1993; Hikosaka et al. 1989a, 1989b, 1989c) and contain GABA (Gerfen 1985; Gerfen and Wilson 1996). Therefore, the discharge of caudate neurons results in a decrease in the discharge of SNr neurons. SNr neurons are tonically active, contain GABA (Chevalier and Deniau 1990; Chevalier et al. 1981a; Chevalier et al. 1984; Chevalier et al. 1985; Hikosaka and Wurtz 1983a, 1983d) and have direct projections to the SC. Thus, decreasing SNr neuronal activity reduces inhibition of SC neurons resulting in a robust discharge of action potentials in SC neurons signaling a command to initiate a saccade (Hikosaka et al. 2000; Hikosaka and Wurtz 1983d). A schematic of the activity profiles of the caudate, SNr and SC during a saccade is shown in Figure 1a.

The results of the original recording experiments in the monkey SNr suggested that SNr neurons were involved preferentially in saccades guided by particular behavioral contexts such as memory (Hikosaka and Wurtz 1983c). Consistent with this hypothesis, injection of muscimol, a GABA agonist, into the SNr produces deficits in memory-guided eye movements (Hikosaka and Wurtz 1985). More recent electrophysiological recordings suggest that SNr neuronal activity is not specific for memory-guided saccades (Bayer et al. 2002; Handel and Glimcher 2000) and may be involved in target selection for saccades (Basso and Wurtz 2002). Therefore, there remain a number of questions regarding the precise nature of the role of SNr in saccadic eye movements.

In this report we describe the results of experiments in which we explore the role of the SNr in saccadic eye movement control using electrical stimulation. Our goal was two-fold. First, in light of the previous muscimol results (Hikosaka and