# June 13 Highlights

**Biological activity of different β-interferons**
There are three different interferon-β (IFNβ) products (two are approved in the United States). There are no in vivo data comparing them directly and their in vitro activities have not been compared. Deisenhammer et al. (p. 2055) studied patients with MS on the three IFNβs (excluding patients with neutralizing antibodies) for their effect on markers of IFN activity: macrophage antigens (MxA) and soluble vascular cell adhesion molecules (sVCAM). They found IFNβ-1b induced higher marker levels than IFNβ-1a. ◆ The accompanying editorial by Reder and Antel (p. 2034) accentuates the importance of defining the actions of IFNβ, notes the technical limitations of such studies, and points out that large ongoing clinical trials will make it possible to determine which effect of IFNβ correlates with clinical benefit.

**Worsening of MS with recombinant human G-CSF**
Openshaw et al. (p. 2147) describe four patients who worsened abruptly with human granulocyte colony stimulating factor in the context of stem cell transplantation treatment of MS (of 10 patients). One patient died of respiratory failure. The mechanism of worsening is not known but the observation is important both in the context of other such transplants and because the factors responsible for the frequent, spontaneous worsening of MS are unknown.

**Estrogens in AD**
A recent *Neurology* article by Henderson et al. (2000;54:295–301) showed estrogen replacement therapy (ERT) to be ineffective in AD. In this issue, Wang et al. (p. 2061) report a controlled study from Taiwan that showed no effect of estrogen in AD on cognition, mood, or cerebral blood flow. ◆ The accompanying editorial by Marder and Sano (p. 2035) discusses the three negative trials of ERT for AD, and considers why the clinical trials contrast with the epidemiologic and basic data suggesting that estrogen should be beneficial. They also note that other therapeutic strategies with estrogen might be effective.

**Treatment of HIV-associated neuropathy**
HIV-sensory neuropathy is the most common painful syndrome in HIV-infected patients. Two articles assess treatment of sensory neuropathy from HIV infection. Simpson et al. (p. 2115) performed a 14-week randomized, controlled trial of lamotrigine in 42 patients. A significant reduction in pain was noted in lamotrigine-treated patients. Skin rash–necessitated drug cessation was more frequent with lamotrigine use (20%) than prior experience with its use for epilepsy would have predicted. Despite this side effect, a larger trial of lamotrigine appears indicated. ◆ Martin et al. (p. 2120) examined whether highly active antiretroviral therapy (HAART) can benefit sensory nerve function. A longitudinal study of 49 HIV-1–infected patients showed that when HAART improved immunodeficiency, pain and temperature perception improved. ◆ The accompanying editorial by Berger and Nath (p. 2037) considers these two articles and another recent article by McArthur et al. (2000;54:1080–1088), which showed that human nerve growth factor decreased pain intensity. The authors point out that although they are promising, these studies must be extended to better define the relationships between specific therapy with HAART, neurotoxic effects of other HIV treatment, and symptomatic treatments with lamotrigine and other agents.

**tPA for stroke with carotid dissection**
A brief communication by Derex et al. (p. 2159) reports tissue plasminogen activator (tPA) treatment of 11 patients with internal carotid artery dissection and an ischemic stroke. tPA was given within 7 hours. Four of the 11 patients made an excellent recovery; one symptomatic hemorrhage occurred.

**Hallpike and the founding of neurotology**
In a well-researched historical article, Baloh (p. 2138) describes the origins of otology from the pioneering work by Ménière that first developed science in a field dominated by quacks as a backdrop for considering the extraordinary, enduring contributions of Charles Hallpike: neurotologic tests that remain in wide use by neurologists today.

**New-variant CJD presenting as focal epilepsy**
A patient described by Silverdale et al. (p. 2187) presented at age 26 with seizures that persisted for 6 years before the patient showed full-blown signs of Creutzfeldt-Jakob disease, subsequently shown to be the new variant prion suspected of being associated with bovine spongiform encephalopathy.

 