experiments. Second, given the stimulation parameters we used (60μA, 300Hz) we would expect that driving excitatory input to the SC directly would evoke saccades with latencies shorter than what we generally observed (Bruce et al. 1985a; Robinson 1972; Robinson and Fuchs 1969). Therefore, as we suggested above the electrical stimulation may initially disinhibit the SC and then subsequently inhibit the SC. One way this may come about is if inhibitory caudate fibers projecting to the SNr (Hikosaka et al. 1993) are activated initially resulting in a suppression of SNr activity. Because our trains were long (400ms), GABA may be depleted allowing SNr neurons to recover from inhibition and then inhibit SC neurons.

A second possibility is that the SNr neurons project to GABAergic interneurons that are known to exist in the SC (Behan and Kime 1996; Behan et al. 2002). A projection to interneurons has not been demonstrated directly (Bickford and Hall 1992). Although, anatomical experiments in rat, cat and monkey suggest that the pathway from the SNr to the SC has more nuances than appreciated by the model developed from the monkey experiments (Beckstead 1983; Beckstead et al. 1981; Deniau and Thierry 1997; Harting et al. 1988). There are at least three pathways from the SNr to the SC. The first is uncrossed, arises from the lateral SNr, and projects to the superficial layers of the SC and the dorsal intermediate layers of the SC. The second is uncrossed, arises from the medial SNr, and projects to the lower intermediate layers of the SC and the deep layers of the SC. The third is crossed and terminates in the contralateral SC (Gerfen et al. 1982; Harting et al. 1988; Harting and Van