In Focus
Spotlight on the December 4 Issue

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Defualt-mode network connectivity in cognitively unimpaired patients with Parkinson disease

Resting-state fMRI at 3 T was performed in 16 cognitively unimpaired patients with Parkinson disease (PD) and in 16 controls. A disruption of the default-mode network was revealed in patients with PD, with absence of structural differences between patients and controls; altered default-mode network function may have a role in the development of cognitive decline in PD.

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From editorialists Filippi & Kulisevsky: “The main unanswered question is whether the observed functional abnormalities are sensitive to the progression of PD cognitive deficits and have a role in the prediction of the risk of developing dementia.”

See p. 2222

Temporal association tracts and the breakdown of episodic memory in mild cognitive impairment

Twenty-five individuals with mild cognitive impairment and 20 controls underwent diffusion MRI and cognitive assessment. Three temporal pathways were reconstructed by tractography: fornix, parahippocampal cingulum, and uncinate fasciculus. In mild cognitive impairment, due to a compromised fornix, alternative pathways may contribute disproportionately to episodic memory performance.

See p. 2233

Neurofascin as a target for autoantibodies in peripheral neuropathies

The authors detected autoantibodies to neurofascin by ELISA in 4% of patients with acute inflammatory demyelinating polyneuropathy and chronic inflammatory demyelinating polyneuropathy, but not in controls. Monoclonal antibodies to neurofascin enhanced and prolonged experimental neuritis; anti-neurofascin antibodies were pathogenic in some patients with inflammatory neuropathy.

See p. 2241; Editorial, p. 2224

Intrinsic epileptogenicity of cortical tubers revealed by intracranial EEG monitoring

Twenty-three intracranial EEG monitoring studies were reviewed from 17 children aged 1.3-7.7 years with tuberous sclerosis complex and intractable multifocal epilepsy, 14 of whom had a history of epileptic spasms. In young children with tuberous sclerosis complex and uncontrolled epilepsy, intracranial EEG may help the identification of epileptogenic tubers.

See p. 2249

Impairment of JCV-specific T-cell response by corticotherapy: Effect on PML-IRIS management?

The immune system was assessed before and 7 days after the administration of IV corticosteroids to 24 patients with relapsing multiple sclerosis. Methylprednisolone treatment decreased the frequency of JC virus (JCV)-specific CD8+ T cells producing interferon-γ and tumor necrosis factor-α, impairing control of JCV and suggesting this should be used to treat but not to prevent progressive multifocal leukoencephalopathy-immune reconstitution inflammatory syndrome.

See p. 2258

Orexin receptor antagonism for treatment of insomnia: A randomized clinical trial of suvorexant

Antagonism of wake-promoting orexins offers an alternative strategy for treating insomnia. The safety and efficacy of suvorexant, an orexin receptor antagonist, was demonstrated in a 4-week trial in 254 patients with primary insomnia, representing successful translation of a genetically identified target to a potential medication target for neuropsychiatric illness.

See p. 2265

Risk factors for intracerebral hemorrhage differ according to hemorrhage location

This study enrolled 597 patients with first-ever spontaneous intracerebral hemorrhage (ICH) and 1,548 controls. Conditional stepwise logistic regression modeling was used to determine independent risk factors for lobar and nonlobar ICH. APOE ε2 or ε4 genotype was associated with lobar ICH and hypertension was associated with nonlobar ICH.

See p. 2275

A novel hereditary extensive vascular leukoencephalopathy mapping to chromosome 20q13

Fourteen family members presented with white matter lesions at MRI examination, 5 of whom were symptomatic. The main clinical manifestations included gait disturbances, transient movement disorders, stroke, and cognitive dysfunction, establishing that this family is affected by a novel autosomal dominant vascular leukoencephalopathy mapping to chromosome 20q13.

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