Epilepsy

Koh et al. (p. 915) address the cause of the hippocampal sclerosis and the cognitive deficits seen in adults with temporal lobe epilepsy. The kainic acid induction of seizures early in life in the young rat predisposed animals to later seizure-induced injury even when there was no direct evidence of seizures having caused injury to the young rat brain. The accompanying editorial by Theodore and Wasterlain (p. 898) discusses the therapeutic implications of the potential benefit of eliminating seizures in young patients. ♦ Kälviäinen et al. (p. 922) detected asymptomatic concentric visual field defects in 13 of 32 (40%) patients treated with vigabatrin monotherapy versus none in 18 carbamazepine monotherapy patients or in 18 controls. Vigabatrin inhibits GABA transaminase and may be toxic to GABA-ergic amacrine cells. This study establishes that vigabatrin alone is toxic; in earlier studies, other drugs were used concurrently.

Dementia

DeDeyn et al. (p. 946) report a controlled trial of risperidone and haloperidol for treatment of behavioral symptoms in demented patients. Both agents were effective but risperidone had fewer extrapyramidal side effects. The accompanying editorial by Cummings and Knopman (p. 899) emphasizes the importance of controlling behavioral

symptoms in terms of their many costs—to the patient with dementia, to his or her family, and to society. They also note the potential limitations of the study and of risperidone. ♦ McKeith et al. (p. 902) provide a progress report on dementia with Lewy bodies (DLB) and review findings that have taken place within the 3 years since their first summary. It is now possible to diagnose a major proportion of DLB patients with a high degree of accuracy, but many still cannot be recognized. Initial success in open treatment studies justifies proceeding with controlled trials of anticholinesterase agents.

Neuromuscular disease

The expedited article by Matsuda et al. (p. 1119) reports that the newly recognized protein dysferlin is localized to the muscle membrane. Dysferlin mutations cause two clinically distinct myopathies: Miyoshi distal myopathy and a form of limb girdle dystrophy. The identical dysferlin mutation can cause either phenotype. It is clear that the phenotypic heterogeneity must relate to other genetic or acquired influences. ♦ Katz et al. (p. 1071) call attention to a distinctive motor neuron syndrome in which the upper limbs develop severe wasting and weakness. The condition results in a neurogenic “man-in-a-barrel” syndrome; it is relatively slowly progressive and typically is confined to lower motor neurons.

Stroke

Raaymakers, for the MARS Study Group (p. 982), reports a prospective study of the chance of intracranial aneurysms being detected by magnetic resonance angiography in first-degree relatives of patients with subarachnoid hemorrhage. The 4% risk of aneurysm was not increased by other risk factors. ♦ Greenberg et al. (p. 1135), in a Brief Communication, report a prospective study of 24 patients with probable or possible amyloid angiopathy who had MRI within 1.5 years following their initial hemorrhage. Gradient-echo MRI detected additional hemorrhages in 38% of patients.

Headache

Gervil et al. (p. 995) report a study of migraine without aura. They first argue for migraine with aura being a disease distinct from migraine without aura. The distinction suggests that the etiology of the two types of migraine should be considered separately. In a population-based twin study, genetic factors played a major role in the etiology of migraine without aura. ♦ Vitamin A toxicity has long been known to cause intracranial hypertension. Jacobson et al. (p. 1114) compared 16 women with idiopathic intracranial hypertension (IIH) with various controls (including obese subjects) and found that serum retinol was elevated in IIH patients. The basis for the elevation was not clear; there was no evidence for excessive vitamin A in the diet.

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