Lieshout 1991; Huerta et al. 1991; Jayaraman et al. 1977; Jiang et al. 2003; Mana and Chevalier 2001). Furthermore, dorsally located neurons in the SNr tend to project more rostrally in the SC compared to ventrally located SNr neurons which tend to project more caudally in SC (Gerfen et al. 1982). These anatomical results suggest that further inquiry into the topographical organization of the SNr-SC pathway in the behaving monkey would be a worthwhile effort.

A recent study of eye movements in PD patients indicates that the BG dysfunction associated with this disease impairs voluntary movement initiation but also impairs the ability to suppress reflexively driven saccades. This lesser appreciated symptom was determined by measuring an increase in saccadic intrusions (errors) during the delay period of a delayed anti-saccade task (Amador et al. 2006; Briand et al. 1999). Although controversial, according to classical models of BG dysfunction in PD the net effect of reduced dopamine tone is to *increase* the inhibitory output of the BG (Albin et al. 1995). Thus, the decreased latency reported here may be interpreted as evidence that the stimulation acts to inactivate the SNr, but also may indicate that electrical stimulation mimics the PD state and increases the inhibitory output. We propose that the stimulation increases the inhibitory output of the BG and produces an inability to suppress a reflexive saccade. Indeed, that the BG are involved in reflex suppression is well-documented (Basso and Evinger 1996; Basso et al. 1996; Basso et al. 1993; Mink and Thatch 1991a, 1991b). Thus, consistent with the recent work in humans (Amador et al. 2006; Briand et al. 2001), our