Hippocampal DWI lesions in transient global amnesia

*Lesion development: no significant DWI lesion hyper-
acutely, but appearance after 48 hours.*

Sedlacek et al. report the delayed appearance of punctuate DWI lesions on one or both sides of the hippocampus in 26/31 patients. This delayed appearance of lesions on DWI may account for previously conflicting reports on the presence of DWI abnormalities in this condition.

see page 2165

In the accompanying editorial David Tong and Murray Grossman discuss the possible mechanisms of TGA in light of the observed DWI findings. While intriguing, the DWI data do not provide sufficient evidence to definitively establish that TGA is a transient ischemic attack due to a vascular cause. Whether such imaging information should alter clinical management is debatable, and likely depends upon the clinical assessment of the patient's overall cerebrovascular risk profile.

see page 2154

Possible mechanisms underlying Todd's paresis

Gallmetzer et al. observed postictal paresis in 44 patients with focal epilepsy. Clonic activity was observed in 56%, dystonic posturing in 48%, and ictal limb immobility in 25% of the preceding seizures, suggesting an active inhibitory process involved in the pathogenesis of postictal paresis.

see page 2160

Atkins diet for narcolepsy?

Husain et al. studied the effects of a low-carbohydrate, ketogenic diet in nine patients with narcolepsy. A modest improvement in daytime sleepiness was observed on this diet.

see page 2300

Seizure outcome with solitary cysticercus granuloma (SCG)

Risk of recurrence of seizures with and without calcific residue in CT scans. Those with calcification have a higher risk of recurrence ($p = 0.01$). Dashed line, calcific residue; uninterrupted line, no calcific residue.

Rajshekhkar and Jeyaseelan studied long-term seizure outcome in 185 patients with a resolved SCG following early withdrawal of antiepileptic drugs. At a mean follow-up of 65.8 months, 157 (84.9%) patients were seizure free while 28 (15.1%) reported recurrence of seizures. Seizure recurrence was associated with history of more than two seizures, breakthrough seizures, and a calcific residue of the SCG on the CT scan.

see page 2236

The clinical-DWI mismatch identifies brain at risk of infarction

Ischemic brain tissue at risk of infarction may be identified by a mismatch between the severity of the acute clinical manifestations and the DWI lesion. Dávalos et al. found that patients with NIH stroke scale score ≥ 8 and ischemic volume on DWI ≤ 25 mL within 12 hours from stroke onset have a higher probability of infarct enlargement and early neurologic deterioration.

see page 2187

continued on page 2149

Seizure-related behavior changes in household dogs

Kirton et al. examine the seizure-related behavior in dogs living with children with epilepsy. Forty percent of dogs responded to seizures while 40% of these could accurately anticipate seizures. Families with dogs reported higher quality of life scores.

see page 2303

Pain in Parkinson disease

Studying 36 patients with PD with predominantly unilateral disease, Djaldetti et al. found that while tactile and warm perceptions were normal, heat pain threshold was lowered in PD patients who experienced pain.

see page 2171

The accompanying editorial by Beata Buzas and Mitchell B. Max notes that the findings of Djaldetti et al show that parkinsonian motor symptoms and spontaneous pain associated with large increases in sensitivity to heat pain are convincing and novel. Researchers in pain, motor systems, and neurodegeneration must join forces to explain it. The sensory findings of the Djaldetti study were definitive, but in the opposite direction from predicted. Rather than the decreased thermal pain sensitivity found in thalamic strokes, the authors found increased evoked pain sensitivity in subjects with Parkinson's disease compared to normal subjects, especially those patients who previously described spontaneous pain. Buzas and Max suggest that non-dopaminergic systems must also be scrutinized in the search for the mechanisms of parkinsonian pain. PD affects noradrenergic, serotonergic, cholinergic, and peptidergic neurons and the degree of neurodegeneration in the locus ceruleus (LC) may be greater than that in the substantia nigra in Parkinson patients. The LC contains the largest group of noradrenergic neurons in the CNS. If degeneration of brainstem noradrenergic systems causes pain in PD, clinical trials with norepinephrine reuptake blockers (e.g., desipramine) or adrenergic agonists such as epidural or systemic clonidine might reduce pain.

see page 2156

Thalidomide neuropathy

In a prospective study, Briani et al. showed that 50% of patients treated with thalidomide for lupus erythematosus developed axonal neuropathy, mostly after 10 months of treatment, independent of cumulative dose.

see page 2288

Cavaletti et al. demonstrated that duration rather than total dose was the major contribution to this potentially severe side effect of the drug.

see page 2291

In commenting on these two articles, Stuart Apfel and Douglas Zochodne note that it was concern over earlier reports of peripheral neuropathy in adults that prevented the approval of this drug in the US by the FDA, largely averting the tragic consequences seen in 46 other countries. Since the 1998 FDA approval of thalidomide for cutaneous manifestations of erythema nodosum leprosum, thalidomide is now used or investigated for treatment of other dermatologic, gastrointestinal, and oncologic conditions. Reviewing the somewhat conflicting data of the two Neurology articles, Apfel and Zochodne recommend that when circumstances permit, the lowest effective dose of thalidomide should be used. When thalidomide is used, regardless of the dose, peripheral nerve function should be closely monitored through review of symptoms, regular neurologic examinations, and periodic screening of sensory nerve electrophysiology.

see page 2158

Neurology[®]

June 22 Highlights

Neurology 2004;62;2148-2149

DOI 10.1212/01.WNL.0000132258.36477.94

This information is current as of June 21, 2004

Updated Information & Services

including high resolution figures, can be found at:
<http://n.neurology.org/content/62/12/2148.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.