



Research report

Time to get a move on: Overcoming bradykinetic movement in Parkinson's disease with artificial sensory guidance generated from biological motion



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H I G H L I G H T S

- Seven idiopathic Parkinson's disease patients were tested in an upper limb movement task.
- Movements were performed with and without point light display (3 different speeds).
- Visual display conveyed biological motion recorded from a healthy adult.
- Patients movement kinematics improved when assisted with a display.
- The kinematics mirrored the temporal characteristics conveyed by the display.

A R T I C L E I N F O

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Paradoxical kinesia describes the motor improvement in Parkinson's disease (PD) triggered by the presence of external sensory information relevant for the movement. This phenomenon has been puzzling scientists for over 60 years, both in neurological and motor control research, with the underpinning mechanism still being the subject of fierce debate. In this paper we present novel evidence supporting the idea that the key to understanding paradoxical kinesia lies in both spatial and temporal information conveyed by the cues and the coupling between perception and action. We tested a group of 7 idiopathic PD patients in an upper limb mediolateral movement task. Movements were performed with and without a visual point light display, travelling at 3 different speeds. The dynamic information presented in the visual point light display depicted three different movement speeds of the same amplitude performed by a healthy adult. The displays were tested and validated on a group of neurologically healthy participants before being tested on the PD group. Our data show that the temporal aspects of the movement (kinematics) in PD can be moderated by the prescribed temporal information presented in a dynamic environmental cue. Patients demonstrated a significant improvement in terms of movement time and peak velocity when executing movement in accordance with the information afforded by the point light display, compared to when the movement of the same amplitude and direction was performed without the display. In all patients we observed the effect of paradoxical kinesia, with a strong relationship between the perceptual information prescribed by the biological motion display and the observed motor performance of the patients.

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1. Introduction

Although Parkinson's disease (PD) is a multifaceted disease, without known provenance, the neurophysiologic changes that take place in the brain are well understood. Idiopathic PD leads to

the progressive degeneration of the dopaminergic cells in the basal ganglia system [1]. This system is responsible for automatic, well-learned movements and is heavily involved in motor programming processes, such as the preparation, initiation and execution of movement and also for maintaining a readiness for action [2]. To perform smooth self-paced actions, such as reaching for a stationary object, the healthy brain needs to supply the basal ganglia with dopamine to allow information to successfully pass between cortical and subcortical structures [1]. However, when patients with PD perform self-paced movement, inadequate activation of the SMA area, anterior cingulate cortex and left putamen has been observed [3].

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Growing evidence in the PD literature suggests that the neurodegeneration of the dopamine producing cells in the basal ganglia impairs the automated and internally paced mode of movement control, but spares the functionality of goal-directed actions [4,5]. The preserved ability of PD patients to ‘overcome’ their compromised motor control (such as slowness of movement and decreased movement range) under specific environmental circumstances is often referred to as *paradoxical kinesia* [6] or *akinesia paradoxical* [7]. The neural theory of paradoxical kinesia assumes that the action triggered by external stimuli bypasses degenerated basal ganglia pathways [8]. In other words, goal-directed movement triggered by sensory information present in the environment is functionally different from self-paced movement where there is no information to prompt movement. Indeed it was not until almost 40 years after these findings that neuroimaging studies confirmed that paradoxical gait stimulated by visual cues induces different brain activation patterns than non-cued gait, mainly enhanced activation in the lateral premotor cortex area of the brain [6].

According to a model proposed by Redgrave [4], paradoxical kinesia can be accounted for by preserved goal-directed loop functionality taking over impaired circuitry of self-generated habitual control. The basal ganglia operate on the basis of micro-circuitries that share a unique somatotopic and behavioural specialisation in processing different sources of sensory information [9]. Four cortical-thalamus loops transfer separate types of signals: sensorimotor, oculomotor, limbic and associative [4]. Ventromedial areas of the basal ganglia receive limbic input; dorsolateral zones receive sensorimotor input, whereas ventromedial and dorsolateral parts are receptive to cognitive information from the frontal cortex [10]. Two separate neural pathways connecting input stations (striatum and subthalamic nucleus (STN) with the thalamus were identified in the seminal work of Albin et al. [11]. The direct pathway was thought to facilitate motor actions with inhibitory connections stretching from the striatum to the substantia nigra compacta and the ventral tegmental area. The indirect pathway was proposed to account for the suppression of the movement with striathalamic interconnections through the globus pallidus external and the subthalamic nuclei.

Recent neurophysiological findings build on the previous model which shows a more multifaceted functionality of those pathways [4]. Although inhibition and facilitation of motor actions are two basic neural mechanisms selecting output to the thalamus and the cortex, the primary division of basal ganglia functionality seems to be linked to the automaticity of the movement [12]. A direct pathway seems to be involved in the habitual control of the movement; that is to say movements that are over-learned and automatic (like walking, making a cup of tea, speech). The habitual control pathway suffers the most from the dopamine depletion associated with PD, whereas the functionality of the indirect pathway involved in the goal-directed control (effortful and slow) is preserved. In this scenario, output from the whole basal ganglia becomes imbalanced due to poor quality and weak excitation from the direct pathway and preserved inhibitory output from the goal-directed system [4]. The increased inhibition with the lack of facilitation of the movement causes slowness of the movement, decreased amplitude of the movement and fatigue. The one-sidedness of basal ganglia output ‘traps’ PD patients in goal-directed control, making every movement effortful and intentional. However, the goal-directed neural loop is sensitive to action-relevant sensory information that can guide self-paced motor actions. When action relevant sensory information is available in the environment improvements in motor performance are observed.

The term ‘cue’ can be explained as a stimulus presented in space and time that provides information about movement initiation and/or execution [13]. In general, cues are divided into spatial and temporal informational sources. Spatial cues can provide

information about *where* to aim a movement (e.g. horizontal lines on the floor conveying information such as “place your foot here”) whilst temporal cues can provide information about *when* to execute the movement (e.g. a metronome that triggers a “move now” response). A good illustration of the difference between goal-directed and intrinsic motor control is the ‘ball rolling down a ramp’ catching task described by Majsak et al. [14]. This study has demonstrated for the first time that paradoxical kinesia also can affect upper limb motion in PD patients. Participants were found to produce movements with higher peak velocities and shorter movement times when they were catching a ball in motion, compared to reaching as fast as they could for a static ball placed at the same distance. In other words, the movement of the ball provides information about when and how they should move if they want to successfully intercept the ball [15]. It has been hypothesised that the availability of dynamic visual information provides us with temporal information that allows us to control our actions respectively [16]. In contrast, when the task is self-paced and the ball is stationary at the same distance, no continuous perceptual information is available from the environment to inform the participant about when and how they should move.

An important consideration in this account is how perceptual information can be used to guide action. The links between the perception and action execution are widely discussed in the literature. One of the most seminal ideas is that neural representations can be shared across perceptual and motor system and stems from the ‘Theory of Event Coding’ proposed by Hommel et al. [17]. This theoretical framework proposes that perception and action share common representation domains and similar neural substrates [18,19]. Our brain uses the same mental representations when we perceive a motor action (and also recollect, imagine or memorise an action) as when we execute the same action (planning and initiation of the movement). It is hypothesised that the neural basis for ‘common coding’ is the mirror neuron system [17]. Mirror neuron cells in the F5 area of the premotor cortex and the inferior parietal lobule in monkeys are activated by both observing and executing a motor action [20]. Unlike the primate studies, based on the single-unit recording of neural activity, evidence for a mirror neuron system in humans is indirect [21]. Studies based on non-invasive electrophysiological methods (e.g. EEG) and brain imaging (fMRI, PET) support the existence of a mirror neuron network in the human brain [22]. Imagining, seeing, hearing or executing an action shares common neural substrates in the brain [23,24]. Those discoveries gave rise to the idea of exploiting the functionality of the mirror neuron system in developing motor rehabilitation techniques that stimulate neural plasticity in stroke survivors [22]. The proposal is that in the absence of actual movement execution the motor representation for the limb still can be stimulated by, for example, observing an action. Christensen, Ilg and Giese [25] have demonstrated a close link between the perception of motor actions and the execution of motor actions, adding more support to the idea that both of those systems use the same neural resources. Perception of temporally or spatially incongruent actions whilst performing a movement was found to interfere with the execution of the movement. The other seminal discovery was that observing a movement synchronous to the movement performed facilitates the recognition of the action observed.

In this paper, we propose a novel approach to understanding paradoxical kinesia, which complements neuroimaging evidence and animal studies by focusing on the role of the temporal information specified in dynamic sensory cues. Cues conveying some form of temporal information, often in an intermittent fashion, have been found to improve spatio-temporal patterns of gait in PD patients [26–28]. The focus of this study was to investigate the use of biological motion of a healthy adult as an alternative cueing paradigm, where the spatio-temporal framework for the movement would be

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