# *This Week in The Journal*

## ● Cellular/Molecular

### *Fast and Synchronous, Slow and Asynchronous*

Takeshi Sakaba

(see pages 5863–5871)

Synaptic vesicles have been divided into distinct pools based on their release kinetics. Fast-releasing and slowly releasing pools were initially detected using stepwise increases in intracellular calcium, but this week Sakaba examined vesicular release in response to more physiological stimuli: action potential-like depolarizations of rat calyx of Held neurons. Using a deconvolution method, the author estimated the quantal release rate and separated synchronous and asynchronous release populations. The analysis suggested that 80% of the fast-releasing and 60% of slowly releasing vesicles were released during a 100 Hz train. Presynaptic capacitance measurements supported these estimates. By blocking the calcium/calmodulin-dependent recovery of the fast-releasing pool, the author was able to examine the slow-releasing pool of vesicles in isolation. Synchronous release primarily consisted of fast-releasing vesicles, whereas asynchronous release was carried mainly by slowly releasing vesicles. It took a 300 Hz train to deplete both vesicle populations.

## ▲ Development/Plasticity/Repair

### *Stroke Recovery and the Contralateral Hemisphere*

Martin Lotze, Jochen Markert, Paul Sauseng, Julia Hoppe, Christian Plewnia, and Christian Gerloff

(see pages 6096 – 6102)

Recovery after a brain injury such as a hemispheric stroke can involve not only both local recovery and repair but perhaps compensation from distant regions, including the contralateral hemisphere. This week, Lotze et al. examined a group of patients with a history of an ischemic stroke involving the internal capsule. These patients initially had significant movement impairment in the affected hand but then recovered almost fully. The authors used functional magnetic resonance imaging to compare activation of motor areas in the damaged and contralateral hemispheres during a finger-tapping task. Cortical activation ipsilateral to the moving hand (contralateral to the damaged hemisphere) was greater in the stroke patients than in control subjects. To "jam" functional activity, the authors used transcranial magnetic stimulation. Stimulation in the contralateral dorsal premotor cortex, primary motor cortex, and superior parietal lobe (SPL) produced timing errors, and SPL stimulation compromised accuracy as well.

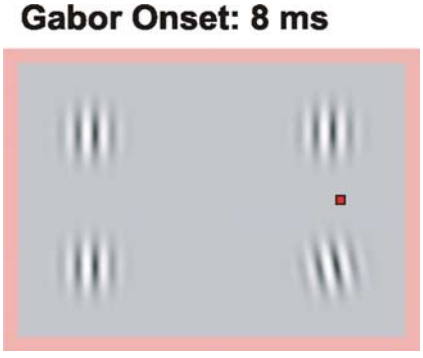
## ■ Behavioral/Systems/Cognitive

### *A BOLD View of Saccadic Suppression*

Ignacio Vallines and Mark W. Greenlee

(see pages 5965–5969)

Fortunately, the visual system doesn't deliver a high-resolution image during saccadic eye movements; otherwise, life would consist of an endless train of blurry images each time we moved or the world around us moved. This visual suppression process must involve cortical areas because it precedes movement. This week, Vallines and Greenlee propose primary visual cortex (V1) as a site of saccadic suppression. They measured BOLD (blood oxygen level-dependent) responses to Gabor patches: blurred images of contrasting lines. The authors first mapped the location encoding the stimulus in V1. The four subjects were then presented with the stimuli for 8 ms before the eye made a saccade to a visual target. Although the stimulus was always presented to a stationary retina, discrimination was degraded and V1 responses were smaller the closer the stimulus was presented to a saccade, consistent with involvement of V1 in saccadic suppression.




Immediately before the onset of a saccade, four Gabor stimuli were simultaneously flashed for 8 ms. The subjects then moved their gaze from a central fixation point to the target (red dot). See the article by Vallines and Greenlee for details.

## ♦ Neurobiology of Disease

### *Tracking Pseudohyperphosphorylated Tau* In Vitro

Neelam Shahani, Srinivasa Subramaniam, Tobias Wolf, Christian Tackenberg, and Roland Brandt

(see pages 6103– 6114)

Accumulation of intracellular hyperphosphorylated tau-containing fibrils is a hallmark of Alzheimer's disease as well as several other tauopathies. This week, Shahani et al. examined the short-term effects of an EGFP (enhanced green fluorescent protein)-labeled pseudohyperphosphorylated (PHP) tau on organotypic cultures. The PHP tau had glutamate residues instead of the normally phosphorylated serine and threonine residues, glutamate acting as an electrostatic mimic for phosphorylated residues. Using Sindbis virus, the authors expressed wild-type and PHP tau in mouse hippocampal slices. Three days later, extracellular lactate dehydrogenase, an indicator of membrane damage, was greatly increased in the PHP slices, as was caspase-3 activity and DNA fragmentation, suggestive of apoptotic cell death. However, some cells showed swollen cell bodies, not consistent with purely apoptotic cell death. Tau-mediated death was concentrated in CA3 and the dentate gyrus. Spine density and morphology were remarkably stable in the presence of PHP tau.