Project 22: Movement control and tonic dopamine

1) Implement the striatal policy gradient model described in Chapter 11 (Section 2.1) of *Computational Foundations of Cognitive Neuroscience*. Simulate the effects of Huntington's and Parkinson's disease on movement speed by selectively degrading the direct or indirect pathways. For the purposes of this simulation, you can operationalize movement speed as the number of time steps between the onset of a go cue and the onset of movement initiation. Compare this with experimental data from Kravitz et al. (2010).

2) What does the model imply about the malleability of bradykinesia in Parkinson's? Show the time course of movement speed with training under different levels of tonic dopamine, and compare with data reported in Platz et al. (1998). Can this model explain why the benefits of physical therapy for Parkinson's depends on the availability of sensory cues (Marchese et al., 2000)?

References:

Kravitz, A. V., Freeze, B. S., Parker, P. R., Kay, K., Thwin, M. T., Deisseroth, K., & Kreitzer, A. C. (2010). Regulation of parkinsonian motor behaviours by optogenetic control of basal ganglia circuitry. *Nature, 466*, 622-626.

Marchese, R., Diverio, M., Zucchi, F., Lentino, C., & Abbruzzese, G. (2000). The role of sensory cues in the rehabilitation of parkinsonian patients: a comparison of two physical therapy protocols. *Movement Disorders, 15*, 879-883.

Platz, T., Brown, R. G., & Marsden, C. D. (1998). Training improves the speed of aimed movements in Parkinson's disease. *Brain, 121*, 505-514.