

Homocysteine, vitamin B₆ and vascular disease in AD patients

In their study, Miller et al. separated both AD patients and controls into those with and without evidence of vascular disease and examined homocysteine levels. They found that elevated homocysteine was associated with patients (and controls) who had vascular disease but not specifically those diagnosed with AD. In contrast, low vitamin B₆ status, which is seen with high homocysteine blood levels and which has been associated with poor memory in older adults was highly prevalent in the AD patients. The influence of homocysteine and B₆ status on AD pathogenesis or progression remains to be determined.

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Sachdev et al. showed in their study of healthy elderly individuals that higher plasma homocysteine levels were related to brain atrophy as suggested by increased size of the lateral ventricles.

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Dratzkowski et al. note that the folic acid use widely recommended for all women of childbearing potential was associated with neurologically symptomatic B₁₂ deficiency. While not unexpected, their report serves as a caution that use of folic acid (e.g., to treat elevated homocysteine levels) requires attending to B₁₂ status.

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The accompanying editorial by Toole and Jack considers how elevated plasma homocysteine levels might cause brain atrophy and contribute to dementia. They note the importance of the VISP and VITATOPS treatment trials that are examining the effect of treatment of hyperhomocysteinemia.

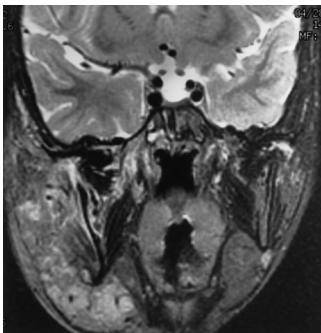
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Neurology residency training at the millennium

The survey of neurology training programs 1999–2000 by Corboy et al. documents that there has been some shift in virtually all programs from inpatient to outpatient training venues but the relative emphasis on inpatient testing persists. Not surprisingly, having the neurology rotation in the third year of medical school correlated with attracting more students into neurology. A lack of adequate financial support for program directors was identified as a major problem.

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Plexiform neurofibromas in neurofibromatosis type 1 (NF1)



Packer et al. review plexiform neurofibromas (PNF). PNFs are a common complication of NF-1 and are occasionally the sole manifestation of NF-1; typically causing cosmetic abnormalities, pain, or local nerve involvement. While treatment of PNFs is surgical, some lesions cannot be totally resected and other approaches are needed. The absence of neurofibromin, the protein product of the *NF-1* gene, initiates a cascade of other molecular events controlling growth. These events and other cellular phenomena are new targets for therapies for PNFs.

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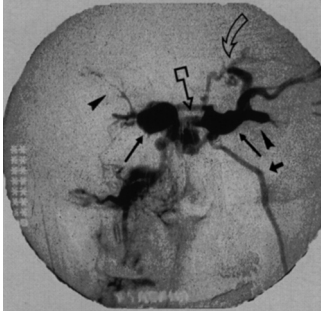
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Increased risk of Parkinson's disease (PD) after depression

Schuurman et al. found that patients who have had depression have a threefold greater risk for developing PD compared with those who never had a major depression. This risk increase was seen across different age groups and for both men and women.

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Predicting cortical venous drainage in cavernous sinus dural AVMs



Cavernous sinus dural AVMs that drain into cortical veins (CVD) have an increased risk for intracerebral hemorrhage and cerebral infarcts. Stiebel-Kalish et al. found that bilateral orbital signs and a postauricular bruit each predict CVD. Patients with bilateral orbital congestion should be recognized as being at increased risk for CVD.

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CO poisoning and white matter hyperintensities (WMHs)

Parkinson et al. compared 73 consecutive CO-poisoned patients with matched controls for WMHs and neurocognitive outcome. Subjects were studied longitudinally at 1 day, 2 weeks, and 6 months following poisoning. Thirty percent of CO-poisoned patients developed cognitive sequelae and 12% developed WMHs. The duration of loss of consciousness but not the initial carboxyhemoglobin concentration correlated with cognitive impairment initially and at 6 months.

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Subthalamic nucleus (STN) stimulation in PD: Results at 3 years

Romito et al. evaluated efficacy and safety of STN high frequency stimulation in 22 PD patients from 1 to 3 years after surgery. Motor disability and ADL scores improved and then did not deteriorate during follow up. Drug doses were reduced by over 60%. This procedure is now FDA-approved in the United States.

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Rapidly progressive hippocampal atrophy with partial epilepsy

Controversy exists regarding the role of epileptic seizures in the development of hippocampal atrophy and mesial temporal sclerosis. Worrell et al. report a patient with new onset partial seizures in whom there was rapidly progressive hippocampal atrophy (over 5 months), supporting the view that epileptic seizures can induce such hippocampal atrophy without antecedent temporal lobe lesions or injury.

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