Serial CT and MRI in outcome prediction of mild to moderate head injury. A series of 67 adults had CT on admission and MRIs within 1-3 and 6-12 months after injury. Outcome was worse in patients with edema and lesions on CT, and also in those with MRI lesions. Early MRI showing frontal lesions and late MRI with focal atrophy in frontotemporal regions, in combination with duration of amnesia, were predictive of outcome. (van der Naalt J, et al. Ann Neurol July 1999;46:70-78).

HEREDO-DEGENERATIVE DISEASES

IRON STORAGE IN FRIEDREICH'S ATAXIA

To test the hypothesis that iron is increased in the cerebellum of patients with Friedreich's ataxia (FA), a multigradient echo magnetic resonance sequence for the three-dimensional imaging of brain iron-induced contrast was used in 12 patients and 23 normal subjects examined at the National Institutes of Health, Bethesda, MD. Relaxation rate (R2) values, the inverse of T2, in the unaffected globus pallidus were equal in FA patients and controls, but R2 values in the dentate nucleus of FA patients were significantly higher. These R2 values reflect an increased iron concentration in the dentate, which supports the hypothesis of oxidative damage as the mechanism for FA. (Waldvogel D, van Gelderen P, Hallett M. Increased iron in the dentate nucleus of patients with Friedreich's ataxia. Ann Neurol July 1999;46:123-125). (Respond: Dr Mark Hallett, NINDS, NIH, Bldg 10, Rm 5N226, 10 Center Drive, MSC-1428, Bethesda, MD 20892).

COMMENT. Increased iron in the dentate nucleus of patients with Friedreich's ataxia, demonstrated by magnetic resonance, points to oxidative damage as the pathogenesis.

LAMOTRIGINE THERAPY IN NEURONAL LIPOFUSCINOSIS

Lamotrigine (LTG) long-term anticonvulsant therapy was evaluated in 29 patients, aged 6-28 years (mean, 14 years), with juvenile neuronal ceroid lipofuscinosis (JNCL), followed for 1-6 years (mean, 3 years) at the Hospital for Children and Adolescents, University of Helsinki, Finland. An initial dose of 0.1-0.5 mg/kg/day was increased every 2 weeks up to a maintenance dose of 1.25-15 mg/kg/day. After 1 year, seizures were decreased by more than 50% in 10, and seizures became less severe in 9 of 22. General patient well-being was improved in 18 of 28. LTG monotherapy was continued in 13 of 19 patients. (Aberg L, Kirveskari E, Santavuori P. Lamotrigine therapy in juvenile neuronal ceroid lipofuscinosis. Epilepsia June 1999;40:796-799). Reprints: Dr L Aberg, Hospital for Children and Adolescents, Pediatric Neurology, PL 280, 00029 HYKS, Finland).

COMMENT. The classical form of JNCL has an onset between 5 and 8 years with visual loss followed by progressive psychomotor retardation, epilepsy beginning at 8 to 13 years, extrapyramidal signs, and behavior disorders. Seizures are usually generalized tonic-clonic and partial, occasionally absence or atonic, and in the final stages of the disease, sometimes myoclonic. Lamotrigine may be an effective anticonvulsant in patients with JNCL.