Complications of subthalamic stimulation (DBS)

Lyons et al. assessed the surgical and hardware complications in 81 Parkinson patients undergoing subthalamic DBS. There were no deaths or permanent neurologic deficits. Complications included: one with hemorrhage, two systemic infections requiring removal, three infections requiring pulse generator removal, 10 misplaced leads, and 26 hardware complications.

see page 612

Downbeat nystagmus and vestibulo-ocular reflex (VOR)

Glasauer et al. compared VOR responses to head impulses in patients with downbeat nystagmus and in controls. No differences were found. Upward and downward VOR responses were symmetrical in patients, arguing against the hypothesis that DBN is caused by vestibular dysfunction.

see page 621

Therapy of gravity-dependent downbeat nystagmus

Helmchen et al. showed that the potassium channel blocker 3, 4-diaminopyridine specifically improves the gravity-dependent component of downbeat nystagmus. This effect may be exerted on the deficient vestibulo-cerebellar inhibition of the physiologically overactive otolith-ocular reflex.

see page 752

The editorial by Halmagyi and Leigh reviews the causes and likely mechanisms of downbeat nystagmus (DBN), as well as the causes of upbeat nystagmus, and notes that in perhaps half of all patients with DBN, no cause is found, and the condition progresses slowly, if at all. They note that these two studies suggest that asymmetries of the rotational VOR from the semicircular canals are less important in DBN than in an otolithic (gravity-dependent) imbalance.

see page 606

ApoE4 promotes sphingolipid and sterol accumulation

In HIV-infected patients with an *APOE4* genotype, Cutler et al. found greater accumulations of sphingomyelin, ceramide, and sterols in multiple brain regions. An *APOE 3/4* or *4/4* genotype may predispose patients to neuronal damage by disrupting sphingolipid and sterol metabolism.

see page 626

*The accompanying editorial by Christopher Power and Kamala Patel notes that these changes in lipids may begin to explain the effect of *APOE4* to increase the risk of degenerative diseases as well as inflammatory diseases such as HAD. This study follows other work from the same group showing that sphingomyelin, ceramide and the lipid peroxidation product 4-hydroxynonenal are increased in the brains and CSF of patients with (HAD) HIV-associated dementia HAD compared to HIV/AIDS patients without HAD and uninfected control patients. Altering lipid metabolism led to changes in ceramide levels that correlated with increased neurotoxicity caused by the viral proteins Tat and gp 120. Lipidomics—the large-scale study of lipid quantity and function—has important ramifications on oxidative metabolism because lipid levels and their peroxidation may impact directly on cellular survival and function.*

see page 608

Economic value of donepezil in moderate/severe AD

Feldman et al. report on the pharmacoeconomics of donepezil treatment of severe Alzheimer disease. They report that donepezil treatment for six months within a randomized, placebo-controlled trial is associated with a total societal cost saving of \$224 per patient.

see page 644

Efficacy of donepezil in mild cognitive impairment

Salloway et al. compared the cholinesterase inhibitor donepezil vs placebo for 24 weeks in 270 patients with mild cognitive impairment (MCI). There was no benefit to the primary outcome variable but donepezil-treated patients demonstrated significant improvements in cognitive function on several secondary outcome measures.

see page 651

continued on page 605

Affective disorders in *DYT1* dystonia families

Heiman et al. assessed affective disorders in *DYT1* mutation carriers, both those affected and unaffected with dystonia, and used family members without the mutation as controls. They found that the rates of early-onset recurrent major depression were increased in both groups of mutation carriers compared to non-carriers, suggesting that early-onset recurrent depression is an expression of *DYT1*.

see page 631

"This study provides further support for the role of the basal ganglia and dopaminergic neurotransmission in the regulation of mood."

*The accompanying editorial by Irene Richard and William McDonald points out that the Heiman et al. study has an imaginative research design which suggests that carrier status confers an increased risk of developing depression. They note that the data demonstrating equivalent rates of depression in *DYT1* carriers with and without dystonia*

motor symptoms provide support for the notion that depression is not simply the expected consequence of a disabling neurologic disorder. They also highlight the link between basal ganglia disease and depression in other movement disorders such as Parkinson disease (PD). Since mutant torsinA may affect pre-synaptic release of striatal dopamine resulting in increased dopamine turnover, it is possible that comorbid depression in dystonia shares similar mechanisms to the depression frequently encountered in PD.

see page 610

Pre-diabetes, diabetes, and cognitive impairment

Yaffe et al. studied 7,027 older women and found that those with pre-diabetes as well as those with diabetes have worse cognitive performance, and are at increased risk for developing cognitive impairment, including mild cognitive impairment and dementia.

see page 658

Depression and pain in neurology outpatients

Williams et al. evaluated depression and pain symptoms in nearly 500 neurology outpatients. Those with co-existing depression and pain had more symptoms and outpatient utilization during the next 12 months, suggesting a need for improved screening and treatment of depression and pain in these patients.

see page 674

Mycophenolate mofetil in immune demyelinating neuropathy

Gorson et al. found no differences in clinical or disability scores in 21 immune neuropathy patients treated with mycophenolate mofetil. Only three patients with CIDP and one with IgM neuropathy improved, allowing a reduction of steroid or IVIG therapy.

see page 715

Neurology®

August 24 Highlights

Neurology 2004;63;604-605

DOI 10.1212/01.WNL.0000139378.17464.2F

This information is current as of August 23, 2004

Updated Information & Services

including high resolution figures, can be found at:
<http://n.neurology.org/content/63/4/604.full>

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.