Treatment and quantitation of MS progression
There is convincing evidence that clinical and MRI exacerbations of MS are prevented by interferon beta treatment. Ge et al. (p. 813) studied the effect of approximately 24 months of glatiramer acetate (Copaxone) on quantitive MRI measures in patients with relapsing/remitting MS. Both T1-enhanced lesions and brain atrophy were decreased by glatirameter acetate (or placebo). ◆ The development of treatment for chronic progressive and secondary progressive MS has been hampered by lack of quantitative measures. In a study of 26 patients with MS versus control subjects, Fox et al. (p. 807) showed that volumetric MRI documents progression of both cerebral atrophy and ventricular enlargement in MS. The accompanying editorial by Jagust and Noseworthy (p. 782) points out the importance of this article for future assessment of new treatments for progressive CNS injury in MS. ◆ Related to the same issues, Cohen et al. (p. 802) studied a new clinical outcome measure: the MS Functional Composite (MSFC). The MSFC assesses leg, arm, and cognitive function. It performed extremely well in terms of intra- and inter-rater reliability.

Frontotemporal dementia (FTD) with parkinsonism
Kertesz et al. (p. 818) studied the clinical and pathologic features of a kindred with FTD and parkinsonism that does not appear to be linked to the chromosome 17 tau locus. The accompanying editorial by Higgins and Mendez (p. 784) reviews FTD (Pick's disease), particularly the autosomal dominant forms that are caused by mutations in the protein tau—"tauopathies." The fact that tau mutations were not detected by Kertesz et al. makes it likely that autosomal dominant FTD parkinsonism can be caused by two or more genes.

Estrogens and AD
Manly et al. (p. 833) found that women with AD had lower estradiol levels than control subjects. Although this study suggests that low estrogens result from AD, Henderson et al. (*Neurology* 2000;54:295–301) found that replacing estrogens was not helpful.

Visual hallucinations: direct occipital cortex stimulation
Lee et al. (p. 849) characterized the visual responses of 23 patients with epilepsy who had electrical stimulation of cortex by means of subdural grids placed over the occipital cortex. They were able to localize the areas responsible for many visual phenomena. As Galetta's accompanying editorial discusses (p. 785), the experience of Lee et al. provides exquisite cortical localization in comparison to data that can be obtained by functional MRI or PET.

Aspirin for subarachnoid hemorrhage?
The treatment of delayed cerebral ischemia (DCI) after subarachnoid hemorrhage remains unsatisfactory. Because increased platelet activity has been hypothesized as a cause for DCI, Hop et al. (p. 872) conducted a controlled trial of aspirin versus placebo in 50 patients who had early surgery for a ruptured aneurysm. The trial showed no increase in hemorrhage in ASA-treated patients and a trend toward benefit from ASA. A larger trial is planned.

Symptomatic versus asymptomatic carotid stenosis
Powers et al. (p. 878) performed PET scans on 111 patients with carotid occlusion: 30 who never had cerebrovascular symptoms and 81 who had symptoms. They examined risk factors for stroke and then followed patients semiannually for approximately 3 years. Only 1 of 30 patients who had no symptoms had a subsequent stroke (versus 15 strokes/81 with symptoms); PET showed evidence of hemodynamic compromise in many of the patients with symptoms.

Macrocephaly in neurofibromatosis
Moore et al. (p. 914) correlated quantitative brain volumes with neuropsychological performance in 52 children with neurofibromatosis type 1. Increased volume of cerebral gray matter and of the corpus callosum was associated with diminished performance. The authors postulate that delayed developmental apoptosis is responsible for both.

HIV dementia
Berger et al. (p. 921) studied postcontrast MRI enhancement in demented versus nondemented HIV-infected subjects. Prominent basal ganglia enhancement correlated with the extent of dementia. Blood–brain barrier disruption in the basal ganglia appears to underlie the subcortical dementia in HIV-infected patients.

Valproate in traumatic brain injury?
Dikmen et al. (p. 895) randomized 279 adults seen within 24 hours of head injury to valproate (either 1 or 6 months) or 1 week of phenytoin. Valproate had no significant effects on neuropsychologial assessment and did not prevent post-traumatic seizures. Moreover, it was of no benefit to cognition. Valproate does not appear to be a useful prophylaxis for post-traumatic seizures.

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