

Parkinson's disease in welders: Evidence for an environmental cause of Parkinson's disease

Racette et al. (p. 8) compared 15 career welders with two control groups with Parkinson's disease. Welders had a younger mean age at onset of Parkinson's disease (46 versus 63 years). Otherwise, Parkinson's disease was similar in the three groups: clinical features, PET scan abnormalities, and pharmacologic responses. ♦ As noted in the accompanying editorial by Rajput (p. 4), there are a number of well-recognized causes of parkinsonism/parkinsonism syndromes, but this study provides some of clearest evidence that typical Parkinson's disease may have environmental triggers.

Impaired motor function in elderly normal subjects: Abnormality of substantia nigra by ultrasound

In Parkinson's disease patients there is increased echogenicity of the substantia nigra on transcranial sonography. Berg et al. (p. 13) studied the substantia nigra by transcranial sonography in 93 healthy elderly subjects without Parkinson's disease. Increased echogenicity of the substantia nigra correlated with symptoms of Parkinson's disease and with abnormal motor function. Thus, abnormal substantia nigra could be either a risk factor for Parkinson's disease, or could be associated with the impaired motor function found in many elderly subjects.

Predisposition to progressive supranuclear palsy?

Baker et al. (p. 25) compared first-degree relatives of progressive supranuclear palsy patients to matched controls in

their performance on a Parkinson's disease test battery that is able to detect early disease in Parkinson's disease patients. Of progressive supranuclear palsy relatives, 39% were abnormal, versus none of controls. This striking abnormality suggests either a carrier state for progressive supranuclear palsy or the effect of a shared environmental exposure.

Competency in Parkinson's disease with impaired cognition

Assessment of competency—the capacity to consent to treatment—is of major importance in the care of patients and in the study of new treatments. Dymek et al. (p. 17) used a standardized competency measure and neuropsychological tests to compare 20 Parkinson's disease patients with impaired cognition with control subjects. Parkinson's disease patients had major deficits in competency, reflecting primarily their abnormalities in executive function.

Effects of valproate and other AEDs on androgens in men with epilepsy

Women taking valproate often have endocrine disorders. Rättyä et al. (p. 31) studied men on monotherapy with valproate, carbamazepine, and oxcarbazepine and found that 57% of patients on valproate had increased serum androgens. Carbamazepine lowered dehydroepiandrosterone sulfate levels.

Cognitive decline in middle-aged adults

Knopman et al. (p. 42) report vascular risk factors for decline in cognition as assessed in a longitudinal study: the

Atherosclerosis Risk in Communities (ARIC) cohort. Tests of cognition were administered 6 years apart to 8,729 white subjects and 2,234 African-American subjects aged 45 to 70 years. Diabetes and hypertension, but not lipid levels or smoking status were associated with cognitive decline.

Mycophenolate mofetil (MM) for myasthenia gravis and other neuromuscular diseases.

Two articles report uncontrolled trials of MM in patients with neuromuscular diseases. MM blocks purine synthesis and has had substantial use in preventing organ rejection. Ciafaloni et al. (p. 97) report that eight of 12 patients with myasthenia gravis improved. ♦ Chaudhry et al. (p. 94) report MM use in 38 patients (myasthenia gravis, inflammatory myopathy, chronic demyelinating neuropathy). Improvement was noted in 24. The studies differ in the time course of improvement: the former more rapidly (2 weeks to 2 months) than the latter (5 months). Toxicity was minimal.

Genetic cause of 5-FU-related multifocal inflammatory leukoencephalopathy (MIL)?

Franco and Greenberg (p. 110) report a 65-year-old woman in whom MIL developed following 5-FU treatment of squamous cell carcinoma. She was not taking levamisole, which has usually been coadministered in other MIL cases. Subsequently, the patient was found to be partially deficient in dihydropyrimidine dehydrogenase (DPD). Because DPD catabolizes 5-FU, this case of partial PD deficiency suggests that other such patients are at risk for MIL and possibly other 5-FU toxicity.

Neurology[®]

January 9 Highlights
Neurology 2001;56;1
DOI 10.1212/WNL.56.1.1

This information is current as of January 9, 2001

Updated Information & Services	including high resolution figures, can be found at: http://n.neurology.org/content/56/1/1.full
Citations	This article has been cited by 9 HighWire-hosted articles: http://n.neurology.org/content/56/1/1.full##otherarticles
Permissions & Licensing	Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: http://www.neurology.org/about/about_the_journal#permissions
Reprints	Information about ordering reprints can be found online: http://n.neurology.org/subscribers/advertise

Neurology® is the official journal of the American Academy of Neurology. Published continuously since 1951, it is now a weekly with 48 issues per year. Copyright . All rights reserved. Print ISSN: 0028-3878. Online ISSN: 1526-632X.

