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Stuttering and the basal ganglia circuits: a critical review of possible relations

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Abstract

The possible relation between stuttering and the basal ganglia is discussed. Important clues to the pathophysiology of stuttering are given by conditions known to alleviate dysfluency, like the rhythm effect, chorus speech, and singing. Information regarding pharmacologic trials, lesion studies, brain imaging, genetics, and developmental changes of the nervous system is reviewed. The symptoms of stuttering are compared with basal ganglia motor disorders like Parkinson's disease and dystonia. It is proposed that the basal ganglia-thalamocortical motor circuits through the putamen are likely to play a key role in stuttering. The core dysfunction in stuttering is suggested to be impaired ability of the basal ganglia to produce timing cues for the initiation of the next motor segment in speech. Similarities between stuttering and dystonia are indicated, and possible relations to the dopamine system are discussed, as well as the interaction between the cerebral cortex and the basal ganglia. Behavioral and pharmacologic information suggests the existence of subtypes of stuttering.
Learning outcomes: As a result of this activity, the reader will (1) become familiar with the research regarding the basal ganglia system relating to speech motor control; (2) become familiar with the research on stuttering with indications of basal ganglia involvement; and (3) be able to discuss basal ganglia mechanisms with relevance for theory of stuttering.

Keywords: Stuttering; Basal ganglia; Dopamine; Dystonia; Cluttering

1. Introduction

Research concerning the nature of stuttering has produced an extensive amount of data during the past century, but the mechanisms behind the speech disruptions and the speech

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initiation problems are still not clear. An intriguing aspect of stuttering is the various conditions which can temporarily alleviate dysfluency in most cases: the rhythm effect (speaking to the pace of a metronome), singing, chorus speech, and altered auditory feedback (Wingate, 2002). The often dramatic improvements in fluency caused by these conditions indicate that stuttering is not the result of some general speech motor instability, instead there seem to be specific causal mechanisms leading to the speech problems.

In this article, possible relationships between stuttering and the functions of the basal ganglia (BG) circuits are reviewed and discussed. This review leads to the proposal that the circuits through the basal ganglia play a key role in the mechanisms of stuttering.

The BG are the largest subcortical structures in the human forebrain, and they are placed in a key position to influence motor behavior, emotions, and cognition (Graybiel, 2000). The idea that stuttering may be related to the BG is not new. As early as 1934, Seeman suggested that stuttering is the result of disturbed BG function (as cited in Van Riper, 1982). More recent suggestions for BG involvement in stuttering come from Rosenberger (1980), Caruso (1991), Wu et al. (1995), Lebrun (1998), and Victor and Ropper (2001), and others.

First an overview of the basal ganglia anatomy and functions will be presented. Thereafter several aspects of basal ganglia functions and disorders will be discussed in relation to stuttering: motor control and timing, lesions, brain imaging, dopamine, emotional influences, developmental changes of the BG, and similarities between stuttering and disorders like Parkinson's disease and dystonia. The BG operate in a close relation with the cerebral cortex, and therefore some important findings about the cortex and stuttering will also be discussed, from the perspective of the basal ganglia functions. Lastly tentative conclusions will be presented. Among the suggested conclusions can be mentioned that the core dysfunction in stuttering is proposed to be impaired ability of the basal ganglia to produce timing cues, that developmental changes of dopamine receptor density in the putamen might explain the frequent pattern of early childhood onset and recovery of stuttering, and that stuttering is likely to be a heterogeneous disorder with subtypes showing different responses to different types of dopaminergic medication.

2. Overview of the basal ganglia anatomy and functions

Even though the understanding of the BG circuits still must be considered as highly incomplete, knowledge has grown rapidly during the last decades. The model presented here is simplified, mainly limited to the aspects most relevant to the discussion. (For more thorough reviews, see for example Mink, 1996, and Victor & Ropper, 2001.)

The basal ganglia consist of a set of interconnected subcortical nuclei. The main input nucleus is the *striatum*, which receives topographical excitatory projections from almost the entire cerebral cortex, especially from the sensorimotor and frontal cortex (Parent, 1996). The striatum and the downstream structures in the basal ganglia are organized in topographically and functionally segregated pathways. The cortical inputs to the striatum are convergent, for example in such a way that sensory and motor cortex areas converge into single striatal zones (Flaherty & Graybiel, 1991).