Studies in rodent models of Parkinson’s Disease have highlighted the challenge of determining whether increases in synchronized and oscillatory activity in the basal ganglia play a causal, or more compensatory or epiphenomenal, role in mediating motor impairment in this disorder. This talk will describe efforts to address this question through recordings of spiking and local field activity from multiple sites in the basal ganglia-thalamocortical circuit, with attention to how loss of dopamine promotes coherent oscillatory activity throughout this circuit in conjunction with changes in behavioral state. Recently, as specific types of beta bursts have been shown to be effective as triggers for closed-loop deep brain stimulation in Parkinsonian patients, studies have also focused on the more dynamic aspects of beta range activity, and consideration of how incidence, duration and amplitude of individual beta bursts may contribute to motor deficits.