in relation to his other cognitive abilities.

MOVEMENT DISORDERS

MRI CHANGES IN SYDENHAM'S CHOREA

Cerebral MRIs of 24 children with Sydenham's chorea and 48 matched controls were compared at the National Institutes of Health, Bethesda, MD. The caudate, putamen, and globus pallidus in the chorea group were all significantly greater in volume, whereas the total hemispheres, prefrontal, midfrontal, or thalamus areas were not increased. (Giedd JN et al. Sydenham's chorea: Magnetic resonance imaging of the basal ganglia. Neurology Dec 1995;45:2199-2202). (Reprints: Dr Jay N Giedd, National Institutes of Health, NIMH, Child Psychiatry Branch, 9000 Rockville Pike, Building 10, Room 6N240, Bethesda, MD 20892).

COMMENT. A cross-reactive antibody-mediated inflammation of the basal ganglia is suggested as the pathophysiology of Sydenham's chorea. The authors admit that volumetric MRI is of limited diagnostic value because of large variability and overlap in basal ganglia size between chorea and control subjects.

Chorea in an infant with holoprosencephaly is reported from the College of Physicians and Surgeons, New York. (Louis ED et al. Pediatr Neurol 1995;13:355-357). MRI showed small, fused frontal lobes with hypoplastic caudate nuclei. This example of chorea associated with a congenital structural anomaly and undersized basal ganglia contrasts with the inflammatory hyperplasia of the caudate in Sydenham's chorea.

NEUROLOGIC SEQUELAE OF DANCING EYE SYNDROME

A persisting disability was found at long-term follow-up in 88% of 54 patients with dancing eye syndrome (DES) reported from the Hospital for Sick Children, Great Ormond Street, London. The disability was severe in 30 (62%), 34 (69%) had a motor disability, 29 (59%) had learning disabilities, and 23 (47%) had a combined motor and learning disability. Neurologic sequelae were independent of the severity of symptoms of the illness and age at onset. A malignancy was diagnosed in only 4: neuroblastoma in 3 and acute lymphoblastic leukemia in 1. An intercurrent illness, usually respiratory, preceded onset of DES in one half the cases. Presenting symptoms included ataxia, abnormal head and limb movements, and opsoclonus. Emotional outbursts of temper and affection were later features. A favorable initial response to corticotrophin or predisolone, observed in all patients, was not predictive of a good neurological prognosis. (Pohl KRE, Pritchard J, Wilson J. Neurological sequelae of the dancing eye syndrome. Eur J Pediatr March 1996;155:237-234). (Respond: Dr KRE Pohl, Newcomen Centre, Guys Hospital, St Thomas's Street, London SE1 9RT, UK).

COMMENT. Dancing eye syndrome (opsoclonus-myoclonus, infantile myoclonic encephalopathy) presents in infancy or early childhood (93% under 3 years) and neurologic sequelae may persist into adult life. Speech deficits, described as occasional in the above series, were more prominent in patient series reported from the Children's Memorial Hospital, Chicago, and the Children's National Medical Center, Washington, DC. (see Ped Neur Briefs Jan 1996;10:2).