saccadic eye movements was reduced by ~6% on trials with electrical stimulation of the SNr across all three monkeys and all 61 stimulation sites. Importantly this reduction was seen only for memory-guided trials (Figure 9a contralateral memory, * $p = 0.03$; visual, $p = 0.81$).

The current model of the role of SNr in the monkey saccadic system emphasizes the control of contralateral saccades. However, with electrical stimulation, we also found that the occurrence of ipsilaterally directed saccades was reduced. For ipsilateral saccades, the reduction in occurrence was ~12% and statistically reliable (Figure 9b ipsilateral memory, $p<0.001$). Like contralateral saccades, the reduction in ipsilateral saccades was evident only in the memory-guided trials (Figure 9b, ipsilateral visual, $p= 0.67$). Indeed, the difference in the reduction of contralateral and ipsilateral saccades was also significantly different (88% versus 12%, $p = 0.02$). This result suggests that there may be differences between the influence of the SNr on the two SCs (Jiang et al. 2003).

In summary, we found that electrical stimulation of the SNr influenced the generation of saccades. Electrical stimulation altered the vector of the saccade produced during SNr stimulation. Frequently saccades were shorter in length and the direction of the saccade vector was rotated in stimulation trials compared to control trials. The alteration in saccade vector was also more prominent for saccades made to targets guided by memory than those with visual stimuli present to guide them. We found that SNr stimulation influenced saccade latency. In general, visually-guided saccades, that tended to be < 200ms without