A magnet for the brain: 20 years of experience

Transcranial magnetic stimulation (TMS) activates the underlying brain relays and networks without risk or pain. Rossini and Rossi review progress in TMS since 1985. TMS represents a formidable tool for exploring function (movement, language, memory, perception, plasticity) in the healthy and in damaged brain. TMS has potential for treatment of a number of disabling neurologic conditions including chronic pain, dystonias, epilepsy, migraine, and stroke.

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Benign MS at 20 years

EDSS scores at 20 years in patients with benign MS (EDSS ≤ 3 at 10 years after onset).

Sayao et al. evaluated the disease status in 169 patients with benign MS (EDSS ≤ 3 , at 10 years from onset) 20 years after onset, by which time almost half were no longer benign. No 10-year EDSS score appeared ideal in predicting benign status.

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The editorial by Sean J. Pittock suggests that if we could identify which patients will remain benign, disease-modifying treatments would not be indicated for them. Since most evidence supports a beneficial effect of disease-modifying agents (DMA) on relapse rate and MRI activity, patients with infrequent attacks and stable MRIs (whose clinical and radiologic profiles strongly differ from those generally enrolled in drug trials) would benefit less from DMAs than those with frequent attacks or evidence of substantial subclinical (MRI-based) disease activity. A watchful waiting approach with regular clinical and MRI monitoring before initiating treatment may be a preferable approach for some patients. However, the watchful waiting should not be misconstrued as a never treat approach. If the decision is made to delay treatment in a patient with a favorable disease profile, yearly clinical and radiologic surveillance is advised, with appropriate initiation of DMA should the clinical (relapses) or radiologic (new/enhancing lesions) status or patient wishes change.

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Beta-blocker treatment may be cerebroprotective during stroke

Laowattana and Oppenheimer found that activated sympathetic tone correlates with poorer prognosis after stroke. Prior beta-blocker therapy was associated with improved clinical presentation and lower coagulation and inflammatory indices as well as reduced sympathovagal balance after stroke. Beta-blocker treatment may be cerebroprotective in high-risk cerebrovascular disease patients.

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Cannabis in painful HIV-associated neuropathy

Abrams et al. studied the short-term effects of smoked cannabis in 50 patients with HIV-associated sensory neuropathy. In a randomized, placebo-controlled trial, cannabis decreased neuropathic pain and pain in an experimental model with an effect comparable to clinically proven oral drugs for chronic neuropathic pain.

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Psychiatric features in SGCE myoclonus-dystonia

Hess et al. evaluated psychiatric features in five families with SGCE mutation myoclonus-dystonia. They found that obsessive-compulsive disorder and alcohol dependence were increased in gene carriers with myoclonus-dystonia. They postulate that obsessive-compulsive disorder is associated with mutant SGCE, and alcohol dependence is reactive to palliative effects.

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Characteristics of HIV dementia in Thailand

Recent in vitro research suggests that HIV-1 subtype may impact neuropathogenesis. The study by Valcour et al. compared neuropsychological profiles among HIV-infected Thais (subtype CRF AE_01) with dementia, matched individuals without dementia, and seronegative controls, describing abnormalities similar to that seen in subtype B.

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Right-to-left shunt and lacunar stroke

Ueno et al. identified potential embolic sources in patients with lacunar stroke using transesophageal echocardiography. Patients with lacunar stroke having neither hypertension nor diabetes mellitus had a higher frequency of right-to-left shunt (82%) than patients with these risk factors or controls.

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Amyloid, hypometabolism, and cognition in AD

Edison et al. found that 90% of patients with AD show diffusely raised cortical amyloid with [11C]PIB-PET and reduced temporal glucose metabolism with FDG PET, which correlates with cognitive performance. High frontal amyloid and normal frontal glucose utilization in AD suggests amyloid plaque formation may not be directly responsible for the neuronal dysfunction.

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The editorial by Bateman and Eidelberg notes that as PIB PET is a molecular amyloid imaging technique, it has the potential of providing quantitative measures of amyloid in the brain, which previously could only be obtained post-mortem. How PIB PET is applied will depend on the questions that are asked. The Edison et al. study gives further support for its applicability in identifying AD. It has an obvious role in research, and is potentially useful as a tool in clinical trials. Whether PET imaging with PIB or related radiotracers will be adopted as a diagnostic test for AD will require large controlled studies of AD compared to other dementias with follow-up pathologic confirmation.

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Localization of increased [11C]PIB uptake in AD vs normal revealed by SPM, $p < 0.00001$. Primary motor (PM) and visual cortical areas, thalamus (TH), brainstem (BS), and cerebellum (CB) show no significant increase in PIB uptake vs control subjects at that threshold.

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