Control of volitional and reflexive saccades in Tourette's syndrome

Adrienne L. LeVasseur1, J. Randall Flanagan1,2, Richard J. Riopelle3 and Douglas P. Munoz1,2

1 Departments of Physiology 2 Psychology and 3 Medicine, Queen's University, Kingston, Ontario, Canada

Tourette's syndrome is characterized by involuntary tics and, although the underlying pathogenesis and pathophysiology of Tourette's syndrome remains unclear, it is suspected that basal ganglia structures are involved. The basal ganglia also play an important role in the control of saccadic eye movements and we therefore hypothesize that Tourette's syndrome patients have abnormal control of saccadic eye movements. In this study, 10 subjects with Tourette's syndrome and 10 age- and sex-matched controls performed four different oculomotor paradigms requiring the execution and/or suppression of reflexive and/or voluntary saccades. In the immediate saccade tasks, subjects were required to look either toward (pro-saccade task) or away from (anti-saccade task) a peripheral target as soon as it appeared. In the delayed saccade tasks, subjects were instructed to wait for a central fixation point to disappear before initiating eye movements. Among Tourette's syndrome subjects, saccadic reaction times were longer in all tasks. Saccadic amplitudes were smaller in Tourette's syndrome subjects, and they made more saccades to reach the eccentric target. The occurrence of direction errors (i.e. reflexive pro-saccades on anti-saccade trials) was normal in the immediate anti-saccade task, suggesting that the ability to inhibit reflexive saccades towards novel stimuli was not impaired in Tourette's syndrome. Timing errors (i.e. eye movements made prior to disappearance of the central fixation point in delayed saccade tasks) were significantly greater among Tourette's syndrome subjects. Moreover, these errors were predominantly made towards the first target of the remembered sequence in a delayed memory-guided sequential saccade task. These results indicate that the ability to inhibit or delay planned motor programmes is significantly impaired in Tourette's syndrome. We hypothesize that altered cortical–basal ganglia circuitry leads to reduced cortical inhibition making it harder for Tourette's syndrome subjects to withhold the execution of planned motor programmes.