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Patients with Parkinson's disease are less affected than healthy persons by relevant response-unrelated features in visual search

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ABSTRACT

Patients with Parkinson's disease (PD) respond more readily than healthy controls to irrelevant stimuli that contain task-relevant, response-priming features. This behavior may reflect oversensitivity to response-relevant features of irrelevant stimuli or failure to select relevant stimuli. To decide between these alternatives, we investigated in a "contingent-capture" paradigm whether PD patients are also oversensitive to task-relevant features that do not prime responses. PD patients and healthy controls had to report the orientation of bars in target color, presented among bars of other colors. Critically, target arrays were preceded by arrays of rings, all gray except one which might be the target color and might be presented at the same position as the upcoming target. Replicating earlier results from young healthy participants (Eimer, Kiss, Press, & Sauter, 2009), signal rings in target color induced an N2pc component over contralateral visual cortex and some positivity at anterior sites (d-P200), both indicative of attentional capture. Correspondingly, signals in target color facilitated correct responding to upcoming targets presented at the same location and impeded correct responses otherwise.

Patients with PD had diminished N2pc, lacked the frontal focus of d-P200, and their responses tended to be less affected than healthy participants' by signal position. Thus PD patients appeared less affected than healthy persons by stimuli with relevant features. This outcome is compatible with the notion that PD patients have poorer internal representations of what is relevant in a given task.

1. Introduction

1.1. Cognitive dysfunctions in Parkinson's disease

The cardinal symptoms of Parkinson's disease (PD) are manifest in problems of executing movements. Cognitive impairments related to movement control have been suggested to be relevant sources of those problems (Brown & Marsden, 1990; O'Brien & Shoulson, 1993; Dubois & Pillon, 1997). Such cognitive deficits include impaired response selection, reduced ability to take unambiguous decisions on how to act, and proneness to impulsive actions (Cools, Barker, Sahakian, & Robbins, 2001; Franz & Miller, 2002; Maddox, Aparicio, Marchant, & Ivry, 2005; Robertson & Flowers, 1990; Wascher et al. 1997). More generally, beyond movement control, PD patients have often been described as having difficulties in clearly distinguishing and flexibly switching between alternatives. These difficulties are part and parcel of what has been called "dysexecutive syndrome", to be distinguished from dementing processes in PD (e.g., Brown & Marsden, 1990; Ceravolo, Pagni, Tognoni, & Bonuccelli, 2012; Cools, Rogers, Barker, & Robbins, 2010; Ravizza & Ivry, 2001; Williams-Gray et al. 2009).

1.2. Sensitivity to response priming in Parkinson's disease

PD cognitive dysfunctions have proven well accessible to quantitative assessment, by measuring how responses to target stimuli are affected by irrelevant stimuli that share some relevant feature with target stimuli. Such "mock-relevant" stimuli might flank or precede the targets as visible or masked priming stimuli. The extent of priming by such mock-relevant stimuli has often been found to be larger in PD patients than in age-matched healthy participants (Machado, Devine, & Wyatt, 2009; Praamstra, Stegeman, Cools, & Horstink, 1998; Seiss & Praamstra, 2006; Troche, Trenkwalder, Morelli-Canelo, Gibbons, & Rammsayer, 2006; Verleger et al. 2010; Wylie et al. 2009).

Although these instances of increased priming in PD have been obtained by measurements of motor output (overt responses, in

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some studies complemented by lateralized readiness potentials of the EEG) some researchers have argued that these changes in motor behavior are due to changes in the control of attention. Specifically, increased priming in PD has been suggested to reflect increased distractibility in PD (Machado et al., 2009; cf. Sharpe, 1990). If so, then PD patients will be oversensitive to mock-relevant stimuli even if these stimuli do not prime any specific response. On the other hand, our recent work led us to make a different suggestion. Based on our model of basal-ganglia functions and dysfunctions (Schroll, Vitay, & Hamker, 2014) Verleger, Schroll, and Hamker (2013) reanalyzed event-related EEG potentials (ERPs) from a flanker task (Verleger et al., 2010) and found that PD patients differed from healthy participants by displaying error-negativity-like potentials that occurred also with correct responses and even when flankers were compatible. Of particular interest to the present study were differences between groups in ERPs during error trials with incompatible flankers. Healthy participants' ERPs were characterized by a particularly early P3 component, indicating that participants had made an unambiguous though faulty early decision based on the flankers (which preceded the targets). This P3 was largely absent in PD patients such that their ERPs during error trials were characterized by the error negativity, which was distinct in the control group only when ERPs were averaged time-locked to the erroneous response rather than to the stimulus. These results suggested that errors tended to be due to unambiguously incorrect decisions in healthy participants but to states of indecision in PD patients. In the present study, we aimed at testing whether such indecision would also occur with respect to visual attention. Specifically, if being impaired in deciding on what is relevant, PD patients are expected to be less sensitive to mock-relevant stimuli. To explore this possibility, we used a "contingent-capture" paradigm, to be described in the following:

1.3. Contingent capture of visual attention

Traditionally, a distinction has been made between exogenous, reflexive and endogenous, voluntary modes of shifting visual attention (Posner, 1980; Müller & Rabbitt, 1989). Yet in their seminal study, Folk, Remington, and Johnston (1992) showed that visual stimuli with sudden onset, supposed to be the typical stimuli for attracting reflexive attention, do so only when being mock-relevant (our term), i.e., when sudden onset is a relevant feature in the task. Showing this to hold for color as a feature as well, Folk et al. suggested that the exogenous, reflexive mode of attention should be reconceived as a mode of "contingent capture": There is a critical difference in spatial cueing effects between signals that do and signals that do not match the targets in one of their features, with only those signals attracting attention that contain some task-relevant feature. In the context of the debate triggered by this finding, Lamy, Leber, and Egeth (2004) and Eimer et al. (2009) used a task that will be adapted here to study contingent capture in PD patients. Circular arrays of six bars in different colors are flashed in each trial (Fig. 1). Participants have to press the left or right key depending on whether the bar in target color is level or upright. Briefly before, an array of rings is flashed at the same positions as the bars, serving as uninformative priming stimulus for the bar array. Five rings are gray and one ring is colored (henceforth called "signal"), either in the target color, rendering the signal mock-relevant, or in another color. When the signal was in target color, targets were better and faster identified when presented at the place of the preceding signal, and less accurately and slower when presented at other positions, compared to signals in irrelevant color (Eimer et al., 2009; Lamy et al., 2004). Unlike the results mentioned in Section 1.2, this could not be due to responses being primed or inhibited, because the colored ring neither provides any information on target orientation nor primes any specific response. Rather, this occurred because the mock-relevant ring attracted attention. As described, this may be measured by changes in error rates and latencies of responses to the following target. Moreover, effects of mock-relevance were measured by Eimer et al. (2009) before any overt responses were made to the target, in event-related EEG activities at two different scalp sites. First, at contralateral visual cortex, a negative deflection was evoked, peaking around 250 ms after signal onset, interpreted as N2pc component that reflects attentional capture by relevant stimuli (Luck, Fan, & Hillyard, 1993; Hickey, Di Lollo, & McDonald, 2009; Pagano & Mazza, 2013; Wu et al. 2013). Second, at frontal midline, larger positivity was evoked by mock-relevant signals than by irrelevant ones, to be called "d-P200" in the following (difference in positivity evoked at 200 ms) and interpreted by Eimer et al. (2009) as indicating top-down inhibition of attentional capture realized by control mechanisms in frontal areas. In our present study, another effect of mock-relevance became evident: Signals in target color frequently evoked eye-movements. Being not reported by Eimer et al. (2009), and indeed largely absent in young adults tested as a pilot group to the present study (though see, e.g., Theeuwes, Kramer, Hahn, & Irwin, 1998), this effect was apparently related to our participants' old age.

1.4. Hypotheses

PD patients' oversensitivity to response priming by mock-relevant stimuli, as previously obtained, for example, in the flanker task, might be entirely constrained to the motor system, in situations when the motor plan is set and awaits a go signal. If so, PD patients will not differ from healthy participants in behavioral and ERP markers of contingent capture because mock-relevant signals do not prime any response in the present task (providing no information about whether the target bar is level or upright).

Alternatively, PD patients' oversensitivity to response priming might indicate some dysfunction that extends beyond their motor system. One possibility is that this oversensitivity indicates increased proneness to attentional capture, possibly due to attenuated inhibitory functions. If so, PD patients will show increased behavioral and EEG signs of contingent capture. Alternatively, oversensitivity to flankers in previous tasks may have indicated weakened ability for distinguishing the relevant features by which targets differ from flankers and, more generally, weakened ability for selecting relevant features. If so, PD patients will show decreased behavioral and EEG signs of contingent capture.

As listed in Section 1.3, indicators of contingent capture will be saccades, N2pc, and d-P200, all evoked by the signals, and priming effects of signals on accuracy and speed in responding to target bars. Accordingly, PD patients' changed sensitivities to mock-relevant signals should become apparent in increased or decreased rates of saccades to signals, larger or smaller signal-evoked N2pc and d-P200, and larger or smaller effects of signal location on accuracy and speed of key-press responses to the following target.

2. Material and methods

2.1. Participants

All participants provided their informed consent. The study was approved by the local Ethics Committee (file #11-182). No participant had impairment of color vision, as verified by appropriate execution of the Color-Word Interference Test (2.2). Participants of the control group were paid for their time. Short-sighted participants wore their glasses throughout the experiment. One PD patient and one control participant were left-handed, as evaluated by the Edinburgh Handedness Inventory (Oldfield, 1971).

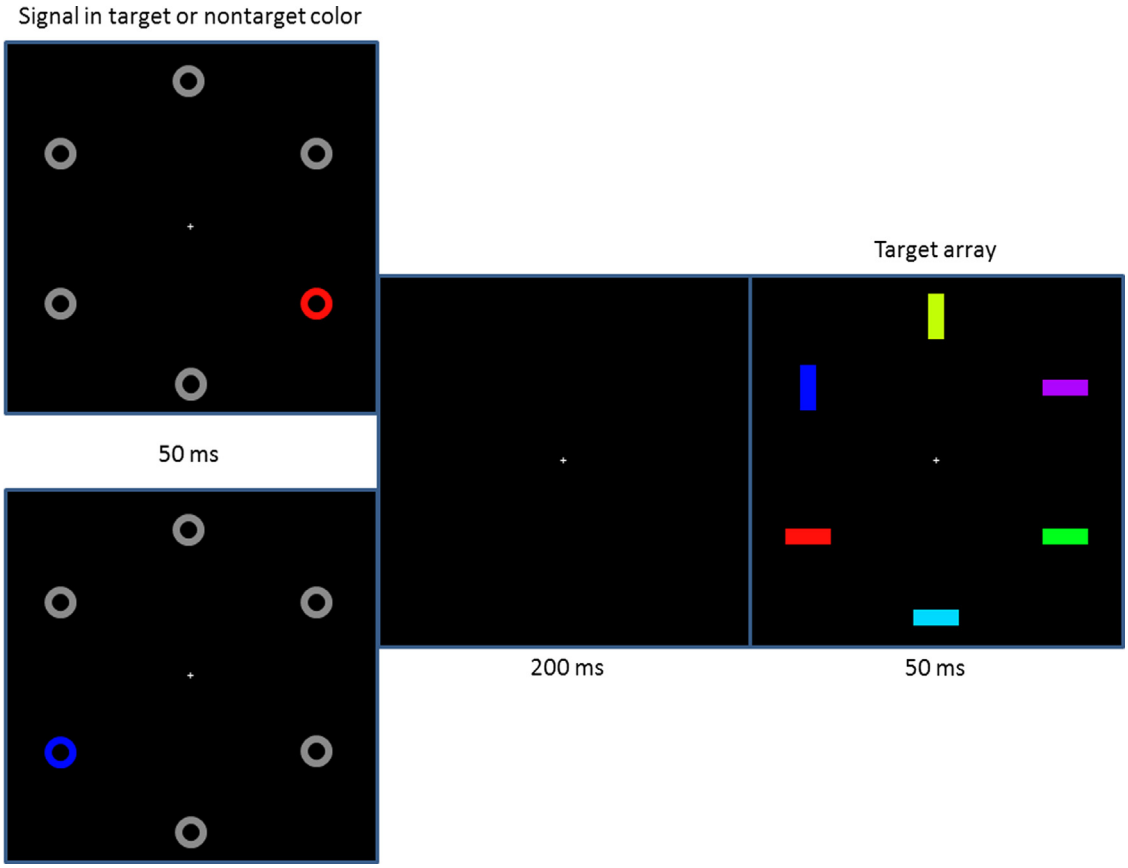

Fig. 1. Examples of the trial sequence. A signal array was presented for 50 ms and was followed, after a blank interval of 200 ms, by a target array (50 ms duration). Each bar in the target array had a different color. Participants had to press a left or right key according to the inclination of the target bar (level or upright). Target color was either red or blue, alternating between blocks. One of the rings in the signal array was colored (either red or blue, in random order across trials) and the others were gray. Target bars and colored signals always were at one of the four lateral positions, randomly determined. Thus, color signals were at the same positions as targets in 25% of trials. The figure is drawn to scale. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

2.1.1. *Participants with PD (n = 13)*

Patients were attending our outpatient clinic. Their individual data are compiled in Table 1. They were 7 men and 6 women, aged 61.5 years on average (range 44–76 yr). PD had been diagnosed 7 years ago on average (0.25–17 yr), scores on the modified Hoehn–Yahr scale (Goetz et al. 2004) were 2.4 on average, ranging between 2 (7 patients) and 3 (4 patients), and UPDRS III scores (Fahn & Elton, 1987) ranged between 7 and 24, mean 16.1 (± 5.6). All patients received dopaminergic medication, with an average daily L-dopa equivalence dose of 519 mg (± 337 mg; range 27–998 mg) and were tested at their best clinical "on". Testing for genetic mutations yielded negative results in the youngest patient. No positive family history of PD was known in any other patient. Another young patient (45 yr, HY score 1.5) was not included in the final sample because he felt hungry, tired, and impatient during the task such that he produced most errors of all participants, including many premature responses before target presentation, which no one else did.

2.1.2. *Healthy control group (n = 12)*

This group consisted of 7 men and 5 women, mean age 60.8 years (range 52–75 yr). According to self-report, none suffered from any illness affecting the central nervous system. Formal clinical examination was not performed.

Table 1
Characteristics of the PD patients.

Patient	Age (years)	Sex	Disease duration (years)	Hoehn and Yahr	UPDRS III	Medication (l-dopa equivalence, in mg/day)
1	67	m	2	2	12	130
2	71	w	10	3	12	997.5
3	64	m	0.25	3	23	655
4	68	w	7	2	24	27
5	56	m	9	2	9	889
6	54	w	4	3	21	200
8	76	m	3	3	23	665
9	50	w	11	2	19	815
10	63	m	2	2	19	210
11	60	m	7	2	17	715
12	72	w	17	2	13	420
13	44	m	2	2.5	10	100
14	54	w	10	2.5	7	925

2.2. *Neuropsychological tests*

To exclude participants with dementia and impaired color vision, and to measure distractibility by standard procedures, a short battery of neuropsychological tests was administered, including a vocabulary test for assessing crystallized intelligence (Lehrl, 1977; norms corrected according to Satzger, Fessmann, & Engel, 2002), the Stroop color-word interference test with its three tasks (reading, naming bar colors, naming colors of color words) for assessing mental speed, impairments of color vision, and distractibility (Bäumler, 1985), and the Auditory Verbal Learning Test for assessing fluid intelligence, excluding dementia, and assessing distractibility in the verbal domain (Helmstaedter, Lendt, & Lux 2001). As Table 2 shows, the two groups did not differ from each other, except for higher values for the control group's vocabulary, consistent with a somewhat higher level of schooling in this group, 42% having graduated from high-school (Abitur), compared to 15% in the patients.

2.3. *Stimuli and procedure*

Participants were seated in a comfortable armchair in a darkened chamber at a distance of about 1.15 m from a 17″ computer screen (1024 × 768 pixels) which was driven by the control computer at 100 Hz. Programmed by Presentation software (www.neurobs.com) this control computer presented the stimuli, recorded responses, and sent stimulus and response codes to the computer that recorded EEG.

Table 2
Results of neuropsychological testing.

Test	PD patients	Control group	t	p
MWT-B (vocabulary)	99 ± 9 (86–115)	109 ± 14 (93–130)	−2.3	0.03*
AVLT 1st trial (immediate span)	114 ± 13 (87–130)	115 ± 18 (74–130)	−0.2	0.88
AVLT, sum 1st–5th trial (learning)	107 ± 16 (76–128)	111 ± 16 (78–130)	−0.6	0.54
AVLT, 6th trial (immediate span with interference)	114 ± 14 (95–130)	111 ± 15 (86–130)	0.6	0.56
AVLT, 7th trial (memory, free recall)	98 ± 17 (68–124)	103 ± 16 (83–124)	−0.7	0.51
Speed of reading	107 ± 12 (93–130)	110 ± 12 (87–124)	−0.6	0.55
Speed of naming colors of bars	112 ± 14 (88–130)	114 ± 13 (91–130)	−0.3	0.76
Speed of naming colors of color words	109 ± 12 (88–126)	112 ± 10 (94–123)	−0.6	0.57

MWT-B=Mehrfachwahl-Wortschatztest B (multiple choice vocabulary test B), AVLT=Auditory Verbal Learning Test. Entries in the "PD patients" and "Control Group" columns are means ± std.dev. (minimum–maximum) of IQ-equivalent scores. These IQ scores are directly given by MWT-B (Lehrl, 1977; Satzger et al., 2002), and were transformed to the IQ scale (mean 100, std. dev. 15) from the means and std. dev. of the normative samples of the AVLT (Helmstädter et al., 2001) and of the color-word interference test (Bäumler, 1985; from mean 50, std. dev. 10). Outlying IQ-equivalent scores > 130 were set to 130.

* denotes significance at $p < .05$.

A white fixation cross (0.15° × 0.15°) was always visible at the center of the black screen. Target arrays consisted of six bars placed along the circumference of an imaginary circle at 2 h, 4 h, 6 h, 8 h, 10 h, and 12 h, with their midpoints 3.7° off screen center (Fig. 1). Each bar had its unique color: red, blue, green, yellow, turquoise, or purple. (CIE *x*/*y* 1931 coordinates computed from our RGB values by freeware programCIEWI:0.634/0.331;0.150/0.061;0.298/0.594;0.376/0.538;0.211/0.280;0.241/0.111). Positions of colors varied randomly across trials, with the restriction that the target color had to occur at one of the four lateral locations (2 h, 4 h, 8 h, and 10 h). Each bar (1.15° × 0.4°) could be level or upright, randomly varying across trials. Participants held a custom-made response box on their lap, with their hands resting on the box and each index finger situated on a button. They had to report whether the bar in target color was level or upright. Half of participants in each group pressed the left button for level and the right one for upright inclination. Assignment was reversed for the other half. Off-line, the time window for responding was constrained to 150–1800 ms after target onset. Target color was red or blue, alternating across blocks (24 cd/m^2 and 18 cd/m^2, measured by an LXcan luminance meter, Scanditronix Wellhöfer, Germany). Half of participants in each group started with red, the other half with blue.

Target arrays were preceded by priming arrays that consisted of six rings, with outer and inner diameters of 0.7° and 0.4°. Ring centers were located at the same positions as the following targets. Five rings were gray (CIE coordinates *x*/*y*0.280/0.312; RGB 140, 140, 140), and one was the colored signal. This colored ring was presented at one of the four lateral locations, randomly selected in each trial, and was either red or blue, in random order across trials. Thereby, depending on whether targets were red or blue in a given block, the signal was either mock-relevant (e.g., red signals in red-target blocks) or irrelevant (e.g., blue signals in red-target blocks).

Both arrays, of prime and target stimuli, were briefly flashed for 50 ms, separated by a blank 200 ms interstimulus interval. The priming array of the next trial appeared 1.8 s after onset of the preceding target array or, for response times ≤ 800 ms, 1 s after the response, such that trial duration was 2.05 s or less. Participants completed 7 experimental blocks, with self-timed pauses between blocks. As mentioned, target color alternated between blocks. The first block consisted of 200 trials, the following ones of 100 trials each, resulting in a total of 800 trials. Participants were asked to keep fixation throughout. Before the first block, participants practiced the task until the experimenter felt that they could master it. Durations of this practice phase varied between participants. Since no formal measurements of its duration were taken, it cannot be stated whether there were any differences between groups.

2.4. EEG recording and processing

EEG was recorded with Ag/AgCl electrodes affixed in an EEG cap (FMS, Munich) according to the 10–20 system from 26 scalp sites, which were 4 midline sites (Fz, FCz, Cz, Pz), 11 pairs of lateral sites (F7 & F8, F3 & F4, FC3 & FC4, T7 & T8, C3 & C4, CP5 & CP6, CP1 & CP2, P7 & P8, P3 & P4, PO7 & PO8, O1 & O2) and the nose-tip. On-line reference was Fz, ground was at the forehead, data were off-line re-referenced to the nose-tip. To control artifacts induced by eye movements and blinks and to monitor eye movements, vertical EOG was recorded from above vs. below the right eye and horizontal EOG from positions next to the outer rims of the eyes. Data were amplified within 0–1000 Hz by a BrainAmp MR plus and stored at 500 Hz per channel. Further processing was done off-line by means of BrainVision Analyzer 2.0.3 (Brain-Products, Gilching near Munich). Data were low-pass filtered at 20 Hz, with an additional notch filter at 50 Hz and were segmented from 100 ms before signal onset to 400 ms afterwards. Longer epochs were avoided in order to minimize the number of epochs to be rejected due to artifacts, particularly due to eye movements towards the target (cf. Eimer et al., 2009, for similar practice). Trials with incorrect responses to targets were not rejected because accuracy is not informative about how the signals had been processed: Particularly when signals and targets had different locations, incorrect responses could rather mean that signals were attended. For each segment, mean amplitudes of the epoch 100 ms–0 ms before the signal were subtracted as baseline. Trials were rejected as including artifacts when amplitudes exceeded ±80 μV in any channel including EOG channels. Because the signals evoked saccades to the left or right in a considerable number of trials (cf. Results), trials with horizontal saccades were not rejected but the transmission of horizontal EOG to EEG was corrected from the EEG by linear regression, using the Gratton method implemented in BrainVision. To obtain ERPs, each participant's data were then averaged for each of four categories: signals in relevant and irrelevant color, presented in the left or right half of the priming array. Minimum number of trials was 142, maximum was 207 (exceeding the expected number of 200 because of random variation), median was 183. ERPs evoked by left and right signals were recombined to be contra- and ipsilateral to signals, as detailed below.

2.5. Quantification and statistical analyses of task performance, saccades, and ERPs

Correctness and speed of responses to the targets were assessed. Error percentages were determined as numbers of incorrect, missing (> 1800 ms), or premature (< 150 ms) responses relative to all responses under a given condition. There were virtually no premature responses, except for the one excluded patient (Section 2.1.1). Error rates were arcsin-transformed, to approach normal distribution. Means of response times in correctly responded trials were computed for each condition. Conditions were defined by relative positions of signal and target, and by signal color. There were four position relations: Signal and target could be on the same side at the same height (i.e., same position) or at differing positions: same side but different height, opposite sides at same height, opposite sides at different heights. For brevity, analyses will be only reported from data pooled across these three different positions. Signal color could be relevant or irrelevant, depending on what was the target color in a given block.

The number of trials with saccades was recorded. Saccades were counted by using the commonly used criterion of 25 μV in the hEOG recording (e.g., Eimer et al., 2009) which roughly corresponds to 1.5° of visual angle. Thus, saccades towards the signal were counted when hEOG (left vs. right outer rim of the eye) exceeded +25 μV for left signals and −25 μV for right signals, and saccades away from the signal were counted when hEOG exceeded −25 μV for left signals and +25 μV for right signals. Both directions were counted independently when occurring in the same trial. The numbers of saccade trials toward and away from signals were expressed as percentages of all trials, separately for relevant and irrelevant signals (irrespective of whether the key-press response was correct and whether there were EEG artifacts).

In the averaged ERPs, N2pc and d-P200 were evaluated. To measure N2pc, difference waveforms contralateral minus ipsilateral to signals were formed for symmetric pairs of left-right electrodes, for example with PO7 and PO8: PO7–PO8 was computed for right-side signals, PO8–PO7 for left-side signals, and then these two differences were averaged. N2pc latencies and amplitudes were determined in these contralateral-ipsilateral differences of the pairs PO7 & PO8, P7 & P8, and P3 & P4 from the largest negative peak 150–300 ms after signal onset. This was done separately for relevant and irrelevant signals. d-P200 was measured as the mean difference between relevant and irrelevant signals 180–220 ms after signal onset at fronto-central recording sites, including midline sites Fz, FCz, Cz, and lateral sites F3, F4, FC3, FC4, C3, and C4. Measurements were made separately for left and right signals, rearranging left and right lateral sites to be contra- and ipsilateral to signal side. d-P200 was quantified as mean amplitude 180–220 ms after signal onset. Note that d-P200 corresponds to Eimer et al.'s (2009) anterior N2, which had negative polarity because differences were inversely formed, from irrelevant minus relevant signals, cf. Section 4.

Analyses of variance (ANOVAs) were used for statistical analyses. The factors will be listed in Section 3. To interpret interactions, ANOVAs were conducted separately for the levels of each of the interacting factors. Degrees of freedom were corrected with the Greenhouse–Geisser method when repeated-measurement factors had more than two levels.

3. Results

3.1. Signal-evoked saccades

Grand means of hEOG, contralateral minus ipsilateral to the signals, are displayed in the upper panel of Fig. 2. The rising waveform after signals in relevant color, starting 200 ms after signal onset, is mainly composed of the time-smeared average of occasional saccades toward signals. Percentages of saccade trials, depicted in the lower panel, were submitted to ANOVA with the factors Signal Color (mock-relevant or irrelevant), Saccade Direction (towards or away from signals), and Group (PD or healthy).

Fig. 2 suggests that PD patients tended to make more saccades than healthy participants. However, no effect involving Group approached significance, $F_{1,23}$ or $F_{2,46} \leq 0.5$, $p \geq 0.50$. Evidently, across both groups, more saccades occurred towards than away from signals, $F_{1,23}=20.6$, $p<0.001$, and with mock relevant than with irrelevant color, $F_{1,23}=28.4$, $p<0.001$. In particular, most saccades occurred toward signals in relevant color, Saccade Direction × Signal Color $F_{1,23}=26.6$, $p<0.001$.

3.2. Signal-evoked EEG lateralization: N2pc

Grand means of signal-evoked contralateral-ipsilateral differences at the PO7–PO8 pair of sites are displayed in Fig. 3. The large negative peak at about 225 ms after onset of signals in relevant color is the N2pc. Amplitudes and latencies of the N2pc peak were entered to ANOVAs with the factors Pair of Recording Sites (which had three levels: PO7–PO8, P7–P8, and P3–P4), Signal Color, and Group (as defined above).

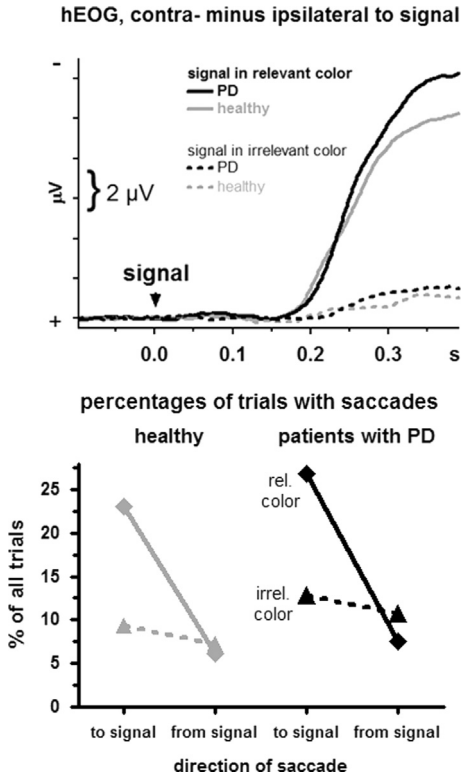

Fig. 2. Signal-evoked saccades. The upper panel displays grand averages of contralateral-ipsilateral difference hEOG potentials from either group (temporally smeared due to averaging across trials), from 100 ms before to 400 ms after the signals. The lower panel displays mean percentages of trials where saccades occurred in the first 400 ms (defined as exceeding ±25 µV in hEOG), both when directed towards the signal and away from it (*x*-axis). In both panels, black color denotes the PD group, gray color the control group. Solid lines (and, in the lower panel, diamonds) are for signals in relevant color, dashed lines (and triangles) for signals in irrelevant color.

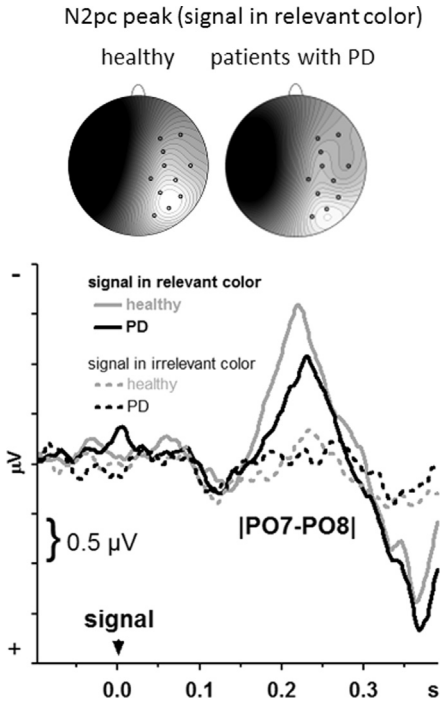

Fig. 3. Signal-evoked N2pc, measured in the contralateral-ipsilateral difference potentials. Displayed are grand-average data across either group, recorded from the PO7–PO8 pair from 100 ms before to 400 ms after the signals. Gray: healthy; black: PD patients; solid lines: signals in relevant color; dashed lines: signals in irrelevant color. At the top of the figure, topographic distributions are displayed at the time of the N2pc peak. These differences are illustrated as right-left differences as if originating from activity of the right side only. Either map is scaled from its negative minimum (white) to its inverse positive value (black), ±1.6 µV for the healthy group and ±1.1 µV for the PD group.

3.2.1. N2pc amplitudes

N2pc was much larger with mock-relevant than irrelevant signals, $F_{1,23}=58.4$, $p<0.001$, without differences between the three electrode pairs ($F_{2,46}<2.3$, $p \geq 0.12$). PD patients had smaller amplitudes than the healthy group with mock-relevant signals, Signal Color × Group $F_{1,23}=6.5$, $p=0.02$; effect of group separately for relevant color $F_{1,23}=4.2$, $p=0.05$; for irrelevant color $F_{1,23}=0.0$, n.s.

3.2.2. N2pc latencies

No effect, including effects of Group, became significant, $F \leq 2.2$, $p \geq 0.13$. When restricting analysis to signals in relevant color because latencies might not be reliably estimated with irrelevant color, Pair of Recording Sites and Group interacted, $F_{2,46}=4.6$, $p=0.02$, because latencies were later at PO7/8 than at P7/8 and P3/4 in PD patients, $F_{2,24}=4.0$, $p=0.03$, but did not differ between recording sites in the healthy group, $F_{2,22}=1.1$, n.s.

3.3. Signal-evoked fronto-central d-P200

Grand means of signal-evoked potentials evoked at FCz are displayed in Fig. 4. Around 200 ms, waveforms were more positive with mock-relevant than irrelevant signals. To quantify this "d-P200", ANOVAs were computed on mean amplitudes 180–220 ms in the difference between mock-relevant and irrelevant signals recorded from anterior sites (F, FC, and C rows) at midline and lateral, left and right sites (like Eimer et al., 2009). The lateral sites were rearranged to become contralateral and ipsilateral to

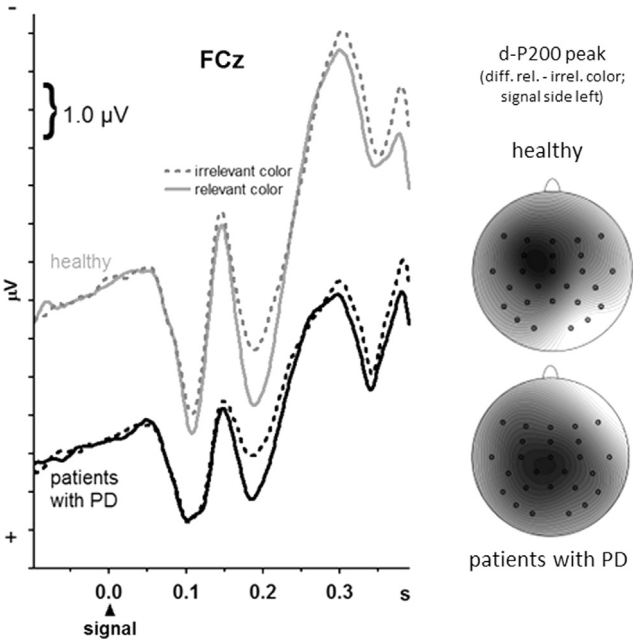

Fig. 4. Signal-evoked d-P200. Displayed are grand averages of potentials evoked by the signal, separately for the two groups. In the left panel, FCz recordings are shown in solid and dashed lines for signals in relevant and irrelevant color. Gray: healthy; black: PD patients. On the right, topographic distributions of the relevant-irrelevant differences are displayed at the peak of this difference ("d-P200"). Either map is scaled from −1.25 μV (white) to +1.5 μV (black). To produce the maps, data were pooled across left and right signals, with recording sites mirrored for right signals, such that the right hemisphere is always contralateral to signals.

signal side (unlike Eimer et al., 2009) because d-P200 appeared to be smallest contralateral to signals (Fig. 4). ANOVA factors were Front-Rear (F, FC, C), Laterality (midline, contralateral, ipsilateral to signal) and Group.

d-P200 was reliably greater than zero, as evident by the significant constant term, $F_{1,23}=16.3$, $p<0.001$. Front-Rear topography interacted with Group, $F_{2,46}=7.0$, $p=0.006$, because, as suggested by the topographical maps in Fig. 4, d-P200 was larger at frontal and fronto-central than at central sites in the healthy group (Front-Rear for the healthy group: $F_{2,22}=4.2$, $p=0.04$) in contrast to PD patients whose topography, if anything, displayed the reversed tendency ($F_{2,24}=3.2$, $p=0.09$). (The interaction could not be resolved to group effects at the single F, FC, or C rows, though, $F_{1,23}\leq 2.2$, $p\geq 0.15$). Independent of group, d-P200 was smaller at contralateral than at midline and ipsilateral sites (Laterality: $F_{2,46}=17.6$, $p<0.001$), most so at central and least so (though still significant) at frontal sites, Laterality × Front-Rear $F_{2,46}=7.2$, $p<0.001$.

3.4. Error rates and response times

Since target arrays were flashed for 50 ms only, the task was difficult. Fig. 5 displays individual participants' error rates (misses and wrong responses) pooled across conditions.

Mean error rates and response times, displayed in the upper panel of Fig. 6, were each analyzed by an ANOVA with the factors Signal Color and Group, as above, and additionally Target Position (at or off signal position). There were no differences between groups for error rates (all effects involving Group $F_{1,23}\leq 1.6$, $p\geq 0.22$) and only a tendency for response times, $F_{1,23}\leq 3.1$, $p\geq 0.09$, but see below for the detailed analysis of initial, middle, and end phases of the task. Across both groups, mock-relevant signal color reduced error rates and speeded responses when targets followed at signal position, and boosted error rates and delayed responses when targets followed at other positions, reflected by interactions of Signal Color and Target

Fig. 5. Individual rates of trials with wrong and missing responses relative to all trials, pooled across all conditions.

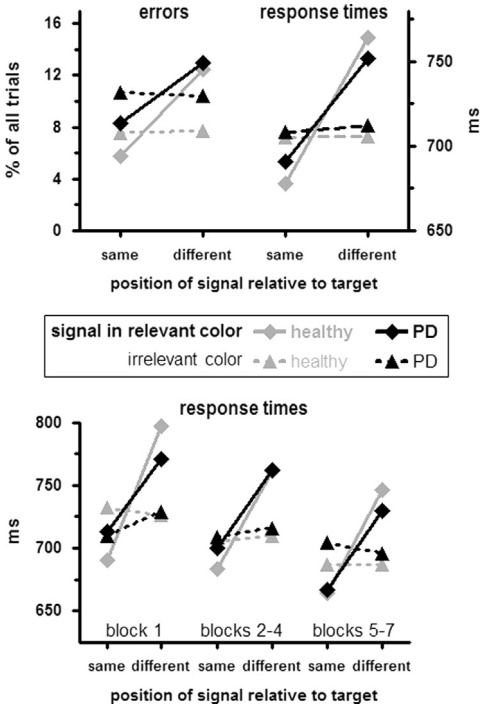

Fig. 6. Mean values of behavioral measures. Error rates and response times are depicted in the upper panel, pooled across blocks, and response times are depicted in the lower panel for the first 200 trials, the middle 300 trials and the final 300 trials. In both panels, black color denotes the PD group, and gray color the control group. Solid lines (and diamonds) are for signals in relevant color, dashed lines (and triangles) for signals in irrelevant color.

Position, $F_{1,23}=21.0$, $p<0.001$ for error rates, and $F_{1,23}=78.5$, $p<0.001$ for response times. Indeed, facilitation at signal position and interference at other positions both were significant, indicated by effects of Signal Color separately for targets at signal position, $F_{1,23}=8.4$, $p=0.008$ for error rates, $F_{1,23}=9.9$, $p=0.005$ for response times, and for targets off signal position, $F_{1,23}=16.1$, $p=0.001$ for error rates, $F_{1,23}=94.2$, $p<0.001$ for response times. Besides, there were main effects of Target Position on error rates, $F_{1,23}=21.7$, $p<0.001$, and on response times, $F_{1,23}=61.1$, $p<0.001$, and effects of Signal Color on response times, $F_{1,23}=8.8$, $p=0.007$, but these main effects were qualified by their described interactions.

As noted, the largest tendency ($p=0.09$) for an effect of Group occurred on response times, indicating a non-significant interaction of Signal Color and Group. Because this issue of absent effects on behavior in spite of existing effects on ERP amplitudes appeared paradoxical and needed further scrutiny, RTs were analyzed

separately for the first block (200 trials), the middle blocks (300 trials), and the final blocks (300 trials). Indeed, in the first block, the distinction between relevant and irrelevant signals was more marked in the healthy group than in the patients, indicated by the interaction of Signal Color, Target Position, and Group, $F_{1,23}=5.3$, $p=0.03$, reflecting both larger effects of signals in relevant color on the healthy group (facilitating and delaying) and some facilitating effects of signals in irrelevant color on PD patients when targets were at signal position. There were no differences between groups in the middle blocks. But in the final three blocks, healthy participants' responses after irrelevant-color signals were on average (across same-side and different-side signals) faster than after relevant-color signals, Signal Color × Group $F_{1,23}=4.5$, $p=0.046$, effect of Signal Color in the healthy group $F_{1,11}=8.0$, $p=0.02$, probably reflecting that healthy participants had made further progress in discarding irrelevant-color signals altogether. This did not occur in PD patients (effect of Signal Color in PD patients in the final three blocks $F_{1,12}=0.1$, n.s.). In sum, this development of differences with time-on-task suggests that PD patients needed more time to distinguish between mock-relevant and irrelevant signals with the same precision as healthy participants.

3.5. Effects of saccades on error rates

In order to investigate possible detrimental effects of signal-induced saccades on behavior, error rates were broken down according to whether a saccade had or had not occurred in the first 400 ms after signal onset. Since some participants made very few saccades in some conditions, data were pooled across signal sides, target positions, and saccade directions (toward or away from signals). Mean values of error percentages split by presence versus absence of saccades are displayed in Fig. 7. Data were entered to an ANOVA with the factors Saccade Presence (yes, no), and Signal Color and Group (as above). Error rates were higher in trials where saccades were evoked by the signal (Saccade Presence: $F_{1,23}=4.4$, $p=0.046$) but this tended to be true in the healthy group only (Saccade Presence × Group $F_{1,23}=3.5$, $p=0.07$; effect of Saccade Presence in the healthy group $F_{1,11}=5.8$, $p=0.03$) whereas error rates were uniformly high in the patients (effect of Saccade Presence $F_{1,12}=1.5$, n.s.).

4. Discussion

Several studies reported increased priming effects on PD patients' responses by "mock-relevant" stimuli, i.e., irrelevant stimuli that contain relevant features. We tested whether this increased sensitivity is restricted to the motor system or affects PD patients' attention as well. In particular, we wondered whether mock-relevant stimuli attract attention more in PD patients than in healthy persons, which would confirm the notion of increased distractibility of PD patients, or whether these stimuli attract attention less in PD patients than in healthy persons, which would be in line with the notion of poorer internal representations of relevance in PD patients. To study this issue, task-irrelevant shapes in task-relevant color were flashed briefly before the target stimuli. Indeed, effects of these mock-relevant signals differed between PD patients and healthy participants in two aspects. One aspect refers to PD patients' EEG activations evoked by the signals. Their N2pc evoked by mock-relevant signals (contralateral negativity at the scalp over visual cortex) was smaller, and their d-P200 (difference between mock-relevant and irrelevant signals) was focused at central recording sites, in contrast to the healthy group's focus at frontal sites. The other aspect refers to PD patients' responses to the following targets. In the first phase of the task, healthy participants' response times to target stimuli were more affected by preceding mock-relevant signals than PD patients' responses. In the final three blocks of the task, the relationship had changed in healthy participants between effects of relevant-color and irrelevant-color signals because healthy participants apparently had learned to ignore signals in irrelevant color altogether whereas PD patients had not. Moreover, different from healthy participants, PD patients' error rates did not depend on whether their gaze had shifted to the preceding signals. Thus, by all these parameters, PD patients appeared less specifically distracted by mock-relevant signals. These results will be discussed in more detail in the following:

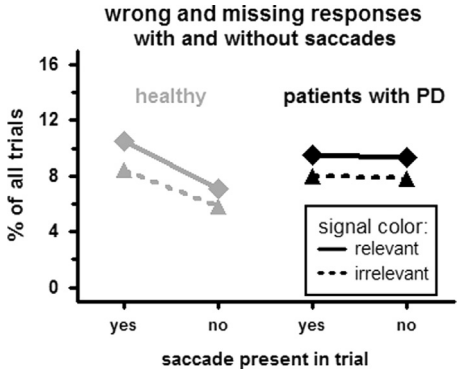

Fig. 7. Mean percentages of error trials, separately for trials with and without saccades in the first 400 ms. Trials were pooled across blocks, signal sides, and saccade directions. Error percentages are shown for signals in relevant color (solid lines) and irrelevant color (dashed lines), separately for trials with and without saccades ("yes" and "no" on *x*-axis).

4.1. Decreased N2pc in PD patients

N2pc reflects the differential response of the contralateral visual cortex to relevance of laterally presented stimuli. To our knowledge, N2pc has been compared between PD patients and healthy persons in only one previous study so far (Praamstra & Plat, 2001) where N2pc amplitudes were equally large in both groups. One reason for this difference from the present results may be that the N2pc-evoking stimuli were the only stimuli presented in that study, requiring immediate responses according to their identity, making the requirement of distinguishing between stimuli less complex than in the present study where the N2pc-evoking signals were mock-relevant only. More generally, our task was more difficult, with PD patients committing 12% errors on average, compared to 5% in Praamstra and Plat (2001). Thus, a certain amount of task load may be needed to reveal subtle reductions in the ability to focus attention. (Cf. Verleger, Talamo, Simmer, Śmigasiewicz, & Lencer 2013, for pertinent evidence from patients with schizophrenia and bipolar disorder. N2pc potentials were evoked by targets embedded among rapidly and bilaterally presented streams of distractors. These potentials were smaller in patients than in healthy control participants, which was in contrast to previous studies where easier tasks were used). In any case, PD patients' lowered N2pc in the present task suggests that the color feature of mock-relevant signals attracted less attention in the patients than in the healthy participants.

4.2. Less distinct frontal d-P200 in PD patients

The d-P200 effect, i.e., more positivity with mock-relevant than irrelevant signals at 200 ms at fronto-central sites, has been reliably obtained in this paradigm, both in the two experiments reported by Eimer et al. (2009) and in the present study. Eimer et al. (2009) conceived of d-P200 as increased negativity evoked by irrelevant relative to mock-relevant signals and interpreted it as indicating

inhibition of contingent capture of attention with irrelevant signals. But d-P200 may be as well interpreted as increased positivity after mock-relevant signals, rather than increased negativity after irrelevant ones. First, even if d-P200 indicates inhibition, such inhibition might be required more critically when dealing with mock-relevant than with irrelevant signals. This might be necessary in order to prevent attention from shifting to signal location because signal location formed an invalid cue for target location, correct in 25% of trials only. Positive potentials with similar latencies early after stimulus onset have indeed been described as indicating inhibitory processes (Eimer & Schlaghecken, 1998; Fortier-Gauthier, Moffat, Dell'Acqua, McDonald, & Jolicœur, 2012; Jaśkowski, Białuńska, Tomanek, & Verleger, 2008; Sawaki, Geng, & Luck, 2012). But none of those positive potentials had its topographical focus at anterior midline. Therefore, another interpretation of d-P200 is nearby: Rather than indicating inhibition, this positive potential might reflect activation evoked by mock-relevant signals, complementary to N2pc. In fact, such pairs of early negative and positive responses to attended features have been frequently described with centrally (rather than laterally) presented stimuli, as posterior "selection negativity" (corresponding to N2pc evoked by lateral stimuli) and anterior "selection positivity" (Anllo-Vento, Luck, & Hillyard, 1998; Kenemans, Kok, & Smulders, 1993; Smid, Böcker, van Touw, Mulder, & Brunia, 1996). Therefore, d-P200 is probably an instance of selection positivity, indicating activation evoked by the relevant feature.

Interestingly, PD patients lacked the frontal focus of this d-P200. This may be interpreted as reflecting some dysfunction of frontal cortex in PD patients. However, combined information from Figs. 3 and 4 suggests a more mundane interpretation of this difference. As noted in Results, d-P200 was less positive at sites contralateral to the signal, more so at central than at frontal sites, i.e., least positive at central contralateral sites. This decreased positivity might be due to overlap with increased negativity coming from posterior sites. Fig. 3 shows that N2pc began to rise at 180 ms, when d-P200 was measured, and that this N2pc reached larger amplitudes in the healthy group than in PD patients at this time-point. Accordingly, the topographic maps in Fig. 4 show that the negativity contralateral to signals at posterior sites was larger in the healthy group than in the patients. Spread of this posterior negativity towards anterior sites might have overlapped the healthy group's d-P200 at central sites, producing the decrease of d-P200 contralateral to signals and shifting the focus of their d-P200 toward frontal sites. By this interpretation, PD patients' more posterior topographical focus of d-P200 does not reflect frontal dysfunction but rather is another reflection of their reduced N2pc which does not overlap their d-P200 to the same extent as in the healthy group, leaving d-P200's focus at central sites.

4.3. Effects on behavior

Three of the 13 PD patients made more errors than the poorest performing healthy participant (Fig. 5) but, as a group, PD patients did not differ in error rates from healthy participants. Remarkably, PD patients' errors in responding to targets did not depend on presence or absence of eye movements to the preceding signals, in contrast to healthy participants who committed more errors when the preceding signal had triggered an eye movement. Thus, misguided overt attention had a smaller share in producing errors in PD patients than in healthy participants, implying that a larger proportion of PD patients' errors had other causes than externally misguided focused attention. This fits the notion of differences between PD patients and healthy controls in their proportions of externally misguided and internally produced errors (cf. Section 1.2; Verleger et al., 2013). A major cause of PD patients' errors in the present task may be that their attention was misguided due to less distinct internal representations of task-relevant features. Thus, we conjecture that the benefits of good representations of target features came with a cost in this task and that the costs of poor representation provided some benefits: Participants who had good representations (i.e., ideally the healthy group) became more distracted by the mock-relevant signals. Participants who had poor representations (i.e., ideally the PD patients) made more errors due to these poor representations but were less distracted by the mock-relevant signals.

Latencies of correct responses were strongly affected by the preceding signals in relevant color, replicating earlier studies in healthy young participants (Eimer et al., 2009; Lamy et al., 2004). In the global analysis on all trials, effects of Signal Color on response times only tended to differ between PD patients and the control group ($p=0.09$). However, when beginning, middle, and end of the task were separately analyzed, group differences emerged. In the beginning, mock-relevant Signal Color had stronger effects in the healthy group than in PD patients, speeding their responses more when targets were presented at signal location and otherwise delaying their responses more than in PD patients. The groups did not differ from each other in the middle phase, apparently because effects of mock-relevance had become smaller in the healthy group, being reduced to the level held by PD patients from task onset onwards. In the final three blocks, healthy participants' responses were faster after irrelevant-color than after relevant-color signals, probably indicating that healthy participants had made further progress in discarding irrelevant-color signals altogether. This did not occur in PD patients. In sum, this development of differences with time-on-task suggests that PD patients needed more time to distinguish between mock-relevant and irrelevant signals with the same precision as healthy participants, again compatible with the notion of less distinct internal representations of task-relevant features.

4.4. Summary and relation to pathophysiology

In sum, PD patients appeared less distracted by mock-relevant signals than healthy controls. This may be concluded from their smaller signal-evoked N2pc, from the tendencies of their response times to be less modulated by the location of mock-relevant signals, and from the independence of their error rates on whether their gaze had shifted to the signals. These effects are compatible with the assumption stated in Section 1 that PD patients have less distinct internal representations of task-relevant features, pointing to deficits in task-control processes. The absent frontal focus of PD patients' signal-evoked d-P200 might be in line with this assumption, but it appears safer to assume that this shifted topographical focus was due to decreased overlap of PD patients' smaller N2pc, thereby being another reflection of reduced attentional capture.

These subtle deficits in visual attention may be similar to earlier electrophysiological results obtained when PD patients had to focus on one of two sources of sounds, with channel selectivity reflected by the difference potential between attended and unattended auditory channels ("processing negativity"). This difference was reduced in patients with PD (Stam et al., 1993; Vieregge, Verleger, Wascher, Stüven, & Kömpf, 1994).

Computational and non-computational models have stated hypotheses on the exact nature of basal-ganglia dysfunctions in PD (e.g., Albin, Young, & Penney, 1989; DeLong, 1990; Frank, 2005; Schroll et al., 2014). These models mostly aim at explaining the disorder's prominent motor impairments, thus neglecting cognitive symptoms. They assume dopamine loss in PD to result both in reduced output of the motor-facilitating direct pathway of basal ganglia and in increased output of motor-inhibiting indirect pathways (Kravitz et al. 2010), thus accounting for reduced motor output in PD patients (review: Schroll & Hamker, 2013). In light of

findings that motor and cognitive areas of basal ganglia have largely equivalent pathway architectures (Alexander, DeLong, & Strick, 1986; Alexander & Crutcher, 1990) and are both affected by dopamine loss in PD (Pavese, Rivero-Bosch, Lewis, Whone, & Brooks, 2011), it appears likely that cognitive areas of basal ganglia are affected by analogous pathway dysfunctions: Facilitation of cognitive schemes and processes via the direct pathway might be reduced, while their inhibition via the indirect pathway might be enhanced. Thus, pathway dysfunctions might not only account for PD patients' motor dysfunctions, but also for their empirically observed difficulties in activating new cognitive schemes and processes (Lees & Smith, 1983; Cools et al., 2001). In relation to our results, therefore, pathway dysfunctions may be the origin of PD patients' less active cognitive representations of currently relevant target colors, leading to their reduced attention on relevant target-color dimensions.

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