Cortical-subcortical circuits in focal epilepsy

Spike-associated BOLD signal increases in the reticular formation and globus pallidus.

Federico et al. used EEG-fMRI to study interictal spikes in malformations of cortical development. They observed spike-associated BOLD signal changes in the lesion as well as at distant cortical and subcortical sites (basal ganglia, reticular formation, or thalamus). EEG-fMRI may help elucidate the mechanisms of epileptic discharges in humans.

see page 1125

EEG-fMRI in polymicrogyria (PMG)

Kobayashi et al. used spike-related EEG-fMRI to investigate epileptogenicity of PMG cortex in nine patients. Activation and deactivation were frequently found, but were often limited to parts of the lesion.

see page 1263

In the accompanying editorial, Detre and Crino review the advantages of combined EEG-fMRI in the localization of seizure foci. They note that these two reports support the idea of heterogeneity in spike localization within and outside malformations of cortical development. EEG-fMRI may be useful not only for presurgical workup, but also for investigating the pathophysiology of seizures.

see page 1108

Case-control study of SUDEP

Langan et al. conducted a case-control study to identify potential risk factors for sudden unexpected death in epilepsy (SUDEP). Generalized tonic-clonic seizures were a risk factor for SUDEP, and supervision of patients at night appeared to be protective.

see page 1131

Modafinil for fatigue in MS

Stankoff et al. report the results of a multicenter, randomized, placebo-controlled, double-blind, parallel groups trial of modafinil for fatigue in 115 MS patients. Self-reported fatigue improved equally in the modafinil and placebo groups during the 35-day treatment period, failing to support effects seen in studies with less rigorous designs.

see page 1139

Aspirin for multiple sclerosis-related fatigue

Modified fatigue impact scale scores during placebo and aspirin treatment.

In a randomized, placebo-controlled crossover trial in 30 patients, Wingerchuk et al. found that aspirin (1,300 mg/d) improved self-reported fatigue more than placebo. Among patients who completed both treatment phases, 39% preferred aspirin and 4% preferred placebo.

see page 1267

The editorial by Schwid and Murray accompanying these two papers reviews the challenges involved in studying fatigue, especially the difficulty in measuring it, and discusses the positions of modafinil and aspirin in the therapeutic armamentarium for fatigue in MS patients. Based on studies reported so far, amantadine remains the only medication with consistent effects on fatigue in MS patients.

see page 1111

Drug-resistant acute akinesia in Parkinson disease

Onofrij and Thomas describe acute akinesia in Parkinson disease unrelated to L-dopa kinetics and unresponsive to the rescue drug, apomorphine. Three percent of patients per year suffer this potentially lethal complication, which may follow drug dose changes or occur in association with concomitant diseases. The akinesia often persists after the precipitating factors have been corrected.

see page 1162

Hippocampus abnormalities in transient global amnesia (TGA)

In their 3.0T high resolution MRI study of the hippocampus in 15 TGA patients, Nakada et al. demonstrated abnormal cavitory lesions in 14 of 15. The structural abnormalities suggest that delayed neuronal loss within CA1 area may occur with TGA.

see page 1170

Cerebral correlates pain unpleasantness

Schreckenberger et al. investigated the cerebral correlates of the affective-motivational component of tonic skin and muscle pain by PET. They found the bilateral dorsal insular cortex as the main structure encoding the unpleasantness of tonic pain perception.

see page 1175

Autosomal recessive ataxia due to polymerase γ mutations

Winterthun et al. describe a distinctive, recessive, mitochondrial syndrome caused by mutations in the mitochondrial DNA polymerase, POLG: episodic features, progressive ataxia and characteristic MRI changes. Skeletal muscle may appear morphologically normal, but multiple mitochondrial DNA deletions are detectable by sensitive techniques.

see page 1204

Cerebral amyloid angiopathy is an independent risk factor in ICH

In an autopsy study of 130 brains of hypertensive patients, Ritter et al. found cerebral amyloid angiopathy to be an independent risk factor for intracranial hemorrhage beyond established risk factors such as hypertension, anticoagulants and alcoholism. The association was stronger in lobar than deep hemorrhages.

see page 1233

Microbleeds in HCHWA-D, a hereditary form of cerebral amyloid angiopathy

Van den Boom et al. found microbleeds in a hereditary variant of CAA. Cerebellar microbleeds were aggravated by hypertension, suggesting that treatment of hypertension may be important in such patients.

see page 1288

Neuroenhanced soldiers

The paper on "Cosmetic Neurology" (mind-enhancing treatments) by Anjan Chatterjee (Neurology 2004;63:968–974) and the accompanying editorials by philosopher Richard H. Dees and neurologist Steven L. Hauser drew major media interest. Here, correspondence from US military officers notes the potential for use of drugs in combatants. Chatterjee, Dees, and Hauser provide thoughtful responses.

see page 1320

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