Research

**Gray Matter Loss and Clinical Deficits in Multiple Sclerosis** 944

Mackenzie-Graham and colleagues study the association between localized gray matter (GM) atrophy and clinical disability in a biology-driven analysis of multiple sclerosis (MS). Magnetic resonance images were acquired from 133 women with relapsing-remitting MS and analyzed using voxel-based morphometry and volumetry. These biology-driven data indicate that specific disabilities in MS are associated with voxel-wise GM loss in distinct locations. Editorial perspective is provided by Michael K. Racke, MD, and Jaime Imitola, MD.

- Editorial 910

**Association Between Hypodensities and Hematoma Expansion** 961

Boulouis et al determine whether hypodense regions, irrespective of their specific patterns, are associated with hematoma expansion in patients with intracerebral hemorrhage (ICH). They analyzed a large cohort of 784 patients with ICH, examined noncontrast computed tomography (NCCT) findings for any hypodensity, and replicated their findings on a different cohort of patients. They found that hypodensities within an acute ICH detected on an NCCT scan may predict hematoma expansion, independent of other clinical and imaging predictors. Editorial perspective is provided by Louis R. Caplan, MD.

- Editorial 914

Clinical Review & Education

**Flow Diversion for the Treatment of Intracranial Aneurysms** 1002

Walcott and colleagues summarize the clinical progression of flow diversion technology, from an experimental treatment to a commonly used method to treat large or complex aneurysms. Flow diversion is a treatment approved by the US Food and Drug Administration for brain aneurysms that redirects blood flow away from the aneurysm, thereby promoting growth of a new endothelial lining across the aneurysm opening. It is a disruptive technology that has changed the way many brain aneurysms are treated. Editorial perspective is provided by Babu G. Welch, MD, and H. Hunt Batjer, MD.

- Editorial 921

**GABRG2 Mutations Associated With Epilepsy Syndromes** 1009

Kang and Macdonald focus on the molecular pathogenic basis for genetic generalized epilepsies associated with mutations in the inhibitory γ-aminobutyric acid (GABA<sub>γ</sub>) receptor γ2 subunit gene, GABRG2. Mutations in GABRG2 have been associated with simple febrile seizures and with genetic epilepsy syndromes, including childhood absence epilepsy, generalized epilepsy with febrile seizures plus, and Dravet syndrome or severe myoclonic epilepsy in infancy. They conclude that the epilepsy phenotypic heterogeneity associated with GABRG2 mutations may be related to the extent of the reduction of GABA<sub>γ</sub> receptor channel function and the differential dominant negative suppression.