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RESEARCH NOTE

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Effects of visual feedback on manual tracking and action tremor in Parkinson's disease

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Abstract Visual feedback is one of the key elements in on-line control of smooth manual tracking. To investigate the effects basal ganglia dysfunction have on visual feedback control, we have tested six advanced Parkinson's disease (PD) patients in comparison with normal controls using visually guided wrist tracking tasks. Tracking performance was assessed under three visual conditions: (1) both guiding target and movement cursor were displayed continuously; (2) the target display was turned off for the second half of each trial; or (3) the cursor display, but not the target, was turned off for the second half of each trial. Thus, for the second half of each trial under conditions 2 and 3, no visual feedback of the relationship between the target and the cursor was available. Results showed that although PD patients had significantly larger tracking errors than controls, and errors significantly increased in both PD patients and controls after withdrawing either visual cue, increases in tracking errors in PD were not significantly different from those in controls. Nor were any significant changes found in the frequency (6–8 Hz) or magnitude of the PD patient's action tremor after withdrawing visual feedback. These results suggest that on-line movement control of wrist tracking movements in advanced PD is not especially reliant on visual feedback. In conjunction with our previous study of multiple sclerosis (MS) patients, the present results confirm that the basal ganglia is less involved in visual guidance of smooth manual tracking than the cerebellar circuits.

Key words Visual feedback · Motor control · Action tremor · Parkinson's disease

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Introduction

It is well known that visual feedback plays an important role in on-line control of smooth tracking movements. Evidence suggests that visual feedback is predominantly processed by the cerebellum, since the visual dependence upon on-line movement control is significantly increased when the cerebello–cerebral pathway is damaged (Stein 1986; Horne and Butler 1995; Liu et al. 1997). Recent functional imaging studies (Brooks 1995; Jueptner and Weiller 1998) suggest that, in contrast to the cerebellar circuit, the basal ganglia–cortex pathway is more concerned with selection of the appropriate movement/muscles rather than with visuomotor guidance. Several studies using visually guided motor tasks have been carried out to investigate changes in kinematics (Jackson et al. 1995; Majsak et al. 1998), control strategy (Flowers 1978) and accuracy (Johnson et al. 1996) of movements caused by dysfunction in the basal ganglia in Parkinson's disease (PD). Questions remain: (1) is on-line control during smooth manual tracking in PD especially reliant on visual feedback? and (2) what is the effect of withdrawing visual feedback on action tremor in PD patients? To answer these questions, we assessed the tracking performance and action tremor in six PD patients using visually guided smooth-pursuit wrist tracking tasks in which the visual display of either the target or the movement was selectively switched off in the middle of tracking. Our visually guided motor task differs from the tasks employed in previous studies (Flowers 1978; Jackson et al. 1995; Johnson et al. 1996; Majsak et al. 1998) in three major aspects: (1) the movements our PD patients performed were relatively slow; (2) movement velocities were pre-defined and constant during each trial rather than self-determined and variable during each trial; and (3) visual feedback was withdrawn at the middle point of a continuous tracking movement. This task enabled us to investigate on-line visual feedback control during slow but targeted movements with constant movement velocities in PD patients.

Method

PD patients and normal controls

Six inpatients at the Department of Neurosurgery, Radcliffe Infirmary, Oxford, four females and two males, aged 59–69 years (65 years on average), were studied. All had been diagnosed as having clinically definite PD for 8–35 years (average 18 years), with off-medication UPDRS (Unified Parkinson's Disease Rating Scores) of 84–127 (average 105). These patients had been selected for possible surgical treatment because they had frequent and prolonged off periods and highly disabling involuntary movements. Tracking performance was assessed in nine hands of these six patients, in both on- and off-medication, and a matched number of hands from five healthy subjects without neurological deficits were also tested as controls. Ethical approval and informed consent were obtained for this study.

Visually guided smooth-pursuit tracking task

Our wrist tracking task was previously described in detail (Liu et al. 1997) and is summarised as follows: a target consisting of a 12×12-pixel hollow square was displayed on a computer screen. It was initially stationary near one side of the screen; at the start of each trial, it moved horizontally at a constant speed to the other side of the screen and then stopped. Target velocities were 13.64, 9.23, 7.50 and 5.50°/s and were randomly allocated among 16 flexion movements giving four trials at each target velocity. The subject's forearm was supported in an adjustable plastic splint fixed to the arm of a chair, adjusted for each subject to firmly hold the forearm while allowing comfortable wrist flexion and extension over a range of 60° (±30° around the neutral position) in the horizontal plane. The subject held a low-resistance hand-held joystick recording wrist flexion and extension. The joystick position was displayed on screen as a 6×6-pixel hollow square cursor. The subject was instructed to make a wrist flexion movement to keep the cursor inside or as near to the moving target as possible, then to move back to the starting position with an unpaced extension movement for the next trial.

Pursuit tracking was recorded under three visual conditions:

Condition 1 (both-on). Both cursor and target were displayed continuously, so that feedback of visual comparison of target and cursor was available.

Condition 2 (target-off). The target, but not the cursor, display was turned off for the second half of each trial. The subject saw the target start each sweep but, as it reached the screen centre, it was extinguished, reappearing in its final position 1 s after the end of each trial. The subject was instructed to keep tracking the estimated position of target and had visual feedback of his or her own movement from the cursor which remained visible throughout.

Condition 3 (cursor-off). The cursor, but not the target, display was turned off for the second half of each trial. The subject saw the cursor at the start of each sweep but, as the target reached the screen centre, the cursor was extinguished, reappearing 1 s after the end of each trial. The subject was instructed to track the continuously displayed target without visual feedback of his or her movement position.

Thus, for the second half of each trial under conditions 2 and 3, no visual information about the spatial relationship between the target and the cursor was available. Each subject was allowed a few practice trials on each task before recording began.

Data acquisition and analysis

The wrist position signals were digitally differentiated and filtered using a zero-phase, four-pole Butterworth filter (corner frequency 25 Hz). A computer algorithm then selected tracking segments beginning 1 s after the target started moving until the end of the trial, thus eliminating the subjects' initial reaction delay and acceleration phases under condition 1. Under conditions 2 and 3, only the second half of the tracking trials were selected, and results were compared with those obtained under condition 1. The computer then determined the target velocity, the mean movement velocity and the standard deviation of movement velocity (SD-MV) for each of the 16 trials. Means and standard deviations over trials were then calculated for each hand tested. The accuracy of voluntary tracking was expressed as the percentage mean velocity of the subjects' movement relative to that of the target; thus, perfect tracking would have a value of 100%. Impaired control of MV was reflected in the absolute percentage error in the movement velocity (EV) relative to the target velocity. The magnitude of action tremor was quantified by calculating the SD-MV; for perfect, smooth tracking, the standard deviation of the movement velocity would be zero. The frequency composition of the tracking records was also computed. The same segments of the velocity records were used as above, and the mean velocity was removed from each segment. The data were padded with zeroes to provide 1024 data points per segment and the Fourier transform calculated. Mean power spectra were calculated from the 16 trials per hand and averaged across patients. Results of tracking error and tremor magnitude were first statistically compared between on- and off-medication under the three different visual conditions using a two-way analysis of variance (ANOVA). No statistically significant differences were found in either index between on/off medication. Therefore, values averaged across on/off medication were used to make the statistical comparison between patients and controls in three different visual conditions. Differences in peak frequency and peak magnitude of action tremor in PD patients between visual conditions were tested using one-way ANOVA.

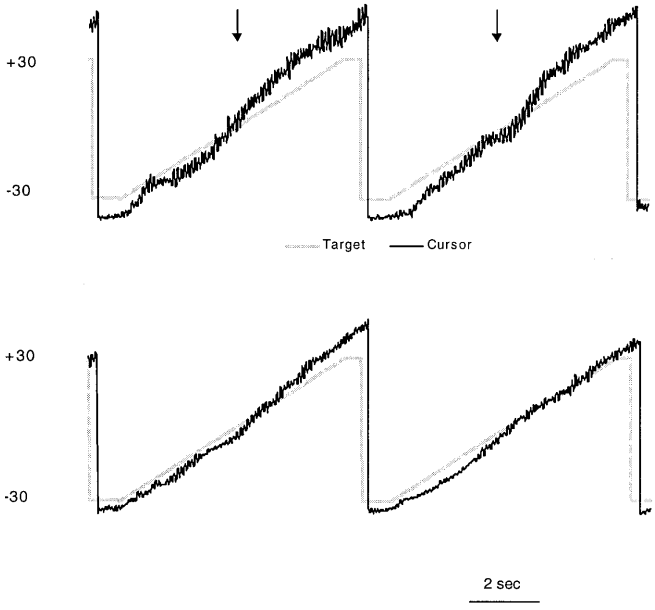

Fig. 1 Tracking with and without display of visual cues. The *ramp/step* indicates the constant velocity motion of the target across the screen; the *heavy line* indicates the tremulous motion of the cursor. During the first half of each trial, both target and cursor were displayed. In the *upper* figure, the display of the target but not of the cursor was turned off for the second half of each trial (*arrows*); in the *bottom* figure, the display of the cursor but not of the target was turned off for the second half of each trial

Results

Examples of two consecutive tracking trials recorded from one patient off-medication are illustrated in Fig. 1.

Table 1 Error in tracking velocity (EV, degrees per second) with and without display of visual cues in Parkinson's disease (PD) patients and controls

Subjects	Visual conditions			ANOVA
(*n*=9)	Both on	Target off	Cursor off	
On-medication	10.7±12.1	28.8±19.6	17.0±13.0	$F_{1,48}$=0.1; *P*>0.8
Off-medication	13.1±13.7	17.0±13.0	22.7±11.9	
ANOVA		$F_{2,48}$=2.9; *P*>0.06		$F_{2,48}$=1.9; *P*>0.2
PD	11.9±12.7	22.9±17.2	19.9±12.4	$F_{1,48}$=35.6; *P*<0.000001
Controls	1.4±0.9	6.5±5.2	5.9±6.6	
ANOVA		$F_{2,48}$=4.5; *P*<0.02		$F_{2,48}$=0.6; *P*>0.6

Table 2 Variability in tracking velocity (SD-MV, degrees per second) with and without display of visual cues in Parkinson's disease (PD) patients and controls

Subjects	Visual conditions			ANOVA
(*n*=9)	Both on	Target off	Cursor off	
On-medication	532.3±899.2	512.0±1113.0	370.9±682.1	$F_{1,48}$=0.1; *P*>0.7
Off-medication	646.9±956.0	479.5±751.5	500.1±523.8	
ANOVA		$F_{2,48}$=0.2; *P*>0.8		$F_{2,48}$=0.05; *P*>0.9
PD	589.6±902.2	495.8±921.4	435.5±593.7	$F_{1,48}$=9.9; *P*<0.003
Controls	6.5±0.8	5.9±1.2	6.0±1.0	
ANOVA		$F_{2,48}$=0.08; *P*>0.9		$F_{2,48}$=0.08; *P*>0.9

Both visual cues are displayed during the first half of each trial. In the top traces, the target but not the cursor display was turned off for the second half of each trial (starting points are indicated by the *arrows*). In the bottom traces, the cursor but not the target display was turned off for the second half of each trial. Comparing the first and second half of tracking records, errors in tracking velocity increased after one or other visual cue was selectively turned off, but no significant change in magnitude of action tremor or shift in its frequency was seen.

Averaged values of tracking error with and without display of either target or cursor in PD patients (on- and off-medication) are listed in the top half of Table 1. Since there was no significant difference between on- and off-medication, the values in patients then were combined and compared with controls. Results are listed in the bottom half of the table. PD patients had significantly larger errors in their tracking than controls ($F_{1,48}$=35.6; *P*<0.000001), and there were significant increases in the tracking error in both patients and controls between tracking conditions with and without visual cues ($F_{2,48}$=4.5; *P*<0.02). However, there was no significant interaction ($F_{2,48}$=0.6; *P*>0.6), which suggests that the increase in error was similar in the PD patients and in the controls.

The averaged values of the standard deviation in tracking velocity with and without display of either visual cue in PD patients (on- and off-medication) and controls are listed and compared in the same fashion as above in Table 2. No significant difference was seen for the PD patients on- and off-medication. The PD patients had significantly larger SD-MV than controls due to their profound action tremor ($F_{1,48}$=9.9; *P*<0.003), but no significant difference was seen in the SD-MV across subject groups between tracking conditions with and without visual cues ($F_{2,48}$=0.08; *P*>0.9). As before, the interaction term of the ANOVA was also non-significant ($F_{2,48}$=0.08; *P*>0.9).

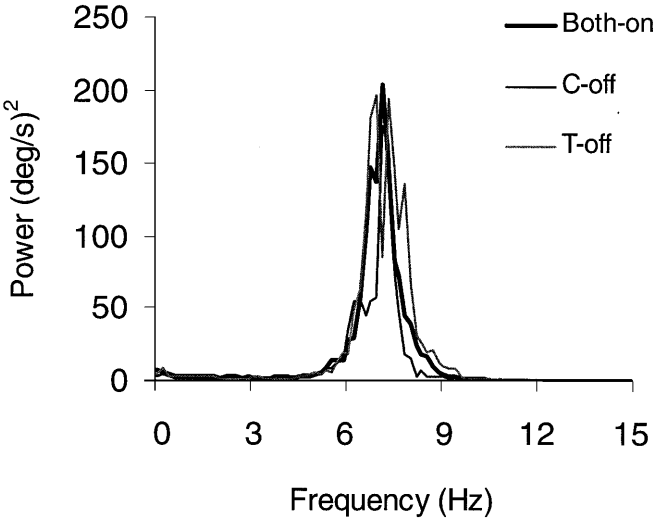

Fig. 2 Averaged power spectra of tracking movements in Parkinson's disease (PD) patients. A peak of action tremor was revealed at 6–8 Hz. No significant change in either magnitude or frequency of action tremor was found between conditions of the target and cursor being displayed (*Both on*), the target (*T-off*) or the cursor (*C-off*) being turned off

The action tremor in these PD patients ranged from 6 Hz to 8 Hz, peaking at 7 Hz (Fig. 2). Neither the fre-

quency (P>0.5, one-way ANOVA) nor the magnitude (P>0.6) of action tremor was significantly altered by withdrawing feedback of visual cues.

Discussion

We have investigated in six advanced PD patients how their on-line control of tracking movements and their action tremor was affected by visual feedback using visually guided wrist tracking tasks, in which visual feedback of either the guiding target or movement cursor was selectively withdrawn during tracking. Compared with the normal controls, the PD patients had greatly increased tracking error and variability in their mean velocity, but increases in tracking errors following withdrawal of display of either visual cue were not significantly different between PD and controls. In addition, neither the frequency nor magnitude of PD action tremor was significantly affected by withdrawing the visual cues. These results suggest that the on-line control of slow and smooth manual tracking in these PD patients did not rely on visual feedback more than in the normal controls, and their action tremor is not visually driven.

In a recent study carried out by Majsak and colleagues (1998), PD patients were required to reach as fast as possible to grasp either a stationary or a moving ball. In response to the visual-driving stimulus of a moving ball, PD patients were able to exceed their self-determined maximal speed of reaching and still maintain movement accuracy, suggesting that the visual cue of target motion was beneficial. Jackson and colleagues (1995) found that PD patients exhibited a constant underscaling of peak velocity in open-loop reaches when vision was occluded approximately 2 s prior to the execution of reaches. Thus, they suggested that PD might be especially dependent on visual feedback to guide movements. In contrast to these reports, using visually guided reaching tasks in which visual feedback was occluded at the onset of reaching movements, others (Jeannerod 1984; Jakobson and Goodale 1991) have found that visual feedback was not necessary for the successful execution of reaching movements. Our results showed that the increase in tracking errors in PD patients in the second half of each trial was not significantly different from those in controls. These results suggest that in our PD patients there was no significant increase in visual dependence during slow-pursuit tracking movements. Despite the differences between tasks used in different studies, it seems that visual dependence of movement control in PD depends largely on the time at which visual information is provided or withdrawn in relation to the movement. Because PD patients have difficulties in internally generated movements (Jahanshahi et al. 1995), external cues help them to prepare and initiate a movement. However, visual dependence for controlling an on-going movement in PD patients does not significantly increase when compared with controls.

Our results support current ideas about the differences between basal ganglia and cerebellar control of movement (Jueptner and Weiller 1998), namely that the basal ganglia may be more concerned with the selection of the appropriate movements/muscles (the efferent motor component) or/and control strategies, whereas the cerebellar circuit calibrates basic motor commands by adjusting movement parameters, monitoring the outcome of movements, and optimising them accordingly (sensory information processing and sensorimotor integration).

The other major finding in the present experiment was that neither the magnitude nor the frequency of their action tremor (6–8 Hz) was significantly affected by withdrawing visual feedback. This suggested that the action tremor in our PD patients does not contain an active response to the visual feedback of their tracking error.

In contrast to these PD patients, our previous study (Liu et al. 1997) in multiple sclerosis (MS) patients with cerebellar involvement showed that their dependence on visual feedback control in the same tracking tasks was significantly increased. We argued that this was due to impairments in the visuomotor transformation carried out by the cerebellar circuit and/or in their short-term motor memory. In addition, visual feedback of the spatial mismatch between the target and cursor that reflected their MS action tremor (3–5 Hz) provoked voluntary error correcting movements of low frequency (1–2 Hz). When visual feedback of the error was withdrawn by turning off either the target or the movement cursor, these compensatory responses were not evoked, and there was significant 30% suppression in the magnitude of the action tremor.

The current theories of the basal ganglia in motor control do not explain why the PD patients studied here do not respond to the visual feedback of their tremor with compensatory movements, while the MS patients who have tremor caused by impaired cerebellar function do make corrective responses. One possible explanation may be that the frequency of action tremor in PD is usually higher (6–8 Hz) than that in MS (3–5 Hz). It may be that PD patients are simply unable to react to the higher frequencies of their action tremor. This is supported by a study on the frequency response of PD in pursuit tracking of sine waves (Flowers 1978). It was found that PD patients lost the tracking at a much lower frequency than the normal controls did, and at frequencies as low as 1.0 Hz they approached the level at which they were performing no better than holding the joystick still and not tracking at all.

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