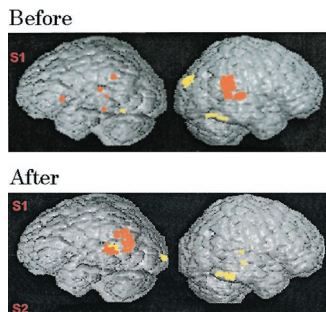


Treatment of dyslexia by remedial training?



Simos et al. found distinct, reduced activation of the posterior portion of the superior temporal gyrus in 8 patients with dyslexia (vs 8 controls). Two months of remedial intervention improved reading skills increased activities in all 8 subjects.

see page 1203

The accompanying editorial by Rosenberger and Rottenberg reviews the history of dyslexia, dating from Samuel Orton and the subsequent studies of Alvan Liberman characterizing the difficulty that the dyslexic person has in analyzing written text into phonemes—related to abnormalities of the dominant temporal lobe. They point out that Simos et al.'s study begs the question as to whether the normalization they see reflects true gain in proficiency, or simply a change in brain activity.

see page 1139

Giant axonal neuropathy (GAN) caused by mutations in the gigaxonin gene

Kuhlenbäumer et al. describe GAN caused by novel recessive mutations in the gigaxonin gene and raise the question as to whether heterozygous individuals may develop a mild subclinical neuropathy.

see page 1273

The accompanying editorial by Hermann and Griffin notes that GAN is an example of disorders affecting neurofilaments (NF) since mutations in gigaxonin alter NF in peripheral nerve axons. Other NF disorders include ALS, several types of muscular dystrophy and myopathy, Charcot Marie Tooth disease, as well as toxic neuropathies.

see page 1141

Hypertension in the elderly and dementia

In a community sample of 2,136 elderly (≥ 65 years old) in northern Manhattan, Posner et al. found that hypertension was not associated with Alzheimer disease or cognitive decline. However, hypertension was associated with vascular dementia, particularly in the presence of heart disease or diabetes.

see page 1175

Prevalence of neuropsychiatric syndromes in lupus

Using standardized definitions, Brey et al. determined the prevalence of neuropsychiatric syndromes in patients with lupus erythematosus. One or more syndromes were present in 80% of patients. Only 21% of patients were cognitively normal. Nervous system involvement is a frequent cause of morbidity in patients with lupus.

see page 1214

continued on page 1138

The pathogenesis of gluten ataxia

Hadjivassiliou et al. found that IgG anti gliadin antibodies bind to human cerebellar Purkinje cells. Anti gliadin antibodies are present both in patients with ataxia and in patients with celiac disease without neurologic illness. Purkinje cell antibodies distinct from anti gliadin antibodies were found only in patients with gluten ataxia.

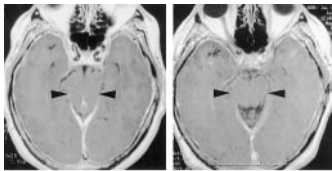
see page 1221

Epilepsy, cysticercosis and toxocariasis

In a case-control study in rural Bolivia, Nicoletti et al. compared patients with epilepsy with controls for serum antibodies against *Toxocara canis* and *Taenia solium*. They found association between late-onset partial epilepsy and *T. solium* (odds ratio 3.66) and *T. canis* (odds ratio 18.22).

see page 1256

Reversible frontotemporal dementia



Before After

Spontaneous intracranial hypotension (SIH) can cause focal neurologic deficits. Hong et al. describe a patient with SIH who developed frontotemporal dementia. The patient's cognitive and behavioral symptoms and the MRI abnormalities of SIH resolved with prednisone.

see page 1285

rTMS and executive function

Moser et al., studying patients with refractory depression, found that repetitive TMS of the dorsolateral prefrontal cortex improved some aspects of executive function, independent of changes in depression.

see page 1288

CNS inflammation in ALS: Elevations of CSF prostaglandin E₂

Almer et al. report that the level of prostaglandin E₂ (PGE₂) is markedly increased in spinal fluids of 82% of patients with ALS. Their data add to previous work showing increased PGE₂ in ALS spinal cord and provide additional support for study of inhibitors of COX-2, an enzyme that synthesizes PGE₂, in ALS. A multicenter trial of the COX-2 inhibitor celecoxib is under way.

see page 1277

Central thermoregulatory failure

Magnifico et al. document with an on-line video a man with episodic paroxysmal unilateral sweating and prolonged hypothermia. Harlequin and Shapiro syndromes were excluded. The syndrome may result from an abnormal hypothalamic thermoregulatory set point.

see page 1300

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