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RESEARCH ARTICLE

Impaired conflict monitoring in Parkinson's disease patients during an oculomotor redirect task

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Abstract Fallibility is inherent in human cognition and so a system that will monitor performance is indispensable. While behavioral evidence for such a system derives from the finding that subjects slow down after trials that are likely to produce errors, the neural and behavioral characterization that enables such control is incomplete. Here, we report a specific role for dopamine/basal ganglia in response conflict by accessing deficits in performance monitoring in patients with Parkinson's disease. To characterize such a deficit, we used a modification of the oculomotor countermanding task to show that slowing down of responses that generate robust response conflict, and not post-error per se, is deficient in Parkinson's disease patients. Poor performance adjustment could be either due to impaired ability to slow RT subsequent to conflicts or due to impaired response conflict recognition. If the latter hypothesis was true, then PD subjects should show evidence of impaired error detection/correction, which was found to be the case. These results make a strong case for impaired performance monitoring in Parkinson's patients.

Keywords Conflicts · Error correction · Saccades · Basal ganglia · Double-step · Countermanding

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Introduction

Because human behavior is at times erratic, most theories of cognition (Logan 1985; Shallice 1988; Stuss and Benson 1986) have an explicit or implicit characterization of a performance monitoring system that configures the overall cognitive system at different temporal scales, such that errors are immediately corrected (and chances of future errors are minimized (Rabbitt 1966, 1968). It has been traditionally assumed that the primary signal driving this aspect of cognitive control is error. In contrast, an alternate view, the so-called conflict monitoring hypothesis, (e.g., Botvinick et al. 2001) contends that the performance monitoring system is driven primarily by the presence of response conflicts and not errors per se. Using such response conflicts as the primary signal that recruits cognitive control, Botvinick et al. (2001) were able to simulate the experimental results of post-error slowing.

Insights into the role of conflict in performance monitoring have also been derived from studies of saccadic countermanding (reviewed in Schall 2001). Typically in a saccade countermanding task, subjects are instructed to make saccade to the appearance of a target. On certain infrequent random trials following the appearance of a target, a second 'STOP' signal occurs that instructs subjects to cancel their planned saccade. The countermanding task has been assumed to involve a race between a GO process, leading to the execution of the preponderant

response, and a STOP process, which inhibits the GO process. Neurophysiological and modeling results identify the GO process with the activity of movement neurons in oculomotor regions of the brain and STOP process with fixation neurons (Boucher et al. 2007). The outcome of the race between these processes determines whether the subject is able to inhibit his/her response or make an error. It has also been found that co-activation of these processes (which are antagonistic to each other), generating response conflict, occurs much more in cases when the subject is able to inhibit the response compared to when the error occurs (Hanes et al. 1998; Boucher et al. 2007). Such a task then allows one to dissociate errors from response conflict generation. In support of the conflict monitoring hypothesis, Emeric et al. (2007) found that slowing down of responses was much more on post-conflict trials compared to post-erroneous ones.

Although traditionally frontal lobes (Shallice and Burgess 1996) and fronto-parietal circuits (Dehaene et al. 1998) have been considered to be the neural basis of cognitive control, two important aspects of basal ganglia (BG) circuitry make it plausible to think of its role in performance monitoring. First, dopamine neurons of the ventral tegmental area and substantia nigra (an important BG structure) project to brain structures involved in motivation and goal-directed behavior, for example, the striatum, nucleus accumbens, and frontal cortex. Evidence supports the idea that these neurons construct and distribute information about rewarding events and/or the likelihood of positive outcomes of actions for performance monitoring (Schultz 1997). This information would be critical for the performance monitoring system. For example, Holroyd and Coles (2002) suggested that absence of reward induces phasic decreases in mesencephalic dopaminergic activity and that is when the system first determines that ongoing events are worse than expected. Second, anatomically the BG is intimately connected to the ACC. In non-human primates, anterior cingulated cortex (ACC) receives inputs from supplementary eye fields (SEF), pre-supplementary motor area (preSMA), dorsal premotor cortex as well as thalamic nuclei (venteroanterior nucleus-pars caudalis and oral part of venterolateral nucleus; Hatanaka et al. 2003). These are sites of efferents from the basal ganglia (BG) network, specifically the pallidum. The ACC in turn projects to premotor and caudal motor areas, SEF, FEF as well as the striatum, particularly in the putamen and the striatal cell bridges. Lateral prefrontal cortex, another area known to be important for cognitive control and error processing, is also connected with the rostral striatum (Dum and Strick 1993). Intimate connections between BG and effector systems provide an additional way through which BG could be a part of the performance monitoring network. For example, in the oculomotor system, it is known that basal ganglia tonically inhibits SC, which seems critical for preventing information overload given the high convergence of information from multiple spatial representations to the SC (Redgrave 1999). It has been suggested that this tonic inhibition could determine the threshold for oculomotor decisions (Lo and Wang 2006) and so by adjusting this, the BG could adjust performance in response to cognitive demands.

Since BG is involved in aspects of cognition, it is not surprising that PD patients (PDs), where the depletion of dopamine in the substantia nigra pars compacta is thought to alter information processing within BG, show a host of deficits that are more cognitive in nature. Apart from classical motor symptoms, patients also show evidence of executive dysfunction (Owen 2004) similar to that seen after frontal lobe lesions, like poor inhibitory control (Joti et al. 2007), poor working memory (Bradley et al. 1989), and difficulty in set shifting (Lewis et al. 2005). In the context of performance monitoring deficits in PD, Angel (1971) found that PDs were slower at correcting their motor errors. This finding was anticipated by the work of Divac et al. (1967) who proposed that the caudate forms part of a neural mechanism for achieving error correction in motor systems. However, behavioral evidence in terms of impaired error processing and correction in PDs is sparse and mixed. Holroyd et al. 2002, for example, did not find any evidence of poor error correction in PDs on a choice reaction time task. The other aspect of performance monitoring, performance adjustments after errors, has been studied by Ito and Kitagawa (2006), who report no significant effect in PD patients in a lexical decision-making task. Considering what is known about BG functioning with respect to executive control, modulation of motor responses, motor learning and information flow from prefrontal to motor areas—issues highly relevant to performance adjustment in response to errors or conflicts for response adjustment—this is surprising.

To study performance monitoring in PD patients, we used a modification of the double-step task, called the redirect task, in which the appearance of the second peripheral target on infrequent random trials served as a *redirect signal* instructing the subject to cancel a preplanned saccade and direct gaze to the location of the second target to obtain reward. Although subjects are able to perform this task fairly successfully, they often make errors when they fail to redirect their saccade to the new target; they then make a sequence of two saccades: the initial erroneous saccade to the location of the original target followed by a second corrective saccade to the new target. Because the appearance of the second target signals the need for canceling the planned saccade, the redirect task is closely related to the countermanding task in terms of the underlying race model (Camalier et al. 2007; Kapoor and Murthy 2008; Murthy et al. 2009; Ramakrishnan et al.

2010). The estimates of the duration of the STOP process, as derived from the countermanding task (called stop signal reaction time or SSRT) and the redirect task (called target step reaction time or TSRT), are both about 100 ms. Neurophysiological correlates of the STOP and GO processes also appear to be similar, and suppression of neural activity among frontal eye field (FEF) movement neurons is quantitatively and qualitatively similar for countermanding and redirect behavior (Hanes et al. 1998; Murthy et al. 2009). Therefore, as noted earlier, like the countermanding task, the redirect task allows for the dissociation of conflict generation from error. Here, we use the redirect task to study deficits of performance monitoring in PD patients.

Methods

In this study, we report the data from two sets of PD patients and aged-matched controls that performed a visually guided and a memory-guided redirect task. The primary aim of using a visually guided task and a memory-guided task was to determine whether these findings in PD patients generalize across tasks. In a previous study in our laboratory (Kapoor and Murthy 2008), we found that a similar race model architecture consisting of a GO and STOP signal could explain redirect behavior in both kinds of tasks. However, since it has been suggested (Schultz and Romo 1992) that the basal ganglia maybe preferentially engaged in behaviors that are internally generated, we wished to test whether performance monitoring may be task specific. For models of executive control, this could be relevant because performance monitoring should ideally not be dependent on the specifics of the task per se as long as both tasks instantiate conflict and error.

Subjects

Patients were from All India Institute of Medical Sciences (AIIMS), New Delhi, diagnosed as having idiopathic PD in the absence of dementia by a consultant neurologist; their motor disabilities being responsive to anti-Parkinsonian medication. Ten Parkinson's patients (mean age 57.6 ± 2.7) and eleven age-matched controls (58 ± 1.4) were tested on a visually guided redirect task (Table 1). Inhibitory control of these patients has been previously reported (Joti et al. 2007). Another group of eleven (see Table 1) Parkinson's patients (54.4 ± 2.2) and matched controls (55.3 ± 2.3) was examined on a memory-guided redirect task. For all subjects, tests were conducted at least 6 h after their last medication, by which time all patients began exhibiting Parkinsonian symptoms. Disease severity varied between Hoehn–Yahr stages 1 and 4 (Hoehn and Yahr 1967). All subjects gave their informed consent in accordance with the institutional ethics committee of National Brain Research Centre and the Declaration of Helsinki.

Table 1 Background data for Parkinson's disease patients in VGR and MGR tasks

	Age (years)	Hoehn-Yahr	Duration (years)
Patient (VGR)			
BS	37	2.5	7
DN	65	3	21
KB	55	1	2.5
MK	56	2	5
MN	57	1	9
PT	67	1	3
RB	65	2.5	4
RG	54	1	4
SB	63	2.5	13
SI	58	2	13
Patient (MGR)			
AKP	55	2	6
PNS	67	1.5	5
RP	55	3	7
OA	58	1	1
AK	40	1.5	3
VM	52	2.5	6
AS	55	4	15
VB	47	4	4
VS	65	4	20
RK	50	1.5	6

Task

Visually guided redirect (VGR)

In this task (Fig. 1a), the majority of trials (60%) were *no-step* trials. In these trials, following fixation for a random duration that ranged from 300 to 800 ms, a red target (1° by 1°; 6.5 cd/m^2) appeared in one of the six possible locations on an imaginary circle (except vertical up and down positions) of radius 12°, centered on the fixation point. The targets remained on the screen for the duration of the task. Subjects were required to make a saccade as soon as the target appeared. In the remaining (40%) trials called *step* trials, the first red target would be followed by a second target (1° by 1° green; 5.83 cd/m^2) at a temporal asynchrony, called target step delay (TSD), which was varied randomly across step trials. An angular separation of ≥90° was maintained between the initial and final targets to replace saccade averaging (Ottes et al. 1984) and prevent localization errors. For such trials, the subjects were instructed to saccade directly to the later appearing green target. This required subjects to cancel their initial plan to

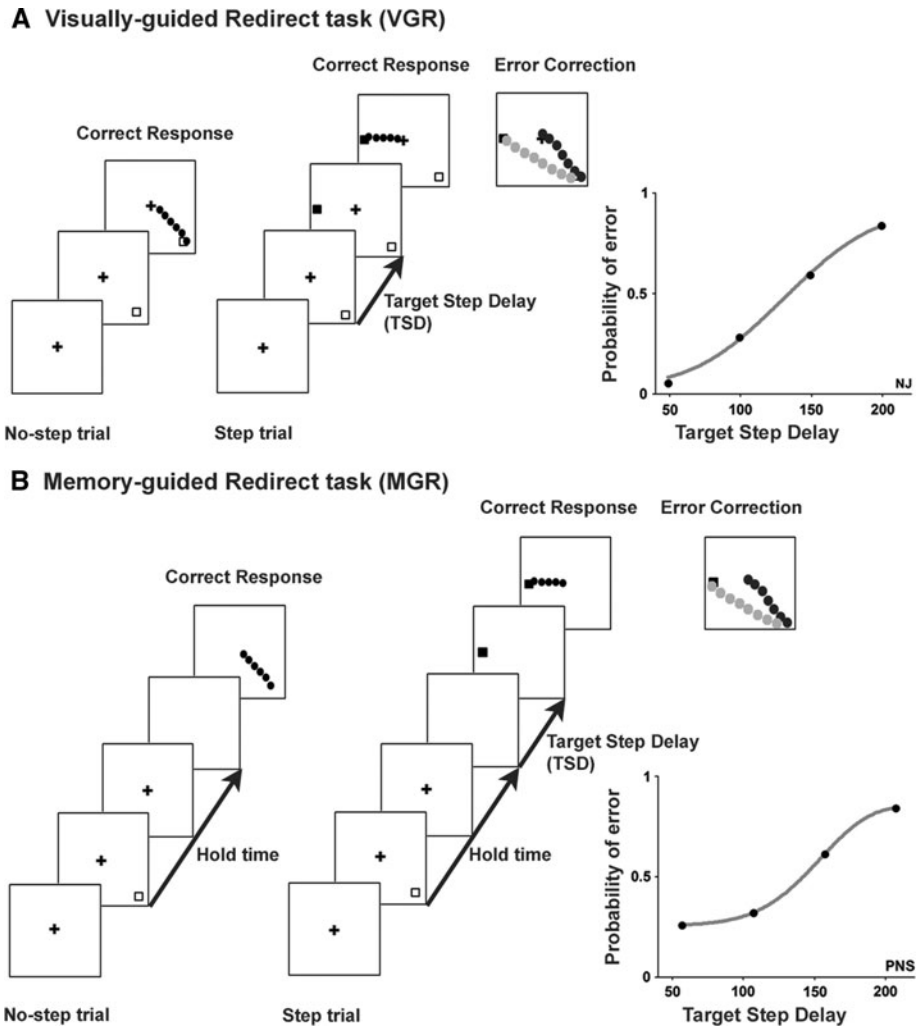

Fig. 1 On step trials, subjects were instructed to cancel the planned eye movement to the initial target and direct gaze to the final target. Step trials are interleaved randomly with no-step trials wherein a second target did not appear. **a** In the visually guided redirect (VGR) task, the cue to make a saccade is the appearance of the initial target. The subsequent appearance of the final target on step trials instructed subjects to cancel the initial planned eye movement and redirect gaze toward the new target. Performance of a subject on a VGR task (*right bottom* panel) plotted as a fraction of erroneous step trials in which subject made a saccade to the first target. The data points represent the fraction of such trials at different target step delays. The *gray line* through the data points represents the best-fit cumulative Weibull function. The plot shows that the probability of making a saccade directed at the initial target increases with target step delay. **b** In the memory-guided redirect (MGR) task, the initial target appeared for a brief duration of 100 ms, but the cue to make a saccade was the disappearance of the fixation spot, which occurred ∼1000 ms (±300 ms) later. The signal for redirection in step trials was the same as in (**a**). The *right panel* shows the performance of a subject on a MGR task

move toward the red target. Step and no-step trials were randomly interleaved. Figure 1a (right bottom panel) describes the performance of a subject on the VGR task.

Memory-guided redirect (MGR)

In this task (Fig. 1b), on the no-step trials (60%), following fixation for a random duration that ranged from 300 to 800 ms, **a** red target (1° by 1°; 6.5 cd/m^2) appeared for ∼100 ms (accurate to the refresh rate) in one of six possible locations on an imaginary circle (except vertical up and down positions) of radius either 12° or 18°, centered on the fixation point. Subjects were to continue fixation for a further 700–1300 ms (called the hold time), following which a cue occurred, in the form of the fixation spot switching off, to make a saccade to the remembered location of the target. Step trials had a second target (1° by 1° green; 5.83 cd/m^2) appearing after a target step delay (similar to the previous task), measured relative to the fixation spot turning off. Subjects were required to saccade directly to the new (green) target, canceling their initial plan to make a memory-guided saccade. Figure 1b (right panel) describes the performance of a subject on the MGR task.

Trials were classified online as correct or erroneous by programming an electronic window centered on each target using TEMPO software (Reflective Computing) that displayed the eye traces in real time and gave immediate auditory feedback to subjects following successful trials only. Trials were scored as successful if subjects fixated their gaze, after the presentation of the GO cue, within a 3° electronic window (visible only to the experimenter) that circumscribed the center of the target or final target in no-step and step trials, respectively. Incorrect step trials were classified online as those trials whose first saccades were directed at the initial target window. Online classification was corroborated offline using MATLAB. In the offline analyses, we also included hypometric saccades directed toward the first target as erroneous trials, since they represented failure to cancel their initial response. Any step trial in which the first saccade fell short by more than 30% of the average saccade amplitude in correct no-step trials was considered an incorrect step response.

In both tasks, subjects were given prior verbal instructions and 50–100 practice trials. Trials were run in blocks of 100, and on an average, a total of 500 trials were completed for each subject. The performance of subjects was assessed by means of a compensation function, which describes the probability of making a non-canceled saccade (erroneous saccade) as a function of TSD (see Fig. 1). The increasing compensation function arises because increasing target step delays the onset of the STOP process, increasing the probability that the GO process will finish before the STOP process. Subjects' performances were quantified by fitting the best-fit cumulative Weibull function weighted by the inverse of the error associated with each data point.

$$W(t) = \gamma - (\gamma - \delta)\exp^{\left(-\left(\frac{t}{\alpha}\right)^{\beta}\right)}$$

where t is the target step delay; α is the time at which the function reaches 64% of its full growth; β is the slope; γ is the maximum value of the function; and δ is the minimum value of the function. We used the cumulative Weibull function to estimate the degree of monotonic progression of the data across the four target switch delays in order to access subjects' performance. Since the term $(\gamma - \delta)$ describes the increase in the probability of making a saccade directed at the first target, we used it as a cancel index to describe the monotonic dependence of the data as a function of TSD and to quantify the degree of cancellation. Most of our subjects had a value of 0.2 or above for this index. The right bottom panels in Fig. 1a and b show the compensation function plots for two subjects on VGR and MGR tasks, respectively.

Recording set up

Experiments were under computer control using TEMPO/VIDEOSYNC software (Reflective Computing, St. Louis, USA) that displayed visual stimuli and sampled and stored eye position and other behavioral parameters. Eye position was sampled at 200 Hz with an infrared pupil tracker (ISCAN, Boston, USA) that interfaced with the TEMPO software in real time. Before starting the recording session, each subject was made to look at 5 positions on the monitor: one at fixation in the center of the monitor and at least 4 (horizontal left, right; vertical up, down) target positions. The monitor was typically 57 cm from the subject. While subjects fixated the targets, we set the horizontal and vertical gain parameters in the ISCAN and TEMPO software, which displayed ISCAN eye data in real time, such that end point of the saccade would typically be in the center of the electronic windows centered on their respective target positions (but visible only to the experimenter). Since the electronic window (for fixation and target position) was displayed throughout the experiment, we could adjust the gains and recalibrate the fixation spot from time to time to compensate for drifts and slight changes in head positions. To facilitate calibration across trials, each trial began only after subjects' eye position was deemed to be within the limits set by the fixation window ±3°. In our experiment, targets were displayed at an eccentricity of either 12° or 18°. The minimum angular separation between the two targets in a step trial was 90°. Thus, the minimum spatial separation between two targets was at least 17°. Thus, the error introduced as a consequence of our calibration procedure (±3°) and the typical accuracy of the tracker ($\sim 1°$) was well within limits to be confident that trials were correctly classified.

Data analysis

All offline analysis was performed using Matlab (Mathworks, USA). The analogue eye position data were smoothed from which blinks were removed. A velocity threshold of 30°/s was used to demarcate the initiation of saccades. The saccade detection algorithm was subsequently verified manually for every saccade. All blink-perturbed saccades were eliminated from analysis. Trials in which saccade latency was <80 ms (anticipatory saccades) were rejected. All statistical tests were done using SigmaStat or Matlab.

Results

Three groups of no-step trials were created depending upon the preceding trial and performance on it. Post-conflict trials were no-step trials that were preceded by a

successfully canceled step trial; post erroneous were the no-step trials that had a preceding erroneous step trial; and no-step trials following no-step trials were grouped as post-no-step trials. If performance history and response conflict are to have any effect on subsequent behavior, then the reaction time (RT) on these three kinds of trials should be different. This analysis was done for both PDs and controls.

In agreement with Emeric et al. 2007, it was found that for normal subjects, increase in RT on post-conflict trials (which presumably generate more conflict) is more than post-error trials, when compared to post-no-step trials (see Fig. 2). We conducted a two-way balanced ANOVA on the data with trial-type (post-no-step, post-conflict, and post-error) and task type (VGR, MGR) as factors. We found a significant difference in the reaction times across the two tasks ($P = 0.009$). Moreover, the interaction between trial type and reaction times was also found to be significant ($P = 0.005$; Fig. 2), though multiple comparisons of the data (using Bonferroni adjustment) showed a significant increase in reaction times on post-conflict trials ($P = 0.004$) only when compared to the post-no-step trials. Subsequent planned comparisons revealed that in VGR, controls showed a significant increase post-error compared to post-no-step (261.2 ± 6.4 (SEM) ms compared to 244.6 ± 6.9 ms, respectively; paired t test $t = -5.9$; $P < 0.001$; $df = 10$). The increase in reaction times, however, was most for post-conflict trials (272.1 ± 8.8 ms compared to 244.6 ± 6.9 ms; paired t test $t = -4.7$, $P < 0.001$; $df = 10$). There was no significant difference in the RT on post-error vs. post-conflict trials (paired t test $t = -1.7$; $P = 0.118$; $df = 10$). In MGR, the mean post-error RT (281.0 ± 9.4 ms) did not differ significantly from mean post-no-step RT (270.6 ± 11.3 ms; paired t test $P = 0.06$; $t = -2.1$; $df = 10$). However, the mean post-conflict RT (304.9 ± 11.9 ms) was significantly higher than the post-no-step RT (270.6 ± 11.3 ms; paired t test $t = -7.3$, $P < 0.01$, $df = 10$). The data for individual subjects in VGR and MGR are shown in the respective panels below. It thus seems likely that more than error, response conflict is the more potent signal for performance monitoring at longer time scales.

To test whether response slowing was impaired in Parkinson's disease patients, we performed a two-way unbalanced ANOVA on the PD patients' data (see Fig. 3). Although we observed a significant difference in the reaction times between VGR and MGR ($P < 0.05$), we found no significant difference ($P = 0.799$) in the reaction times of post-no-step, post-conflict, and post-error trials. In VGR, PD patients showed a modest decrease in reaction times on post-error trials (273.3 ± 13.8 ms) and post-conflict trials (265.9 ± 12.2 ms) when compared to the post-no-step trials (275.6 ± 12.2 ms); though this decrease from the post-no-step RT was not significant (post-error paired t test $t = 0.32$, $P = 0.76$, $df = 9$; post-conflict paired t test $t = 1.56$, $P = 0.15$, $df = 9$). In the MGR, patients showed a trend similar to the controls, wherein the mean post-no-step RT (298.9 ± 5.7 ms) was lower than that of mean post-error (315.5 ± 9.8 ms) or post-conflict

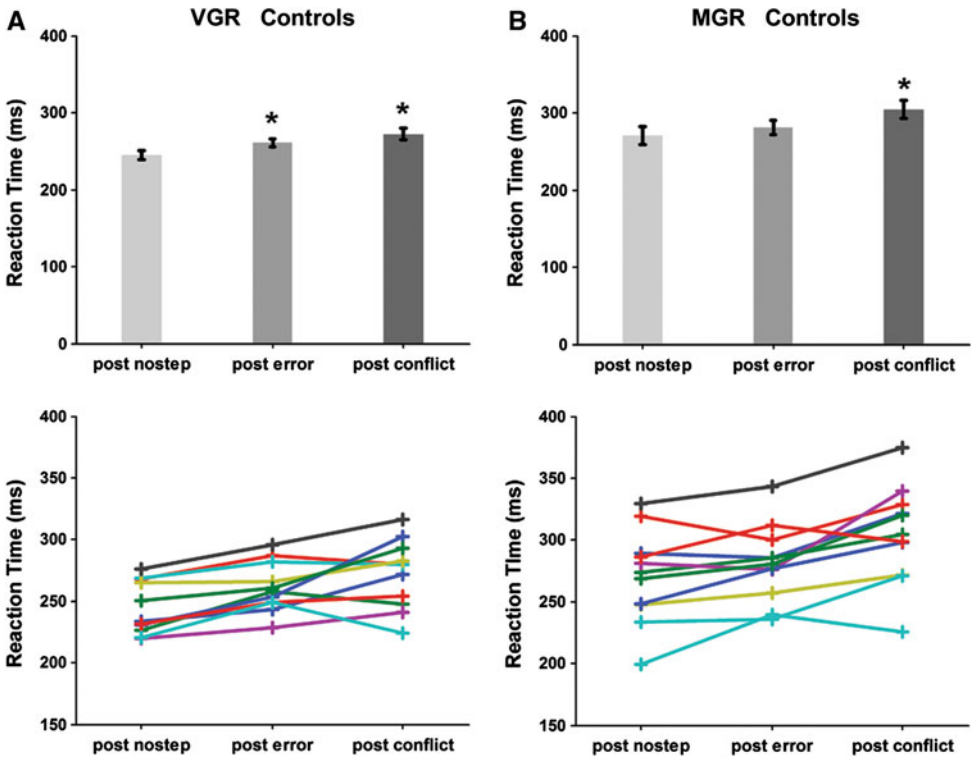

Fig. 2 Controls in VGR and MGR showed higher RTs in post-step trials than post-no-step trials. The increase was maximum for post-conflict trials, being significantly higher than post-no-step in both tasks. The data for individual subjects are shown in the panels below

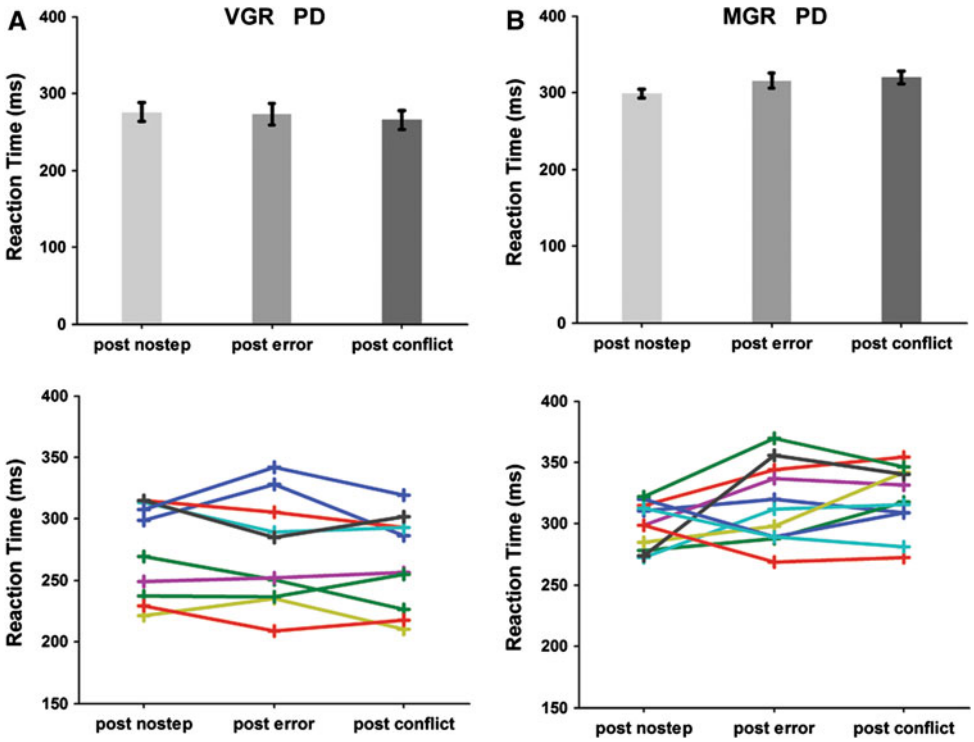

Fig. 3 **a** In VGR, PD patients show no change in RT post-error or post-conflict. **b** Although in MGR they do show a trend toward increasing RT post-error and post-conflict, similar to controls, the trend is not significant. The data for individual subjects are shown in the panels below

(319.9 ± 7.9 ms). However, unlike in controls, these RT differences were not statistically significant (post-error paired t test $t = -1.49$, $P = 0.17$, $df = 10$; post-conflict paired t test $t = -2.08$, $P = 0.06$, $df = 10$; Fig. 3).

Poor performance adjustment could be either due to impaired ability to slow RT subsequent to conflicts or due to impaired response conflict recognition. If the latter hypothesis was true then PD subjects should show evidence of impaired error detection/correction. Errors were defined as those step trials where subjects failed to cancel the saccade directed to the first target. The extent of errors in control subjects and PD patients across the two tasks were estimated using a two-way unbalanced ANOVA. The percentage of errors committed in VGR and MGR tasks were not different ($P = 0.99$). Moreover, we did not find a significant difference in the percentage of errors committed by the PD and controls across the two tasks ($P = 0.10$). In both the tasks, the frequencies of errors committed were same for both controls (VGR 37.2 ± 1.3%; MGR 34.7 ± 1.4%) and PD subjects (VGR 32.3 ± 1.5%; MGR 34.9 ± 1.4%).

To test whether PD subjects were impaired on error detection/correction, PD patients and controls were analyzed for the percentage of errors they corrected. A two-way ANOVA was done on the error correction data. The percentage of error correction in VGR was significantly greater than that in the MGR ($P \leq 0.001$). More importantly, the percentage of errors that were corrected was significantly poorer in PD patients relative to controls ($P \leq 0.001$). Planned comparisons for MGR revealed that PD patients were significantly poorer in error correction when compared to the controls ($t = -4.78$; $p < 0.001$; $df = 20$). Whereas the average (±standard error) of corrective movement in PD patients was only 28.16% (±5.19%) of their errors, controls corrected about 58.89% (±4.73%) of their errors. A similar trend was also observed for VGR; while controls corrected 87.71% (±3.24%) of their errors, PD patients corrected 82.97% (±2.90%) of their errors. However, this trend was not significant ($t = -1.09$; $P = 0.294$; $df = 19$). If conflict recognition does contribute to error correction, then better error correction is expected at lower TSDs. This is because at lower TSDs, the co-activation of GO and STOP processes (which are antagonistic to each other), generating response conflict, is expected to be more relative to longer TSDs, when the potential overlap is smaller. We therefore further analyzed the extent of error detection/correction at short vs long TSDs for controls and PD subjects (see Fig. 4). Consistent with the conflict recognition hypothesis, in VGR, controls showed significantly greater extent of error correction at short TSDs (93.7 ± 1.3%) when compared to the long TSDs (83.8 ± 2.1%) trials (paired t test $t = 7.56$; $P < 0.001$; $df = 10$). In MGR also, controls showed a similar trend. Percentage of error correction at the short TSD (62.5 ± 4.9%) was significantly (paired t test $t = 4.86$; $P < 0.001$; $df = 11$) greater than at the long TSD (42.4 ± 6.5%). However, PD subjects showed no significant difference in error correction at short vs long

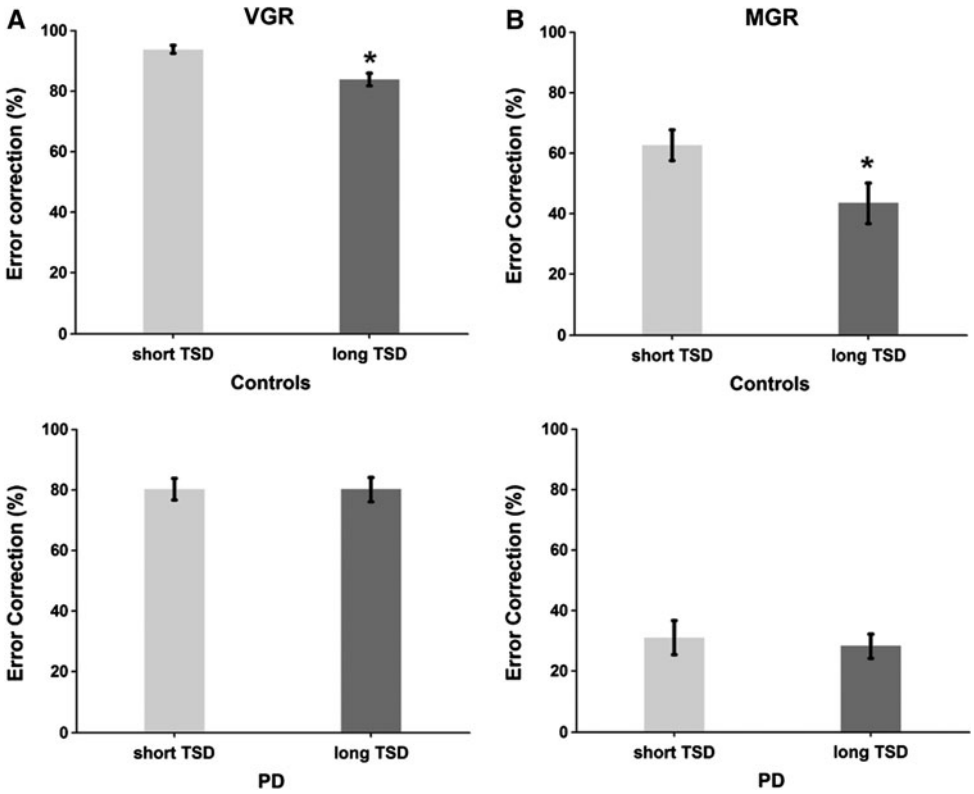

Fig. 4 Fast online error detection/correction at short versus long TSDs. Controls in both VGR (**a**) and MGR (**b**) showed significantly decreased extent of error detection/correction at long TSDs when compared to short TSDs. No significant difference was observed for PD subjects (*bottom panels*)

TSDs. In VGR, PD subjects corrected 80.2 ± 3.6% and 80.1 ± 4.1% of their errors (paired t test $t = 0.04$; $P = 0.97$; $df = 9$) at short and long TSD, respectivley. Similarly in MGR, the error correction for PD subjects was found to be 31.1 ± 5.9% at short TSD and 28.3 ± 4.2% at long TSDs (paired t test $t = 1.21$; $P = 0.25$; $df = 10$).

Discussion

In this study, we report a primary deficit in conflict monitoring among PD patients that prevents the slowing down off subsequent responses. A consequence of impaired conflict monitoring could in turn produce impairment in error detection/correction that leads to compromised fast online error correction as well. We discuss the implications of these results below.

A critical aspect of supervisory control is performance adjustment that causes a slowing down on subsequent trials. While the signal for adjustment has traditionally been thought to be error, recent evidence has put forth the case for the presence of response conflicts being the primary signal for adjustment. Our finding of more robust slowing down on post-conflict step trials favors the latter view. The basis for this result can be better understood by considering the nature of control underlying performance in the redirect task that requires among other things a race between a GO process and a STOP process toward a threshold (Camalier et al. 2007; Ramakrishnan et al. 2010). The outcome of step trials, as to whether the initial saccade will be canceled, depends upon the outcome of the race between these two processes. Neurophysiological evidence puts the GO process with the buildup of movement neurons and the STOP process with the activity of fixation neurons (Boucher et al. 2007). The neurophysiology of the countermanding task, which is conceptually similar to the redirect task (Kapoor and Murthy 2008), suggests that co-activation of movement and fixation neurons, can instantiate a form of response conflict because two conflicting response units are simultaneously active and occurs maximally on the compensated step trials (Hanes et al. 1998; for review see Stuphorn and Schall 2002). In contrast, in erroneous step trials, the activation of STOP process may not be maximal therefore resulting in lesser conflicts between the GO and STOP processes. Thus, considered in the light of neurophysiological evidence, our results suggest that the presence of conflict is a more important determinant of performance adjustment than the presence of errors per se.

In contrast to control that manifest across trials, impaired post-conflict slowing may also arise indirectly as a consequence of PD patients making more reflexive saccades toward the suddenly presented saccade than do controls (Kingstone et al. 2002; Roll et al. 1996; Briand et al. 1999; Chan et al. 2005). Thus, it is possible that the reason why there was no significant change in RT in the post-conflict trials when compared to no-step trials could be that PD

patients just made reflexive saccades to the suddenly appearing target and not due to impairment in slowing RT subsequent conflicts or due to impaired conflict recognition. There are potentially three reasons why we believe the absence of post-conflict slowing is not a direct consequence of PD patients making more reflexive saccades. First, significant post-conflict slowing was also absent for memory-guided task saccades that are not reflexive in nature. Second, a comparison of the no-step reaction times in visually guided and memory-guided redirecting, reveal similar patterns between PD and controls, i.e., in both groups memory-guided saccades have longer latencies than in the visually guided condition. Further, correct no-step saccadic reaction time in both the memory-guided and visually guided conditions are actually larger in PD subjects compared to controls. Taken together, our results suggest that PD patients have impaired post-conflict slowing.

Poor performance adjustment could be a manifestation of poor conflict recognition or due to poor modulation of decision or response parameters required to bring the change in performance. Poor recognition of conflict could offer a unitary explanation for the findings of poor error correction and poor response adjustment. Error correction could be driven in part by the detection of conflicts and so an impaired recognition of the presence of response conflict could lead to poor programming of corrective actions. Our findings for impaired error correction in PD subjects, particularly at short target step delays, when conflict is expected to be high, lends support to the conflict recognition hypothesis. The absence of a significant effect across all target step delays, particularly in VGR, is a consequence of the nature of the redirect task where a higher proportion of errors is expected at higher target step delays (see Fig. 1). Hence, the error correction data tends to be biased for higher target step delays where signals other than conflict (these could be feedback or explicit error signals) may play an important role in driving error correction. Neurophysiological evidence for an impaired error/conflict detection system in PD also derives from a number of electrophysiological studies that have examined the so-called error-related negativity (ERN), an event-related potential (ERP) that is considered the most important evidence for the existence of a neural system for performance monitoring in humans. The ERN, whose amplitude is correlated with errors and the magnitude of post-error slowing down (Gehring et al. 1993; Dehaene et al. 1994; Ridderinkhof et al. 2004; Ullsperger and von Cramon 2004; Williams et al. 2004), is found to be attenuated in PD patients (Willemssen et al. 2009; Falkenstein et al. 2001; Stemmer et al. 2007; Ito and Kitagawa 2006; but see Holroyd et al. 2002).

However, dissociation between errors and conflicts has been reported previously (Swick and Turken 2002) and behavioral dissociation involving PD patients has been recently reported by Ito and Kitagawa (2006). In their study, they found that although PD patients exhibited impaired fast error correction, performance adjustments after errors were found to be normal. This observation is in contrast to what we report here. While differences in task structures preclude a strong justification for this discrepancy, it is interesting to note that redirection necessarily involves instantiating conflict via the opposing GO and STOP responses. Such conflict might have been less in the lexical decision-making task of Ito and Kitagawa 2006 and hence performance adjustments might be more, dependent on explicit error detection signals. This dependency between conflict-induced and error-induced corrections might also be sensitive to the inter-trial intervals, which were typically much shorter in our task as well.

In this paper, while we have chosen to focus on the similarity across tasks, particularly the increase in RT post-conflict, there are some differences in controls that are task specific. In particular, the post-error signals in MGR do not show an increase in RT like VGR. One possible reason for this difference may be the absence of a visual target in MGR that may drive post-error slowing, as in VGR. Similarly, differences between correction rates in MGR versus VGR could be because error correction maybe more "automatic" in visually guided redirect, because of the presence of the target compared with memory-guided redirect. In addition to aspects of the task, differences between PD and controls may also reflect differences in motivational requirements between the two tasks. These differences notwithstanding, our results demonstrate impairment in PDs of performance monitoring that is not task specific. For models of executive control, this is important because performance monitoring should ideally not be dependent on the specifics of the task per se as long as both tasks instantiate conflict and error. While these results suggest that models of executive control may need to consider the contribution of basal ganglia, changes in brain organization as a result of PD may preclude a straightforward interpretation of relating deficits in performance monitoring to normal basal ganglia function per se. Thus, it is possible that these deficits might also be due to changes in frontal circuits that operate in close unison with basal ganglia.

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