### **December 10 Highlights**

#### **Neuropathy**

Carpo et al. (p. 2206) report that GM-1 antibodies have a relatively poor sensitivity and specificity for diagnosis of multifocal motor neuropathy. Although this study appears to question the clinical use of this test, the accompanying editorial by Holloway and Feasby (p. 1905) points out that reports of most proposed new diagnostic tests meet only some of the criteria essential to establish the value of the test in clinical practice. [Neurology has adopted the standard CONSORT requirements for clinical trials. We plan to develop similar criteria for proposed diagnostic tests.]

#### Prion disease

Puoti et al., in their expedited publication (p. 2173), report that brain tissue from over a third of patients with Creutzfeld-Jakob disease (CJD) contains two different types of prion protein. Moreover, they suggest the possibility that all cases contain a mixture of prion types that may be related to regional neuroanatomic differences. ◆ As accented by the editorial by Dickson and Brown (p. 1903), Puoti et al. suggest that purported classifications of CJD and its variants based on molecular characteristics of prion protein obtained from a limited brain sample need to be interpreted with caution. Futhermore, the use of prion protein typing in epidemiologic association studies of transmissible forms of prion disease, such as the relationship of bovine spongiform encephalopathy to new variant CJD, may need reappraisal.

#### **Dementia**

Ott et al. (p. 1937) report definitive results from a longitudinal, prospective study of more than 6,000 elderly subjects (The Rot-

terdam Study), showing that Type 2 diabetes is a major risk factor for dementia. The editorial by Lovestone (p. 1907) considers the mechanism(s) by which diabetes causes dementia. ◆ Also from The Rotterdam Study is the article by Mehta et al. (p. 1959), which examines whether head trauma with or without the APOE- $\epsilon 4$  allele proved to be a risk factor for dementia with AD. Head trauma with loss of consciousness was not a risk factor with or without consideration of  $APOE - \epsilon 4$  status.  $\bullet$  In a longitudinal study of subjects ranging in age from 59 to 71 years, Tzourio et al. (p. 1948) examined the relationship between treated and untreated hypertension and decline in MMSE scores. Cognition declined in a relatively short period (2 years) in hypertensive patients. Treated patients declined less than untreated. ◆ It is unclear whether AD causes depression or whether depression is a risk factor for AD. Berger et al. (p. 1998) analyzed data from a 3-year longitudinal study of 222 subjects who were older than 74 years and found that patients who developed AD were depressed before developing AD cognitive signs. Depressive symptoms may be an early sign of AD. ◆ Stern et al. (p. 1942) prospectively studied patients with AD and found that patients with higher levels of education and occupational status declined more rapidly than those with low occupational status, consistent with the hypothesis that patients with high levels of education and attainment have exhausted their cognitive reserve by the time they are diagnosed with AD.

# Cortical excitability: effects of the menstrual cycle

Smith et al. (p. 2069) use paired transcranial magnetic stimula-

tion (TMS) to study cortical excitability during the follicular (estrogen rising, progesterone low) and luteal (both estrogen and progesterone high) phases of the menstrual cycle. They found more TMS inhibition of excitability during the luteal phase—equivalent to the effects of benzodiazepine drugs.

#### Oromandibular dystonia

Tan and Jankovic (p. 2102) report an extensive experience (162 patients) with long-term treatment of oromandibular dystonia (of various etiologies) with botulinum toxin injection of masseters or submentalis muscle complex. Major benefit persisted in most patients. The mean duration of response was 16 weeks with most patients having major, long-term benefit.

## Neurogenic orthostatic hypotension

Norepinephrine release by sympathetic neurons is impaired in neurogenic orthostatic hypotension. Freeman et al. (p. 2151) studied DL-DOPS, a norepinephrine precursor, in 10 patients with orthostatic hypotension (multiple system atrophy or pure autonomic failure). They found marked improvement in orthostatic BP decrease with DOPS in comparison with placebo. There was a trend toward symptom improvement, but the study was very short—only a single day.

## Sodium channelopathy in hypokalemic periodic paralysis

The expedited publication by Bulman et al. (p. 1932), which reports a family in which a mutation in a Na-channel is associated with hypokalemic attacks of weakness, challenges the suggestion that hypokalemic periodic paralysis is a calcium channelopathy.



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