The basal ganglia and temporal processing: evidence from Parkinson's disease

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Abstract

Parkinson's disease (PD) is characterized by basal ganglia dysfunction due to dopamine loss, which makes it an ideal 'model' for investigating the role of the basal ganglia and dopamine in temporal processing. Furthermore, bradykinesia, or slowness of movement, is one of the primary symptoms of PD and leads to the prediction of timing deficits in this disorder. Empirical evidence has established that patients with Parkinson's disease have deficits in both motor timing and perceptual timing tasks, which increase with severity of the motor symptoms. Temporal processing in both the millisecond and seconds ranges is impaired in this disorder. These deficits in temporal processing are generally more severe off levodopa medication and are reduced when patients are tested on medication. Imaging of PD patients during performance of a synchronization-continuation motor timing task has shown that while the fronto-striatal circuits are engaged during performance of this task by healthy controls, PD patients rely on activation of the cerebellum for task performance. Dopamine replacement medication increases striatal and frontal activation and striatal-frontal connectivity in PD during motor timing compared to when tested off medication. While evidence from PD has confirmed the importance of the basal ganglia and dopamine in temporal processing, the specific and differential roles that the striatum and the frontal areas play in temporal processing remain to be clarified.

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