WHITAKER BIOMEDICAL ENGINEERING INSTITUTE

DEPARTMENT OF BIOMEDICAL ENGINEERING
FRIDAY SEMINAR SERIES

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"Cortical Control of Midbrain Oculomotor Reflexes"

DATE: December 16, 2005

TIME: 1:00 p.m. – 2:00 p.m.

PLACE: **Traylor 709**

Host: Dr. Reza Shadmehr

Abstract:
A Critical issue in both psychology and biology is to understand how reflexive and voluntary behaviour is co-ordinated to enable adaptive behaviour and to provide for a continuity of coherent experience. To survive and function effectively in the world, we need to be able to respond reflexively to potential dangers (turning toward a sudden movement seen out of the corner of the eye), and also to be able to search the world strategically based on internal goals (looking both ways before crossing the street, or scanning a crowd for the face of a friend.). Reflexive eye movements are mediated by phylogenetically primitive circuits form the eye to the brain stem. Voluntary orienting, on the other hand, is controlled by more recently evolved regions of the oculomotor cortex in the frontal and parietal lobes. Eye movements are controlled through opposing neural circuits: that is, there are mutually inhibitory connections between those that anchor the eyes at the current point of fixation, and those that move them to a new location. For reflexive saccades, fixation cells are located in the rostral pole of the superior colliculus, while 'movement cells' coding saccades of progressively increasing amplitude are located at progressively more caudal loci in the colliculus. This opponent processing can be measured by a simple effect called the fixation offset effect (FOE): for both reflexive and voluntary eye movements, the time to initiate an eye movement is longer if the eyes are gazing at a fixation point when the command to move the eyes is given than if the fixation point offsets.

The difference in saccade latency between fixation offset and fixation overlap conditions (the FOE) is a measure of the degree to which rostral pole neurons are under external control by the fixation point. Manipulations of strategic set that bring these neurons under voluntary control, and thereby reduce the influence of the external stimulus at fixation, will decrease the size of the FOE. This modulation of the FOE thereby provides a measure of cortical control over collicular circuitry.

I'll report evidence from both healthy adults and neurological patients with cortical and thalamic lesions, that: 1) there are independent neural substrates controlling fixation for reflexive and voluntary saccades; 2) that these fixation mechanisms are under strategic control. By manipulating the readiness to make either a voluntary or reflexive eye movement, or conversely a readiness to inhibit an eye movement, the size of the FOE can be increased or decreased and; 3) that this strategic modulation of the FOE is dependent upon the frontal eye field.

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