjua FN, Contant CF, Jankovic J. Bilineal transmission in Tourette syndrome. Neurology Sept (1 of 2) 1999;53:813-818). (Reprints: Dr Joseph Jankovic, Department of Neurology, Baylor College of Medicine, 6550 Fannin St #1801, Houston, TX 77030).

COMMENT. Both parents of patients with TS are at increased risk of tics, OCD, and ADD. In the general school population, the prevalence of tics by history is about double that observed at individual interview, and less than 1%.