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%.