Diffusion-weighted MRI in stroke

Two articles and two editorials consider the developing role of diffusion-weighted imaging (DWI) in assessment of stroke. Lansberg et al. (p. 1557) compared CT and DWI at onset (within 7 hours) of 19 patients with acute stroke. They assessed DWI at 36 hours and T2-weighted MRI at 30 days (for final infarct volume). DWI was deemed more accurate and more sensitive for detection of large infarcts. Acute DWI estimation of lesion volume correlated with 30-day infarct volume. ◆ Albers et al. (p. 1562) performed DWI and conventional T2 and proton density MRI 6 to 48 hours after stroke onset in 40 consecutive patients. DWI detected major findings in 50% of cases that were not evident on conventional MRI, including: different vascular territory than suspected clinically; multiple lesions in different vascular territories; and evidence that lesions were old rather than acute. ◆ The accompanying editorials consider the implications of these and reported studies for the role of DWI in acute stroke management. Hacke and Warach (p. 1548) argue that because DWI clearly provides data additional to other imaging techniques, DWI is now of importance for the assessment of stroke. Conversely, Powers (p. 1549) argues that DWI has not yet been assessed by randomized controlled trials and falls short of the standards for trials of DWI (or any new test). Powers also points out that there are five levels of clinical efficiency that need to be considered when assessing DWI—starting with the technical accuracy of the test, but demanding that diagnostic and therapeutic impact, and most importantly, patient outcome, must be shown to be benefited by DWI. ◆ Finally, the commentary by Zivin and Holloway (p. 1552) succinctly presents the argument for publishing two articles that fall short of the most rigorous, accepted standards for new diagnostic tests. The Lansberg et al. and Albers et al. articles present important new data on DWI. Powers' arguments are an agenda for stroke research. Furthermore, the Editors believe that without the studies Powers demands, techniques such as DWI may not receive the long-term, universal acceptance (and financial commitments) that Hacke and Warach defend. The *neurologist* is not the actual buyer.

Stroke progression

Dávalos et al. (p. 1568) examined ferritin, total iron, and glutamate in plasma and CSF of 100 consecutive patients with acute cerebral infarction. Patients were followed for progression. Both plasma and CSF ferritin levels were higher in patients who progressively deteriorated. The observations support the hypothesis that iron-dependent free radical formation may promote brain ischemic injury.

Spinal cord injury

Hiersemenzel et al. (p. 1574) investigated the excitability of spinal neuronal circuits below the level of the cord lesion as patients with spinal cord injury (SCI) evolved from spinal shock to spasticity. They studied 16 SCI patients, examining M-waves, F-waves, H-reflexes, and flexes reflex, correlating these measures with clinical spasticity. Remarkably, severe spasticity developed without electrophysiologic changes that could account for the spasticity.

Neuroprotection for PD?

The Parkinson Study Group (p. 1583) report a brief, controlled trial of the low-affinity NMDA-channel blocker remacemide in 200 patients with early PD (before the use of levodopa). Remacemide was not of symptomatic benefit but was generally well-tolerated. The study paves the way for a large study of remacemide as a neuroprotective agent in PD.

Dementia in PD and dementia with Lewy bodies

Hughes et al. (p. 1596) followed nondemented subjects for up to 10 years: 83 with PD and 50 controls. In the interval, 38% of PD patients became demented. The severity of PD correlated with dementia but there was no evidence that early-onset PD differed from late-onset PD in terms of dementia incidece. ◆ Fluctuating cognition was quantitated by Walker et al. (p. 1616) in a study comparing dementia with Lewy bodies (DLB), AD, and vascular dementia (VaD). Fluctuating cognition was documented in DLB and distinguished DLB from AD and VaD. This important documentation of clinical impression about DLB focuses attention on discovering why patients with DLB have such fluctuations.

Susceptibility and severity of Guillain-Barré syndrome

Factors triggering acute Guillain-Barré syndrome (GBS) have been determined for a large proportion of patients: i.e., cytomegalovirus, Epstein-Barr virus, and *Campylobacter jejuni.* The risk factors for developing GBS following one of these triggering infections are less clear. Van der Pol et al. (p. 1661) studied leukocyte receptors for immunoglobulin in 31 patients with GBS. A specific subclass genotype was much more frequent in patients with GBS and predicted a more severe disease course.

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