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Review

Motor automaticity in Parkinson's disease

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ABSTRACT

Bradykinesia is the most important feature contributing to motor difficulties in Parkinson's disease (PD). However, the pathophysiology underlying bradykinesia is not fully understood. One important aspect is that PD patients have difficulty in performing learned motor skills automatically, but this problem has been generally overlooked. Here we review motor automaticity associated motor deficits in PD, such as reduced arm swing, decreased stride length, freezing of gait, micrographia and reduced facial expression. Recent neuroimaging studies have revealed some neural mechanisms underlying impaired motor automaticity in PD, including less efficient neural coding of movement, failure to shift automated motor skills to the sensorimotor striatum, instability of the automatic mode within the striatum, and use of attentional control and/or compensatory efforts to execute movements usually performed automatically in healthy people. PD patients lose previously acquired automatic skills due to their impaired sensorimotor striatum, and have difficulty in acquiring new automatic skills or restoring lost motor skills. More investigations on the pathophysiology of motor automaticity, the effect of L-dopa or surgical treatments on automaticity, and the potential role of using measures of automaticity in early diagnosis of PD would be valuable.

Contents

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Introduction

Parkinson's disease (PD) is one of the most common progressive neurological degenerative disorders. The primary manifestations of PD are motor dysfunction, including slowness of movements (bradykinesia), resting tremor, rigidity, gait disturbance and postural instability. In this review, bradykinesia is used to encompass the terms hypokinesia (smallness of movement, as in micrographia) and akinesia (lack of movement, as in freezing or lack of facial expression).

Bradykinesia is the most important reason for motor difficulties in PD, which can affect almost all activities in daily life (Hallett, 2011). Several pathophysiology mechanisms have been proposed to explain bradykinesia, like a failure of basal ganglia output to reinforce the cortical mechanisms that prepare and execute the command to move (Berardelli et al., 2001), loss of motor energy, difficulty with internal driving of movement and the concomitant excess influence of external sensory control of movement, and sequence effect (Hallett, 2011). However, these mechanisms can only provide partial explanation, and, as such, our understanding of the pathophysiology underlying bradykinesia remains incomplete.

Since the 1980s, it was already recognized that PD patients have difficulty in "automatic execution of learned motor plans", and this problem is likely due to impairment of basal ganglia function (Marsden, 1982). It has been shown that PD patients have a greater abnormality of automatic associated movement than intended voluntary movement, which may be one of the bases of clinical symptoms in the early stage of the disease (Hoshiyama et al., 1994). However, even though impaired motor automaticity is likely an important reason underlying bradykinesia in PD, it has been generally overlooked and much less investigated compared to other motor deficits. In this review we discuss the contributions of defective motor automaticity to bradykinesia in PD, and its neural mechanisms.

What is motor automaticity

Automaticity is the ability to perform movements without attention directed toward the details of the movement, particularly for movements that require low levels of precision or for movements that are commonly made (Bernstein, 1967). After a period of training, and passing through several distinct phases, including fast (early), slow (later), and consolidation stages (Doyon and Benali, 2005), even some complex motor skills can be well established and achieve the automatic phase (Fitts, 1964). At this stage, motor skills can be performed requiring minimal cognitive resources and are resistant to interference and the effects of time. In fact, most of our daily behaviors are carried out automatically. From getting up in the morning, brushing teeth, eating breakfast with spoon, fork or chopstick, to walking or driving to work, people usually pay little attention to these motor behaviors. For example, people can talk on cell phone while walking; even when focusing on talking, they still can maintain walking smoothly without difficulty.

Because improvement on a task after extensive practice does not guarantee that it is automatic (Lang and Bastian, 2002), motor automaticity is commonly evaluated by a dual-task paradigm (Wu et al., 2004), which requires to perform a secondary task at the same time as a first motor task. The secondary task is usually a cognitive task, e.g., a counting task, but also can be another motor task. If both tasks are performed correctly without obvious interference, the first motor task can be said to be automatic (Passingham, 1996; Wu et al., 2004). If the performance of one or both tasks deteriorates under dual-task conditions, then the motor task is not automatic.

Neural mechanisms of automaticity in healthy people

Several studies have investigated neural correlates during motor automatic processing with dual-task to prove automaticity (Wu et al., 2004, 2005b, 2008; Lehe´ricy et al., 2005; Poldrack et al., 2005; Balsters and Ramnani, 2011; Wu et al., in press) (Table 1), a common finding is decreased activity in extensive brain regions, e.g., the dorsolateral prefrontal cortex (DLPFC), anterior cingulate cortex (ACC), rostral supplementary motor area (pre-SMA), premotor cortex (PMC), parietal cortex, and cerebellum, accompanied by enhanced effective connectivity between motor areas, like the cerebellum, cingulate motor area, supplementary motor area (SMA), and putamen during automatic processing. The stronger connectivity might be a reflection of enhanced synaptic strength during the automatic process (Hebb, 1949). The reduction of brain activity and enhancement of network connectivity together suggests a more efficient neural code for controlling a motor task (Wu et al., 2008; Mazzoni, 2008). In addition, there was weakened connectivity from the DLPFC to motor areas. The DLPFC and ACC are critical in the attentional networks (Jueptner et al., 1997; Petersen et al., 1998; Isomura et al., 2003). The diminished activity and connectivity in these two regions indicates that when a movement achieves automaticity, the attentional networks become less necessary. A recent study investigated changes in the architecture of functional

Table 1
Neural correlates of automaticity in healthy people.

Reference	Task paradigm	Neural changes during automatic process
Wu et al. (2004)	Self-initiated, self-paced, sequential finger movements	Decreased activity in the cerebellum, pre-SMA, CMA, caudate nucleus, PMC, parietal cortex, and prefrontal cortex
Lehe´ricy et al. (2005)	Explicit learning of a sequence of finger movements over a month of training	Decreased activity in the anterior putamen, thalamus, STN, globus pallidus, cerebellum, pons, ACC, rostral premotor and prefrontal areas; Increased activity in the posterior putamen
Poldrack et al. (2005)	A serial reaction time task	Decreased activation in the ventral PMC, inferior prefrontal gyrus, DLPFC, SMA, caudate, putamen and globus pallidus
Wu and Hallett (2005b)	Self-initiated, self-paced, sequential finger movements	Decreased activation in the pre-SMA, PMC, and parietal cortex
Wu et al. (2008)	Self-initiated, self-paced, sequential finger movements	The cerebellum, CMA, pre-SMA and putamen had stronger connectivity with the motor networks; The precuneus had less connectivity with the motor networks
Balsters and Ramnani (2011)	A conditional learning task	Decreased activation in Crus I of cerebellar cortical lobule HVIIA, a target of the prefrontal cortex
Wu et al. (in press)	A visuomotor association task	Decreased connectivity from the DLPFC and anterior putamen to the motor regions; Strengthened connectivity from the posterior putamen to the motor cortex; Re-attending to automatic movement induced more activation in the DLPFC, anterior cingulate cortex, and rostral supplementary motor area, but did not change the activity and connectivity in the striatum

Only results from the studies with dual-task to prove automaticity are summarized here.
ACC, anterior cingulate cortex; CMA, cingulate motor area; DLPFC, dorsolateral prefrontal cortex; PMC, premotor cortex, pre-SMA, rostral supplementary motor area; STN, subthalamic nucleus.

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