Multiple sclerosis

Two major issues in developing better treatments for MS are 1) understanding how currently available treatments produce their benefit and 2) establishing biomarkers that can quantitate the progression of subclinical disease, as MS clearly progresses without obvious clinical signs. There is good evidence that MRI is such a biomarker. Less expensive and more convenient surrogate markers are needed. The article by Waubant et al. (p. 1397) reports that the ratio serum metalloprotease-9 (MMP-9) activity relative to a tissue inhibitor of MMP correlated with MRI evidence of progressive disease activity. ♦ Trojano et al. (p. 1402) examined MMP-9 during treatment of relapsing-remitting MS with interferon-beta 1b. A decline in MMP-9 correlated with response to treatment. ♦ The editorial by Bever and Rosenberg (p. 1380) considers the importance of changes in MMP-9 as well as whether the reported changes in MMP-9 are the cause or the result of improvement. Moreover, putative therapeutic agents that inhibit MMP-9 will soon enter clinical trials.

PD and progressive supranuclear palsy

Two or more genes cause PD. These genes are rare but have stimulated renewed interest into a possible genetic basis for PD. The article by Nicholl et al. (p. 1415) looked at five genes that have been considered candidates for PD. In the large population they studied, none were correlated with PD. ♦ Woo et al. (p. 1570) studied the brains of patients with Lewy body disease (LBD). There was no evidence from studies of brain genomic DNA or of RNA for any specific transcript of RNA being associated with LBD. ♦ The editorial by Markopoulou and Langston (p. 1382) considers how the strategies used by these two

studies provide evidence against a role of some candidate genes in the pathogenesis of PD. They also point out why previous studies may have provided misleading information and discuss experimental strategies that may lead to new insights in the pathogenesis of PD. ♦ Higgins et al. (p. 1421) are pursuing the possibility that mutations in the *tau* gene cause progressive supranuclear palsy (PSP). In their study, all 22 unrelated PSP patients had an identical difference in *tau* gene sequence: four specific polymorphisms. It is not yet clear why these variations in the *tau* gene are seen in all PSP patients. Do they increase susceptibility to PSP? Are they markers for a nearby gene? Or do they indicate that *tau* mutations are the cause of PSP?

Dystonia, essential tremor

Brin et al. (p. 1431) and Brashers et al. (p. 1439) studied botulin toxin type B in patients with cervical dystonia. Type B toxin was effective both in patients who were still responsive to type A toxin and in patients who had become resistant to type A toxin. The benefit persisted for 12 to 16 weeks in both groups. ♦ Pahwa et al. (p. 1447) studied nine patients disabled by essential tremor who were treated with sequential implantation of thalamic stimulators. Ventral intermediate nucleus on one side was implanted, followed 6 to 26 months later by implantation on the other side. Blinded evaluations at 3 months as well as evaluations at 6 and 12 months showed marked improvement in comparison with preoperative scores. Six of nine patients experienced dysarthria during stimulation.

Headache, migraine, cerebral venous thrombosis, and intracranial hypertension

The Rozen et al. (p. 1468) epidemiologic study of migraine in Olmsted County documented an

increasing incidence of headache, particularly migraine, in women over the decade 1980–1990. ♦ Bioussé et al. (p. 1537) identified 59 patients with cerebral venous thrombosis in whom the only symptoms and signs were those of increased intracranial pressure. Most patients had both headache and papilledema but a few had only one or the other. CT did not show the venous abnormalities in a majority of patients. Diagnosis depended on MRI including MR venography. Studies of the course and responses to treatment, although uncontrolled, gave indications of a relatively good prognosis.

Epilepsy

Sawrie et al. (p. 1511) obtained follow-up studies of memory 6 to 12 months postsurgery in 65 patients who had temporal lobe resections for temporal lobe epilepsy (TLE). Operated patients were compared with unoperated TLE patients. As others have found, the authors documented objective memory loss postsurgery in one quarter to one half of the patients. Importantly, they showed that patients with memory loss did not complain of it. Symptoms of memory loss were infrequent and, when present, were not related to the objective loss that was documented.

Ataxia

The Clinical/Scientific Note by Pellecchia et al. (p. 1606) reports a 34-year-old man in whom progressive ataxia and peripheral neuropathy were associated with celiac disease. Importantly, there were no clinical symptoms or signs of celiac disease and the diagnosis depended on the findings on duodenal biopsy and serum antigliadin antibodies. The patient improved on a gluten-free diet. The mechanism of cerebellar injury in celiac disease is not clear. Vitamin E deficiency was excluded.

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