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The effect of Parkinson's disease on time estimation as a function of stimulus duration range and modality

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Abstract

The present research sought to investigate the role of the basal ganglia in timing of sub- and supra-second intervals via an examination of the ability of people with Parkinson's disease (PD) to make temporal judgments in two ranges, 100–500 ms, and 1–5 s. Eighteen non-demented medicated patients with PD were compared with 14 matched controls on a duration-bisection task in which participants were required to discriminate auditory and visual signal durations within each time range. Results showed that patients with PD exhibited more variable duration judgments across both signal modality and duration range than controls, although closer analyses confirmed a timing deficit in the longer duration range only. The findings presented here suggest the bisection procedure may be a useful tool in identifying timing impairments in PD and, more generally, reaffirm the hypothesised role of the basal ganglia in temporal perception at the level of the attentionally mediated internal clock as well as memory retrieval and/or decision-making processes.

Keywords: Timing; Time perception; Temporal processing; Basal ganglia; Bisection; Duration

1. Introduction

In recent years, there has been increasing interest in the neural mechanisms subserving the processing of time, and, in particular, the question of whether the neural substrates and circuitry involved in the temporal processing of intervals of very brief duration (milliseconds) differ from those underlying longer timing intervals (seconds-to-minutes range; Ivry & Keele, 1989; Ivry & Spencer, 2004; Lewis & Miall, 2003b; Rammsayer, 1997). One enduring issue concerns the contribution of frontal-striatal circuits to timing and time perception across these different time intervals (for reviews see Ivry & Spencer, 2004; Meck & Benson, 2002).

Timing research has been heavily influenced by the prominent information-processing model, the Scalar Timing Theory (Gibbon, 1977; Gibbon, Church, & Meck, 1984). The model entails three distinct stages in which temporal information about an event is abstracted, encoded and acted upon (clock, memory, and decision). The essence of the model follows thus: an internal pacemaker with an attention mediated switch emits pulses stored by an accumulator to form an expressed interval of subjective time that approximately corresponds to objective ('real') time. After the designated passage of time has lapsed, the sum value of these accumulated pulses is stored in reference memory which frees the counting procedure to begin again. Expected time values, or pulse totals, are accrued in memory over many trials and inherent pulse values are placed upon certain corresponding events. Any subsequent interval of time discrimination is then compared to existing remembered time references and matched accordingly to the targeted event (Gibbon, 1977; Gibbon et al., 1984). According to this model, each stage is independent and capable of selective readjustment or realignment, and

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alterations to each stage of temporal processing give rise to specific patterns of accuracy and variability in duration judgments (Meck, 1983, 1996). In its simplest form, the scalar property holds that errors of time estimation are strictly proportional to the target times being estimated. This scalar variability can reflect changes in either the clock, the memory, or decision stages. Non-scalar variability (where time estimation errors are disproportionate to target times), however, is assumed to arise from dysfunctional memory encoding or decoding (Meck, 1983, 1996).

A central role for the basal ganglia in the temporal processing of intervals has been strongly implicated by animal research primarily focussed on the internal clock and memory stages of the model. Meck (1983) demonstrated that clock speed and memory stages can be dissociated, both with respect to the brain circuitry that underlies them, and in the time course of their responsiveness to pharmacological manipulations. Whereas administration of cholinergic drugs in rats previously trained to estimate a target duration appeared to modify time representations in reference memory (Meck & Church, 1987), the administration of dopaminergic drugs was shown to alter rats' internal clock speeds, with dopamine antagonists (e.g., haloperidol) typically leading to a 'slow clock', as manifested by underestimations of time intervals (i.e., perceive less time as having elapsed than has actually passed) and overestimations when required to reproduce the timespan, and dopamine agonists typically having the opposite effect (Meck, 1983; Meck, 1996; but see Harper, Bizo, & Peters, 2006). Studies with human subjects have also reported impaired temporal duration discrimination after the administration of the dopamine antagonist haloperidol, indicating that temporal information processing in humans depends on central dopaminergic activity (Lustig & Meck, 2005; Meck, 1996; Rammsayer, 1997; Rammsayer, 1999).

The fact that dopaminergic systems thought to be closely associated with aspects of interval timing are impaired by Parkinson's disease, make this disorder of special interest. Parkinson's disease (PD), a common neurological disorder affecting some 1% of people over the age of 50, is perhaps the most widely studied human model of basal ganglia dysfunction (Saint-Cyr, 2003). Pathological features include marked degeneration and atrophy of the substantia nigra and a consequent major reduction of the dopaminergic projection to the striatum (Agid, Javoy-Agid, & Ruberg, 1987). The resulting dysfunction of striatal circuits is expressed in PD by motor and cognitive symptoms, including the disordered timing of movement, usually manifesting in the form of bradykinesia and/or akinesia (Barbosa, Limongi, & Cummings, 1997; Morris, 2000), and the lengthening of normal information processing time that parallels patient bradykinesia, referred to as bradyphrenia (Rogers, Lees, Smith, Trimble, & Stern, 1987). Cognitive changes in PD may also be closely linked to prefrontal dysfunction with an emergence of neuropsychological impairments in the form of working memory impairments and attentional deficits (Owen et al., 1992; Taylor, Saint-Cyr, & Lang, 1986).

Empirical findings in PD research have tended to reveal a range of specific motor-timing deficits in PD, including increased reaction time (RT) and movement time (Bloxham, Dick, & Moore, 1987), reduced ability to maintain fixed rhythms in tapping tasks (Elsinger et al., 2003; O'Boyle, Freeman, & Cody, 1996), and impaired speech time processing (Breitenstein, Van Lancker, Daum, & Waters, 2001). Dopaminergic-related slowing in PD has also been related to a disturbance in the perception and production of a range of time intervals (for a review, see Meck & Benson, 2002). Time perception studies offer the advantage that the experimental tasks usually have very little or no motor requirements, so that, at least on the surface of it, they are likely to be relatively pure reflections of timing processes. These studies typically use a variety of different methods including verbal time estimation tasks, in which participants are asked to estimate the duration of a given time period (e.g., the length of time a stimulus is presented); duration production tasks, which requires participants to produce a target duration (e.g., press a button after an experienced duration of 30 seconds); and reproduction tasks, in which participants have to evaluate and reproduce a target duration by comparing time during an evaluation phase with one elapsing during the reproduction phase. Nevertheless, there is considerable controversy about whether the timing processes impaired by the disrupted nigrostriatal dopaminergic system in PD affect temporal processing for both short and long durations (Ivry & Spencer, 2004; Rammsayer, 1997, 1999) and whether such deficits reflect a failure of the basal ganglia to perform its timekeeping functions (i.e., a disturbance in the internal clock speed of the scalar model) as opposed to impairment of other more cognitively mediated processes (i.e., disruption to memory and/or decision stages of the scalar model).

A large number of studies of people with Parkinson's disease, many conducted within the framework of scalar timing theory, have almost uniformly reported temporal processing difficulties for intervals within the seconds-to-minutes range (Lange, Tucha, Steup, Gsell, & Naumann, 1995; Malapani, Deweer, & Gibbon, 2002; Malapani et al., 1998; Pastor, Artieda, Jahanshahi, & Obeso, 1992; Perbal et al., 2005; Riesen & Schnider, 2001), leaving little doubt that frontal-striatal circuits are crucially involved in time estimation of longer intervals. The precise nature of this deficiency, however, remains less obvious. For instance, two early studies showed that compared with controls, non-medicated PD patients underestimated durations of different lengths in verbal estimation tasks and overestimated the same durations in reproduction tasks, with the greater magnitude of overestimation occurring at longer interval (scalar variability) (Lange et al., 1995; Pastor et al., 1992). Both sets of authors interpreted the results as evidence that the internal clock was running at a slower rate in patients than control participants. Notably, in both