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Abnormal gaze strategies during problem solving in Parkinson's disease

T.L. Hodgson *, B. Tiesman, A.M. Owen, C. Kennard

Division of Neuroscience and Psychological Medicine, Imperial College School of Medicine, Charing Cross Hospital Campus, 10th Floor East Wing, St Dunstans Road, London W 6 8RP, UK

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Abstract

We have taken a novel approach to the study of problem solving involving the detailed analysis of natural scanning eye movements during the 'one touch' Tower of London task. Control subjects and patients with idiopathic Parkinson's disease (PDs) viewed a series of pictures depicting two arrangements of coloured balls in pockets within the upper and lower halves of a computer display. The task was to plan (but not execute) the shortest movement sequence required to rearrange the balls in one half of the display (the Workspace) to match the arrangement in the opposite half (the Goalspace) and indicate the number of moves required for problem solution. As problem complexity increased, control subjects spent proportionally more time fixating the Workspace region. This pattern is found regardless of whether subjects were instructed to solve problems by rearranging balls in the lower or upper visual fields. The distribution of gaze within the Workspace was also found to be problem dependent, with gaze being selectively directed towards the problem critical balls. In contrast, PDs were found to make more errors in the task and failed to show any dissociation in the amount of time fixating the two halves of the display. This pattern suggests that the patients had difficulty in encoding and/or maintaining current goals during problem solving, consistent with a role for fronto–striatal circuits in mechanisms of working memory and attention.

Keywords: Eye movements; Executive; Basal ganglia; Cognitive; Planning; Dopamine

1. Introduction

It is well established that eye movements can be influenced by psychological processes and that complex visual tasks use specialised gaze shifting strategies [10,11,24,26,33,50,61]. Detailed analysis of these strategies can provide a richer description of task performance than is afforded by gross measures such as reaction times and error rates. By focussing on this active component of cognition, researchers have been able to shed light on the internal cognitive processes subserving performance of a number of behaviours [6,12,26,27,31,34].

We have recently taken this approach by measuring eye movements while subjects plan solutions to Tower of London (TOL) problems [27]. This task was developed with the aim of testing the subtle deficits in behaviour, which are observed following frontal lobe damage in man [55], but it has also proved sensitive to a number of other neurological conditions including Parkinson's disease [38] and schizophrenia [39]. The test involves the presentation of two different arrangements of coloured discs or balls (Fig. 1). The subject's task is to rearrange the first array of balls (referred to in this paper as the 'Workspace') so that it matches the second array of balls (referred to here as the 'Goalspace') using the minimum number of moves possible. The positioning of the balls is constrained to the location of three pegs or pockets in each half of the display. Due to this, complex problems demand that the sequence of moves is carefully planned in advance before attempting the first move. Failure to engage in advanced planning of the sequence will result in initial moves blocking subsequent ball moves.

* Corresponding author. Tel.: +44-20-8846-7712; fax: +44-20-8846-7715.

E-mail address: t.hodgson@ic.ac.uk (T.L. Hodgson).

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Owen et al. [49] have devised a 'one-touch' version of the TOL in which the incentive for individuals to plan solutions internally is enhanced still further. In this variant of the task, subjects are required to inspect the problems visually and then make a single motor response to indicate how many moves would be required to reach an ideal solution. In this way, the one-touch task isolates the cognitive planning component of the test by demanding the internal planning of solutions without actually executing the appropriate moves. Earlier studies have established formally identical patterns of impairment on the one-touch task and classical TOL tasks in patients with frontal-lobe damage and in patients with Parkinson's disease [45,47,49] even when the same patients are performing the two tasks [42].

Our studies in control subjects have shown that during the solution of relatively simple 3 move problems several discrete phases are observed in ocular scanning during the one-touch TOL task. These correspond to an initial problem assessment during which gaze is equally distributed between the Goalspace and Workspace, followed by a solution elaboration phase when subjects bias their gaze towards the Workspace

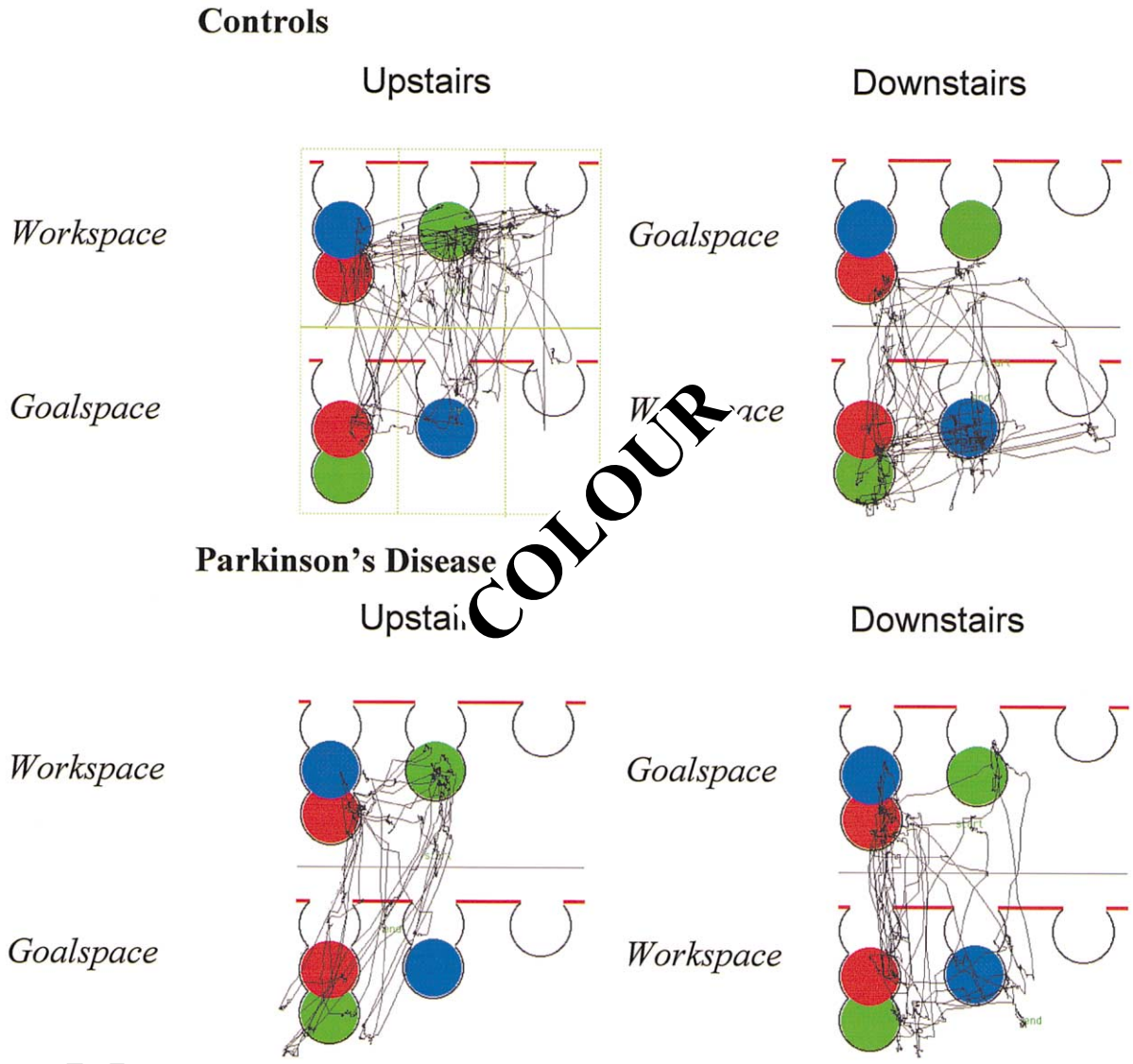

Fig. 1. Example X–Y plots for different subjects planning solutions to the same five move Tower of London problem. Half the subjects were instructed to solve problems in the 'Downstairs' manner by rearranging the balls in the upper visual field to match the lower. The other group of subjects solved problems in the converse 'Upstairs' manner. Although all subjects were presented with an identical set of problem pictures, the eye movements made by the control subjects differed systematically dependent upon instruction set, with gaze being strongly biased towards the Workspace during solution planning. In contrast, Parkinson's disease patients failed to show this pattern regardless of whether a correct or incorrect response was given. Fixations were classified offline according to where they landed in a 3×2 analysis grid (shown top-left corner) superimposed over each problem picture.

region. As problem complexity increases, the total time spent fixating the Workspace region increases strongly with the total number of moves required for problem solution. Fixations on the Workspace are also distributed in a problem dependent manner, such that gaze is selectively biased towards balls relevant to the problem in hand. One upshot of these observations is that the relative time spent fixating the Workspace provides a useful measure of the time spent planning or elaborating problem solutions as opposed to assessing problem goals.

One group of patients who are impaired at performance of the TOL task are those with Idiopathic Parkinson's disease (PD). These individuals suffer a depletion of the neurotransmitter dopamine within the striatum of the basal ganglia, which becomes progressively more severe as the disease takes its course. Although the symptoms of the disease are predominantly motor in nature, it has become apparent in recent years that even in the early stages, subtle cognitive impairments may be observed (e.g. [8,17,29,49,53,57]). These are most likely to arise from the disruption to the reciprocal loops between the striatum and structures in the prefrontal cerebral cortex [1,32]. However, cognitive deficits may also result from dopamine depletion within the frontal cortex itself, due to degeneration of the meso-limbic dopaminergic system [9,43]. A steady degradation in performance of patients in the TOL task has been observed as the disease takes its course. In the early stages, the time taken for patients to plan problem solutions may be significantly increased. In more severely affected patients, PDs make an increased number of response errors in both the standard and one-touch version of the task [47,49].

Here we describe how Parkinson's disease patients move their point of gaze whilst attempting to solve TOL problems. By examining the relative time they spend looking at different components of the problems, we hope to understand better what aspects of planning are impaired in PD. For example, it is possible that PDs possess essentially normal processes of problem solving, but have slowed motor imagery (bradyphrenia) [17], leading to an increase in the time taken to solve problems. This would be expected to exert a disproportional effect on the solution elaboration phase of planning, reflected by an increase in the amount of time fixating the Workspace. In contrast, inefficient problem solving might also arise from an inability to identify and maintain relevant goal information (goal-processing deficit). If the goal-processing hypothesis were correct, then we would expect patients to spend an increased amount of time fixating the Goalspace relative to the Workspace region of the display.

2. Methods

2.1. Subjects

All of the participants in the study gave their informed consent and the local ethics committee approved the work. None of the subjects had encountered the TOL task earlier.

2.1.1. Control group

Eight age matched control subjects were tested. The mean age of the control group was 68.4 years with a range of 61–75 years. All were neurologically normal and had normal or corrected to normal vision. Eye movement data for one control subject failed to record due to lack of disk space and, therefore, could not be analysed further.

2.1.2. Patient group

Ten subjects with idiopathic Parkinson's disease were tested, with five subjects allocated to each instruction condition. Mean age of the patient group was 61 years, with a range of 53–71. None of the PDs showed any signs of dementia as assessed by the mini-mental state questionnaire. The severity of their Parkinson's disease was assessed using the abbreviated Webster Scale [60]. The Webster scale is essentially a sub-set of the United Parkinson's Disease Rating Scale (UPDRS) [20], which focuses on the clinical motor signs of PD. It provides a more detailed and objective assessment than the more commonly used Hoen and Yahr scale [30], but is quicker to administer than the full UPDRS. This allowed us to perform patient assessment at the same session as the planning/eye movement test. The duration of the disease and relevant medication was also noted (Table 1).

2.2. Materials/stimuli

Each subject viewed 20 pictures showing two arrays of three coloured balls positioned in pockets. Pictures subtended 17 by 20 degrees of visual arc and were displayed using a Macintosh 2ci computer with a 17-in. colour computer monitor. Balls were coloured either red, green or blue. The two arrays of balls were located in the upper and lower visual fields. The left most location in each array could contain a maximum of three balls, the middle location contained a maximum of two balls and the right location had space for only one ball. The arrangement of balls in the lower field was always fixed, whilst the position of balls in the upper field varied from trial to trial (Fig. 1). At the start of each trial a central fixation cross was displayed for 500 ms. For calibration purposes, the subject was asked to look at this cross,

Table 1
Details of individual Parkinson's disease patients who participated in the study

Patient	Age	Webster score	Disease duration (years)	Medication
JA	53	10	6	Seleginine, Sinemet CR
JR	56	12	4	Unmedicated
EL	66	12	4	Madapor, Pergolide
DR	71	13	5	Madopor, Seleginine
PS	63	6	4	Sinemet, Pergolide
RH	55	6	4	Sinemet CR
BH	65	12	3	Sinemet CR
CB	70	5	5	Sinemet
EB	55	10	4	Sinemet
CH	56	5	10	Madapor

which was extinguished simultaneously with the presentation of problem pictures.

2.3. Procedure

Subjects were instructed to plan but not execute the problem solutions (i.e. the 'one touch' TOL task). Once the subject thought that they had worked out the correct solution to each problem they pressed the mouse key and gave a verbal response to indicate the minimum number of moves required to solve the problem. Errors trials were those for which the subject indicated the wrong number of moves. Prior to the start of the experiment, participants were given written instructions concerning the task indicating how the balls could be moved from one pocket to another and could not be placed directly underneath another ball without moving obstructing balls to an alternative location. They were told to plan the entire sequence of moves 'in their heads' before giving their response. The experimenter asked each subject to show them how they would solve a simple example problem in order to confirm that they had understood the task correctly. Both groups of subjects were then given two blocks of practice using a simpler two-ball task [27] prior to the experimental block.

2.4. Instructions

Both patients and controls were randomly allocated into two equal groups which received different instructions on how to solve the task. 'Upstairs' subjects were told to plan the sequence of moves required to rearrange the balls in the top part of the display to match the bottom half (i.e. Workspace in the upper visual field, Goalspace in the lower visual field). In contrast, the 'Downstairs' group were required to rearrange the balls in the bottom half of the display to match the top half (i.e. Workspace in the lower visual field, Goalspace in the upper visual field) (Table 1).

2.5. Eye tracking and analysis

Eye movements were recorded using the EyeLink system (Sensorimotoric systems GMbH), a video based pupil tracker, with head movement compensation system sampling at 250 Hz. Subjects were seated at a comfortable viewing distance in front of the display monitor approximately 60 cm from the computer screen. Pupil position was monitored via two miniature infra-red CCD video cameras mounted on an adjustable head-band. Subjects were instructed to keep head movements to a minimum and no active restraint of head movements was required to obtain sufficiently accurate gaze position recordings. Eye movements were analysed offline using custom software written in 'C' on the Macintosh. Fixations were categorised according to where they landed on a 3×2 grid, which divided the pictures into six sectors of equal area (Fig. 1). Eye movement traces were visualised by the experimenter and played back at slowed speed superimposed over the picture that was being viewed during that trial. Any obvious offset in eye position due to slippage of the head-band or gross head movements were corrected for at this stage. The experimenter could also reject any fixations, which were contaminated by eye blink or eye-lid clipping artefacts. Individual saccades were then identified using a semi-automated procedure, as periods in the eye position signal where the instantaneous, absolute velocity rose above 30° per s for more than two data samples. Fixations were identified as pauses between saccades longer than 50 ms in duration (in order to exclude short fixations preceding corrective saccades). Fixation duration, horizontal and vertical position and grid location were then outputted to text files for statistical analysis (Fig. 1).

Table 2
Mean total time to solve problems for problems with different minimum moves to solution for patients and control groups

	Minimum moves to solution				
	One	Two	Three	Four	Five
Controls	4234 ± 863	5930 ± 783	7497 ± 1581	12 565 ± 1805	16 817 ± 2151
PD	4409 ± 444	5742 ± 1253	8418 ± 961	10 506 ± 1560	13 392 ± 2270

3. Results

3.1. Response times

A two-way ANOVA with subject group (Control/PD) and problem difficulty (one to five moves) as factors confirmed that response times increased strongly with the minimum number of moves required to solve each problem (main effect of difficulty: $F(4,60) = 26.61$, $P < 0.0001$). However, there was no significant main effect or interaction effect of subject group on response times in the task (Table 2).

3.2. Errors

A two way ANOVA with subject group and problem difficulty as factors showed that error rates also increased strongly with sequence length (main effect of difficulty $F(4,72) = 18.59$, $P < 0.0001$). There was also a difference in the total number of errors between the control and patient groups although this was found to be significant only for the most difficult problems (interaction group × difficulty: $F(1,18) = 6.19$, $P < 0.05$)(Table 3).

Earlier reports have suggested that error rates were not significantly increased for mildly affected patients and that the deficit in performance becomes more pronounced as the disease progresses. We, therefore, examined the effect of disease severity by correlating the total number of errors made in the task with each patient's Webster Score. This analysis revealed a significant correlation between the Webster rating and error rates in the planning task ($R^2 = 0.80$; $F(1,7) = 27.80$, $P < 0.005$)(Fig. 2). In contrast, no significant correlation was found between response times and disease severity, as indexed by the Webster score (Fig. 2).

3.3. Basic saccadic measures

Basic saccadic measures were analysed using two-way ANOVAs with subject group and problem difficulty as factors, with either fixation duration, number of eye movements or amplitude of saccades as the dependent variables. Both the total number of eye movements made and the duration of individual fixations increased strongly with problem difficulty for both subject groups (main effects of problem difficulty: $F(4,60) = 20.02$, $P < 0.0001$; $F(4,60) = 9.45$, $P < 0.0001$, respectively). However, there was no significant difference between patients and controls in the total number of eye movements made or the duration of each individual fixation ($F(1,15) = 0.36$; $F(1,15) = 0.007$). Under some conditions eye movement amplitudes are found to be hypometric in PD [29]. However, no significant difference in saccade amplitude was observed between the two subject groups ($F(1,15) = 0.19$).

3.4. Gaze times

Further analysis of the eye movement data was based upon the distribution of gaze times within each segment of a 3×2 analysis grid superimposed on top of each problem picture (Fig. 1). Data for control subjects were entered into a two-way ANOVA with visual field (Goalspace/Workspace), and problem difficulty as factors. Consistent with our earlier investigations [27], the total time spent fixating the Workspace increased more strongly with problem difficulty than did the total time fixating the Goalspace (interaction visual field × problem difficulty: $F(4,20) = 6.57$, $P < 0.002$). This was found to be the case regardless of whether the Goalspace was in the upper or lower visual field, such that a three-way ANOVA with instruction condition (Upstairs/Downstairs), visual field (upper/lower) and problem difficulty as factors produced a significant interaction between instruction condition and visual field ($F(4,20) = 6.57$, $P < 0.002$)(Table 4 Fig. 3). Further inspection of the eye movement data revealed that this increase in total fixation time reflected an increase in both the number of eye movements, as well as the duration of individual fixations (Table 4).

Table 3
Mean number of response errors per block at each sequence length for controls and PDs (four trials were presented at each sequence length)

	Minimum moves to solution				
	One	Two	Three	Four	Five
Controls	0.1	0.3	0.6	1.4	2.0
PD	0.2	0.8	1.2	2.0	3.2

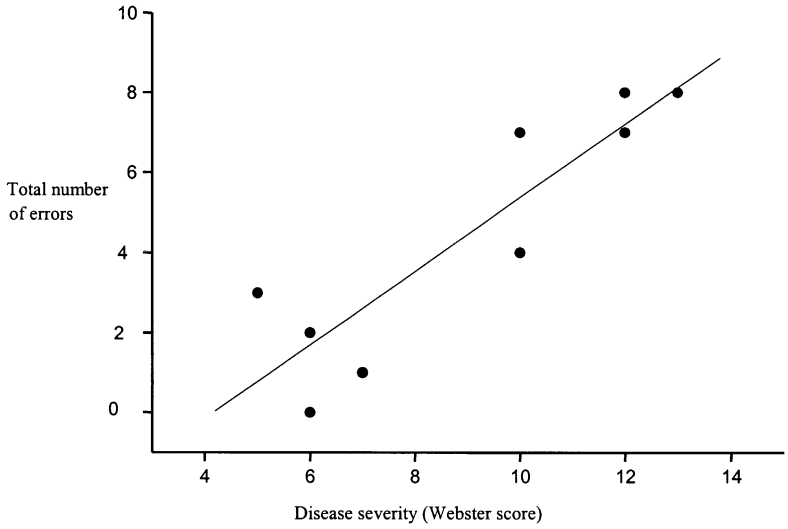

Fig. 2. Graph showing correlation between the total number of errors in the TOL task and motoric symptoms of Parkinson's disease as indexed by the Webster score rating (least squares linear fit line shown).

In contrast, the identical analysis for the PD patients showed no significant difference in the amount of time spent fixating the two parts of the display at any level of problem difficulty (main effect of visual field $F(4,32) = 0.49$). Even at the most difficult problem level, there was no dissociation in the average time spent fixating the Goal and Workspace (Table 4 Fig. 3). This difference in gaze strategy was confirmed using a three way two within, one between groups ANOVA with subject group, visual field (Goalspace/Workspace) and problem difficulty as factors. A significant three way interaction was detected, confirming that there was a significant dissociation between the two groups for the most difficult problems (group × field × difficulty interaction: $F(4,60) = 4.08,\ P < 0.01$).

The abnormal gaze strategy used by PD could be explained simply by the fact that patients made more errors than controls. Similarly, any tendency for the patients to press the response key impulsively before problems had been solved could also result the observed dissociation in gaze times. In order to investigate the effect further data from all subjects were subjected to a three way ANOVA with group, response type (Error/No error) and visual field (Goalspace/Workspace) as factors. No significant interaction was found between response type and any other factor, indicating that PDs' non-selectivity of gaze did not depend upon whether an error or correct response had been given (group × response × visual field $F(1,64) = 0.056$). Likewise no correlation was found between total response times and the relative difference in gaze times between the two halves of the display ($R^2 = 0.41$, $F(1,5) = 0.71$, $P = 0.04$), suggesting that there was no direct relationship between the two variables (Fig. 3).

Table 4
Mean total gaze time per trial spent in upper and lower visual fields for each subject group and instruction condition

	Downstairs		Upstairs	
	Upper	Lower	Upper	Lower
Controls	6551 ± 1995	12 231 ± 1772	9482 ± 2232	4714 ± 1653
PD	5237 ± 1067	7384 ± 1946	5143 ± 1858	9019 ± 3393

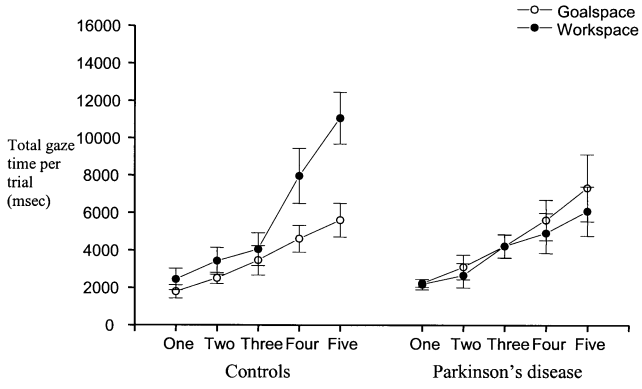

Fig. 3. Total gaze duration per trial in the Goalspace and Workspace regions of TOL problems. Control subjects show a strong increase in the time spent fixating the Workspace on more complex problems, reflecting elaboration of problem solutions. In contrast, PDs do not show any difference in the time spent fixating the two parts of the display even for the most demanding problems.

Table 5
Mean probability of a given saccade resulting in a lateral shift between zones in the analysis grid in each half of the display

	Minimum moves to solution				
	One	Two	Three	Four	Five
PDs					
Goalspace	0.11 ± 0.01	0.11 ± 0.03	0.17 ± 0.02	0.18 ± 0.04	0.18 ± 0.04
Workspace	0.10 ± 0.02	0.14 ± 0.02	0.16 ± 0.03	0.15 ± 0.03	0.17 ± 0.03
Controls					
Goalspace	0.08 ± 0.02	0.11 ± 0.02	0.13 ± 0.04	0.13 ± 0.03	0.13 ± 0.03
Workspace	0.17 ± 0.04	0.15 ± 0.03	0.19 ± 0.02	0.28 ± 0.03	0.25 ± 0.05

3.5. Saccadic shifts between grid locations

Next we examined the likelihood of particular transformations in gaze position occurring between different regions of the display. For example, the probability of a given fixation being followed by another fixation at a grid location horizontally adjacent to it was calculated and analysed according to whether the two fixations occurred in the subject's Goalspace or Workspace. For control subjects, the probability of making a lateral shift between grid locations was significantly greater in their respective Workspace. This difference was most apparent for the most difficult problems (visual field × problem difficulty interaction: $F(1,6) = 6.68$, $P < 0.05$)(Table 5). However, as with the analysis of total gaze times, the identical ANOVA for the PD group failed to show this dissociation in lateral saccadic shifts between the two halves of the display (main effect of visual field: $F(1,9) = 0.02$) (Table 5).

A similar analysis of the probability of making a vertical shift in fixation between adjacent grid locations did not show any significant changes dependent upon problem difficulty or subject group.

3.6. Problem dependent gaze shifts

Our earlier studies showed that gaze is not distributed evenly across all the locations within the Workspace region, but is systematically biased towards problem relevant items [27]. We carried out the same analysis on the present data by comparing trials on which the left-sided balls were the most important items for problem solution, with those for which the centrally located ball was the critical move to make. For the Downstairs group, the central ball in the Workspace is always the blue ball, and consequently, these problems are often termed 'blue-ball' problems. For four move problems, blue-ball trials always require a shunting maneuver in which the central ball is moved to a temporary location whilst one or more intervening moves are made using the other balls. Failure to realise that this is the critical maneuver leads to an impasse in which the problem cannot be solved without undoing earlier moves.

A two-way ANOVA with problem type (blue/non-blue) and grid location (left/centre) as factors confirmed that the elderly control subjects displayed the expected interaction between problem type and gaze time per trial spent looking at the left and central locations within the Workspace ($F(2,12) = 10.69$, $P < 0.005$). Gaze was biased towards the lateral balls on 'non-blue ball' problems and the central location on 'blue-ball' trials. In contrast, PDs failed to show this selectivity in gaze for problem critical items. There was no significant difference in the time PDs spent fixating the different locations in the Workspace dependent upon problem type ($F(2,18) = 1.81$, $P > 0.1$).

Once again, in order to understand this effect further we carried out a trial by trial analysis on the data from the PD group, in which correct and error response trials were separated. A three-way ANOVA with problem type, response type (error/no error) and location revealed that eye movement patterns made on correct trials differed systematically from those on incorrect response trials (problem type × response type × location interaction $F(4,32) = 3.11$, $P < 0.05$). Correct trials showed the expected dissociation between blue and non-blue ball problems, but for error trials there was no dissociation in the distribution of gaze times between the two types of problem (Fig. 4).

4. Discussion

The performance of control subjects in the current study replicates our earlier findings with younger controls [27]. Efficient solution of TOL problems is associated with a characteristic pattern of eye movements, which cannot be explained purely by the features of the visual stimulus the subject is looking at. Specifically, subjects always bias their gaze towards the Workspace part of the display when planning/elaborating solutions, regardless of whether they are instructed to manipulate the balls in the upper (Upstairs group) or lower (Downstairs group) part of the picture. Clearly, it

is difficult to accommodate these results within accounts of eye movement control which emphasise the salient features of the visual image as the most important variable controlling the distribution of fixations [41,56]. Instead, particular tasks are associated with dedicated attentional 'sets' or strategies, which direct gaze to different regions of space or stimulus features dependent upon current task goals [5,10,11,24,33,61].

As well as having implications for models of eye movement control, our observations also bear on the nature of problem solving itself. For example, it is often assumed that cognitive planning involves the construction of a detailed internal program for the entire movement sequence, which has to be encoded into memory and then later recalled to control execution of the correct solution (e.g. [21]). But recent research has suggested that this classical view of planning may be incorrect [12,52,58] and our findings also support an alternative view in which only the key features of problems are encoded during planning. For example, we did not find evidence for stereotyped sequences of eye movements corresponding directly to the rehearsal of a fully formed action sequence. However, we do find that normal subjects selectively bias gaze towards one or two problem critical locations and this selectivity is crucial for efficient performance of the task [27]. It has also been suggested that point of gaze may constitute a parsimonious 'deictic' code for cognition [6]. Rather than constructing detailed representations of the external world, spatial co-ordinates may be used to guide re-foveation of parts of the visual scene whenever information about the location's contents are required. Similarly, for the TOL task a major component of the planning phase may involve determining the location of one or two problem critical balls. The full sequence of moves does not need to be memorised in detail during solution rehearsal because once the key location has been foveated other action systems would be directly cued into executing the appropriate behavioural sequence to solve the problem. In not so many words, we may solve problems 'with our eyes' rather than 'in our heads'.

Although the data from control subjects have interesting implications on their own, the primary motivation of this particular study was to compare the eye movements made by older controls and Parkinson's disease patients. This comparison revealed a striking dissociation between the two groups. In contrast to control subjects, PDs show no dissociation in gaze times between the two ball arrays. Total time fixating the Workspace and Goalspace was equally distributed for all problem difficulties. Error rates were also significantly increased, but unlike earlier reports, no significant difference in response time was found between the two groups.

The behavioural differences between the two subject groups are unlikely to reflect purely visual or oculomotor abnormalities in PD, as they were modulated by the complexity of the task. Simple problems were completed accurately by both groups, suggesting that patients understood the task and were quite capable of making a simple comparison between the upper and

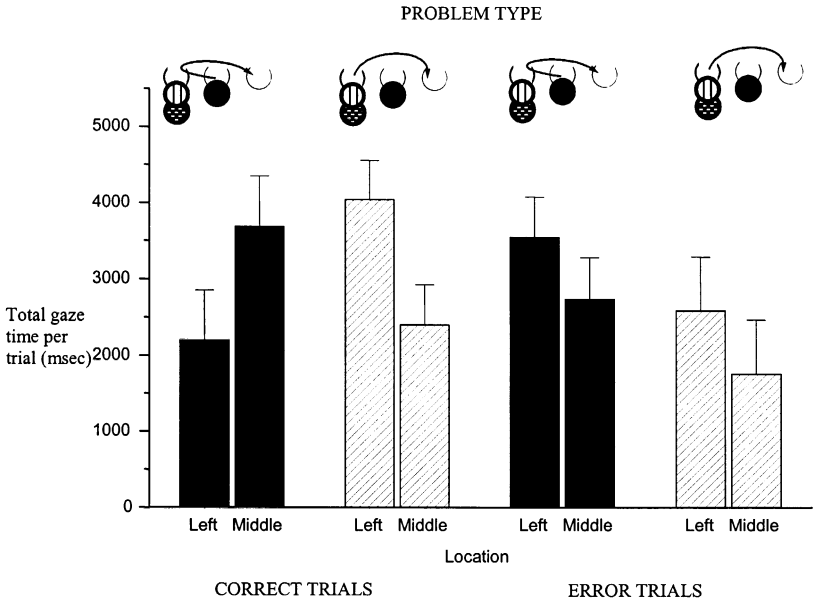

Fig. 4. Mean total gaze time spent by PDs looking in left and middle ball locations within the Workspace for different problem types on correct and error trials (four move problems only). On correct trials, more time is spent fixating balls, which are critical for solution of the current problem. Gaze is selectively biased towards the centrally located blue ball on 'blue ball' problems and towards the left-sided balls on 'non-blue ball' problems. In contrast, error trials fail to show any problem dependent bias in gaze duration.

Table 6
Relative times fixating Goalspace and Workspace for two unmedicated PD patients

	Goalspace	Workspace
Controls		
Elderly controls	4603 ± 712	7962 ± 1478
Young Controls	1223 ± 331	2172 ± 190
Unmedicated PDs		
Patient JR	6760	6984
Patient AN	2105	2494

Data for elderly controls and patient JR shown for four move problems taken from the present study. Data for young controls and patient AN shown for three move problems in simpler version of task described elsewhere [28]. Both unmedicated patients show a selective increase in fixation times on the Goalspace relative to their peer group.

lower arrays of balls. Neither was there a significant dissociation between groups in the relative time fixating the Goal and Workspace arrangements on simple problems. It was only for more complex problems that an abnormal distribution of eye movements became apparent.

The number of errors made by PDs correlated strongly with disease severity, confirming the existence of a steady decline in cognitive ability as the disease progresses [57,40]. But what is the exact nature of this cognitive dysfunction and can eye movement data be used to throw light on this question? We originally reasoned that eye movement measures would allow us to discriminate between two potential explanations for abnormal problem solving in PD; a general slowing of mental imagery (bradyphrenia) and defective encoding/maintenance of current goals on-line (goal-processing deficit). The data came out clearly in favour of the goal-processing hypothesis. No dissociation was observed between Goal and Workspace fixation times suggesting that PDs have difficulty encoding and/or maintaining task goals leading to increased response errors. More detailed analysis suggests that this abnormal gaze strategy, although accompanied by increased error rates, did not exclude correct planning on a subset of trials. Patients correctly directed their gaze towards the problem critical balls on correct but not incorrect response trials.

In contrast to earlier investigations of cognitive planning in PD (e.g. [49]), no significant increase in total response time was observed in our PD group. Heterogeneity of cognitive deficits in the general population of PDs may have contributed to this discrepancy with earlier work, but it also raises the possibility that our patients were impulsively pressing the response key before they had completed the correct solution. This in itself would lead to both response errors and a distribution of gaze times more similar to that seen for control subjects on simpler problems (Fig. 3). However, several aspects of the data make it unlikely that errors and impulsivity directly explain abnormal gaze patterns. For example, we have recently found that a sub-set of control subjects make a large proportion of errors during the TOL [27]. However, unlike PDs, these individuals bias their gaze towards the Workspace in the same manner as efficient planners do. We also found that patients' relative increase in Goalspace gaze durations did not vary significantly between error and non-error trials. Similarly, correlation of response time with the difference in gaze times between the two halves of the display also failed to yield a significant relationship. These analyses imply that the lack of selectivity between the two display regions cannot be directly attributed to impulsivity and errors. On the contrary, it suggests that the patients have a consistent problem in encoding and maintaining task goals, which leads to premature responses and errors on some (but not all) trials.

Another factor that complicates our understanding of cognitive dysfunction in PD is the potential role of medication. It is possible that some of the effects observed may be attributable to various aspects of medication as both L-Dopa [23], and scopolomine [18] have been shown to detrimentally affect performance in PD patients. While it is not possible to resolve this issue entirely, the fact that the majority of studies show improved performance in patients on L-Dopa, either compared with the 'off L-Dopa' condition [35], or when compared with matched groups of patients who are not on L-Dopa (e.g. [44,47,49]), suggests that the deficits observed in the current study are not a direct result of dopaminergic medication. Further, all our patients showed clinical signs of Parkinson's disease (e.g. rigidity, bradykinesia) indicative of a hypo-dopaminergic state, rather than a drug induced excess of the chemical. A single patient (JR) in this study was unmedicated but also showed a lack of selectivity between fixation times between the two halves of the display (Table 6). We have also tested a young (31 years) unmedicated Parkinson's individual (patient AN) using a simpler but formally identical version of the task in which problem difficulty varies between one and three moves. Once again, in contrast to his peers, this patient showed no bias in fixation towards the Workspace region of the display (Table 6). Taken together, the evidence from earlier studies and our own investigations suggest that medication effects could not have influenced the major finding of the current study.

Abnormal goal-processing in PD could itself be due to dysfunction in a number of sub-processes which are often subsumed into the concept of an 'executive' or 'supervisory attentional' mechanism [3,55]. This poorly defined process has been invoked to explain the control of non-routine aspects of behaviour, which are not

clearly specified by an external sensory context or earlier learnt action plans. The current findings are consistent with the view that prefrontal–striatal networks play a critical role in cognitive planning and executive processes [15,37,4], but the exact components of executive control which are impaired in PD are more difficult to discriminate using the current data set. However, a number of different explanations for the patients' deficit present themselves for further investigation.

The simplest account of the present findings is that trans-saccadic working memory for the colour and location of balls is impaired in PD. Put simply, patients keep forgetting the arrangement of balls in the Goalspace every time they look away. Due to this, they spend proportionately less time fixating the Workspace and more time fixating the Goalspace. Alternatively, it is also possible that normal subjects use covert attention to continuously monitor the goals in peripheral vision whilst fixating the Workspace. If this were the case then the present results imply an impairment in the control of covert attention in PD. Although we cannot discriminate between these two accounts using the current data set, recent studies of a closely related block-copying task indicate that covert attention is not used to monitor goals in this manner. Instead, the colour and location of relevant items are acquired during separate fixations and are held in working memory across gaze shifts between different regions of the display [26].

More speculatively, our results are also consistent with another type of impairment in PD. Rather than a deficit in visual working memory, PDs may have difficulty with the attentional selection of different behavioural sets or schema which would normally be activated at different stages of the task. Patients may be unable to disengage from a scanning set in which the location and colour of balls are acquired, in order to switch to solution elaboration mode in which imagined representations come to the fore and gaze is directed to the Workspace [38,54,22]. PDs remain locked into a scanning mode in which goals are repeatedly assessed and proper rehearsal of problem solutions is consequently impeded. Interestingly, the ability to rapidly switch between gaze control sets would seem to be necessary for the performance of a range of complex tasks ranging from solving math problems [19] to making a cup of tea [34]. Yet to date only a very few studies have explicitly examined this aspect of eye movement control in normals and neurologically impaired populations [25,59,28].

Ultimately, only further experimentation will permit a discrimination between different explanations for the goal-processing deficit in PD. For example, the use of gaze contingent display changes could allow us to probe how much information is retained in memory between successive fixations. However, one recent theory suggests that it may not always be useful to conceive of covert attention, working memory and cognitive set as reflecting the operation of discrete and entirely dissociable sub-processes. An interesting alternative view is that they represent aspects of the same generic function subserved by prefrontal cortex, namely the top–down modulation of activity within posterior cortical regions. According to this model, the prefrontal cortex maintains a constellation of synaptic facilitations, biases, associations and inhibitions which modulate an organisms behaviour by changing the neural 'context' within which posterior sensory and motor regions operate [13,14,2,36,51,16]. Interestingly, a crucial role in this framework is hypothesised for extra-cellular dopamine in the modulation of prefrontal neuronal activity. Recent neural network simulation studies also suggest that different degrees of dopaminergic disruption within prefrontal cortex can lead to quite different behavioural symptoms, which would conventionally be interpreted as reflecting deficits in discrete cognitive functions [7]. This account predicts that the goal-processing deficit in PD occurs due to dysfunction in a range of cognitive operations, all of which have their origin in dopaminergic depletion within fronto–striatal networks.

5. Summary and conclusions

In summary, we have shown that as well as making increased response errors, Parkinsons' patients use abnormal gaze strategies during the TOL task. Rather than directing their eyes towards the Workspace during planning as controls do, PDs divide their attention equally between the Goalspace and Workspace region of the display. The pattern is not consistent with slowed motor imagery but suggests that the planning deficit in PD is due to abnormal encoding and maintenance of current goals. This goal-processing abnormality may reflect disruption to trans-saccadic working memory or attentional control due to dopaminergic depletion within prefrontal–striatal networks.

6. Uncited references

[46,48].

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