PREDICTIVE OCULAR MOTOR CONTROL IN PARKINSON'S DISEASE

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SUMMARY

A comparison was made of predictive eye movements of both the saccadic and pursuit ocular motor systems in parkinsonian patients and aged-matched normal controls. In a predictive task our patients, who were mildly or moderately affected, showed a reduced tendency to make anticipatory saccades compared with normal controls. Although there was some impairment of pursuit during a nonpredictive task as shown by an increased phase lag, a normal amount of improvement took place with a predictive task. This difference between prediction in the saccadic and pursuit system is possibly explained by an increased reliance on a visual input by parkinsonian patients which prevents them making use of verbal instruction to generate anticipatory (eye in advance of target) saccades. Improvement of pursuit with a predictive target track is possible in such patients since normally in these circumstances the target is mainly followed rather than anticipated. The metrics of saccadic and smooth pursuit eye movements (saccadic velocity and pursuit turnover velocity) were normal.

INTRODUCTION

Tracking experiments, a fruitful approach to the study of movement strategies in Parkinson's disease, typically requires the subject to follow, with their eyes or limb, a target displayed on a screen which either moves in a regular or an irregular pattern. In normal subjects target movement in a regular pattern enables predictive tracking of the eye or limb to take place (Noble et al., 1955). This requires the formation of a mental representation of the target path, followed by the design of a motor program to give a suitable output. As a result of this, the lag and error between the eye or limb and the target is reduced and the subject's tracking performance is therefore improved. In some circumstances the eye or limb may actually precede the target. In several papers, Flowers (1976, 1978a, b) has shown a number of hand-tracking abnormalities in Parkinson's disease, particularly in those experiments in which a predictive motor strategy was required. In a subsequent paper, however, Flowers and Downing (1978) failed to demonstrate a predictive deficiency in an ocular motor (OCM) tracking experiment and, therefore, concluded that only the limbs showed a predictive abnormality in Parkinson's disease. This seemed to us an unexpected
finding since it would imply that the neural organization of predictive movements for eye and limb functions differently in Parkinson's disease, in contrast to other clinical conditions. For example, patients with prefrontal lesions exhibit defects in high level motor programming which are equally present in the OCM and somatic motor systems (Luria et al., 1966; Guitton et al., 1982). In addition, Flowers and Downing only examined one aspect of ocular motor behaviour, the smooth pursuit system. We considered that the saccadic ocular motor system should be assessed separately because of important functional differences which exist between these two ocular motor subsystems.

In this paper we report the results of tests of both saccadic and smooth pursuit eye movements in mild to moderately affected parkinsonian patients compared with age-matched normal controls. It will be shown that these patients, although having a relatively normal basic oculomotor performance for their age (for instance, saccadic velocity and smooth pursuit configuration), displayed abnormalities of ocular motor prediction similar to those which have previously been reported in the limbs. A preliminary account has already appeared (Bronstein and Kennard, 1984).

MATERIALS AND METHODS

Clinical Material

Eight patients with idiopathic Parkinson's disease were selected for the study. They were mild to moderately affected (Hoehn and Yahr, grade I–III) and clinically did not have signs or symptoms of dementia or coexistent cerebrovascular disease (see Table 1). Their mean age was 65 years (range 53–70 years) as was that of the control group of 8 age-matched normal subjects (range 50–82 years), who were hospital patients with no neurological abnormality, plus one medical secretary.

Recording Techniques

Eye movements were recorded by the infrared reflection technique (Stark et al., 1962). This system provided linear recordings over a range ±20 deg with a bandpass of 300 Hz. Head movements were

<table>
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<th>Subject</th>
<th>Sex</th>
<th>Age (yrs)</th>
<th>Tremor</th>
<th>Rigidity</th>
<th>Bradykinesia</th>
<th>Drugs</th>
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U = unilateral. B = bilateral.
recorded by means of a light helmet, worn by the subject, which was attached to a low torque potentiometer via a rigid rod. The visual target was a white spot of light (30 min of arc), reflected onto a curved white screen placed 150 cm away from the subject. Target, eye and head positions were recorded on paper by an ink-jet recorder (Mingograph), and onto magnetic tape for computer analysis. Gaze position (eye position in space) was derived off-line by adding eye and head position signals in a differential amplifier.

Subjects performed saccadic and smooth pursuit experiments in one session. The experiments were performed with the head motionless and then repeated while the subject was encouraged to move his head.

**Saccadic Experiments**

The target moved in a square wave step function across the screen in the horizontal plane in three different conditions.

- **Random saccades (RS).** The target moved randomly between 9 different positions on the screen (1 central and 4 on each side) generating 8 possible target amplitudes, 5, 10, 15, 20, 25, 30, 35 and 40 deg. The time interval between target displacements was randomized between 0.6 and 1.2 s. The subjects were told that the target would jump randomly on the screen and were asked to follow it as accurately as possible. The test duration was 50 s generating 55 steps.

- **Hidden predictive saccades (HPS).** This protocol consisted of a sequence of 20 regular target displacements which, without any warning, appeared in the middle of a random sequence. The target moved symmetrically 30 deg from 15 deg left to 15 deg right and back, with a fixed time interval of 0.9 s. Test duration and instructions to the subject were identical to RS.

- **Predictive saccades (PS).** This protocol consisted of 33 regular 30 deg target displacements between 15 deg left and 15 deg right, with a fixed time interval of 0.9 s (0.55 Hz). Pilot studies and experiments in the literature (Stark et al., 1962) have shown that this time interval regularly produces predictive saccades in normal subjects. The subjects were told in advance that the target, in contrast to the previous test they had undergone, would move regularly on the screen, jumping between two fixed points. No instruction as to whether or not they should try to predict or anticipate the target was given. The duration of this test was 30 s.

For eye-head coordination experiments, time intervals between target displacements were 60 per cent longer in order to allow complete head stabilization before a new target step took place.

**Analysis of results.** Eye saccadic velocity was measured with computer software described elsewhere (Smith et al., 1981) and included all saccades from RS, HPS and PS tests, except those produced in advance of the target displacement (anticipatory saccades). The computer program generates an amplitude/peak velocity plot of saccades and fits an exponential curve to the data. The asymptote of the peak velocity (k) is used to express saccadic velocity.

The latency of saccades produced in RS was measured by the computer. The latency of each saccade occurring during HPS and PS were determined by hand from the chart recordings since the computer program would only measure saccades produced after a target displacement had taken place.

Gaze displacement during head movement was measured by the same computer program as described for eye saccades. In addition, the latencies of head movements occurring during combined eye-head experiments were measured by computer.

**Smooth Pursuit Experiments**

The target moved horizontally with a constant peak amplitude of 30 deg, under two different conditions:

- **Random smooth pursuit (RSP).** The target moved sinusoidally in an unpredictable manner at one of five different frequencies, 0.08, 0.16, 0.32, 0.48 and 0.65 Hz. One frequency was used for a random period of between 1.3 and 2.6 s, after which it randomly changed to a different frequency. Two stimulus sequences of 50 s each were presented. The subjects were instructed to follow the target with their eyes as accurately as possible and were informed that it would be moving irregularly and at a variable speed.
Predictive smooth pursuit (PSP). Identical target frequencies were then individually presented. Two sequences of 45 s each were used; the first sequence continued for 22 s at a target frequency of 0.08 Hz (approximately 2 cycles), and then the second for an identical period at 0.16 Hz (approximately 6 cycles). In the second sequence three 15 s periods, with target frequencies of 0.32 Hz (5 cycles), 0.48 Hz (8 cycles) and 0.65 Hz (10 cycles), respectively, completed the test. The subjects were informed that the target would move regularly and the experimenter told them during the sequences when the target frequency was changed. No instruction was given as to whether or not they should anticipate the target.

Analysis of results. Phase error of smooth pursuit was measured by hand for two target frequencies, 0.32 and 0.65 Hz. Their zero velocity points (maxima and minima) of both smooth pursuit eye movement and of target movement were determined in saccade-free regions. The difference between eye and target position, expressed in degrees, corresponded to the phase error, typically phase lag. Phase measurements were made for the eye, gaze and head in RSP and PSP. Approximately 7 (range 3 to 15) half cycles were measured for each condition.

Turnover velocity of the smooth pursuit system was measured by computer as described elsewhere (Bittencourt et al., 1982). In brief, the program simultaneously measures eye and target velocity and, by comparing the two, is able to identify whether smooth pursuit eye movements or catch-up saccades are being used to track the visual target. The data are sampled at 256 Hz and a plot is produced of the percentage of time spent in actual smooth pursuit during tracking as a function of target velocity. Normally, as the speed of the target increases, eye tracking becomes more and more saccadic so that less time is spent in smooth pursuit. The target velocity at which the percentage of time spent in smooth pursuit drops by 3 dB, is the turnover velocity and provides a measure of the point at which smooth pursuit breaks down into mainly saccadic tracking. Gaze movements were treated in a similar manner by computer analysis.

A computer program (BMDP2V) for analysis of variance and covariance with repeated measures was used for statistical analysis. Comparisons between groups (patients and controls) under different test conditions (eye-gaze, 0.3–0.6 Hz, random-predictive) were made and interactions between these various groups and conditions were also available. Other statistical tests used are mentioned in the Results.

RESULTS

Saccadic Movements

Random saccades. Latencies of saccades were slightly but significantly prolonged in the parkinsonian patients ($F = 6.96, P = 0.02$); interaction between eye and gaze conditions were not significant. The latency of head movements was prolonged ($t = 2.23, P < 0.05$; Student’s t test). No differences were found in the velocity for either eye or gaze (Table 2).

| TABLE 2. MEAN LATENCY AND PEAK VELOCITY OF SACCADES IN PARKINSONIAN PATIENTS AND NORMAL CONTROLS |
|------------------------------------------------------|------------------------------------------------------|------------------------------------------------------|------------------------------------------------------|------------------------------------------------------|
| Latency (ms) ± SD | Eye | Gaze | Head | Peak velocity (deg/s) ± SD | Eye | Gaze |
| Parkinsonian     | 221 ± 24* | 221 ± 24* | 328 ± 41* | 612 ± 125 | 563 ± 104 |
| Controls         | 185 ± 21 | 193 ± 24 | 272 ± 57 | 645 ± 141 | 543 ± 114 |

* $P < 0.05$. 
EYE MOVEMENTS IN PARKINSON’S DISEASE

Predictive saccades. Figs 1 and 2 show examples from the recordings of a patient and a normal control during the HPS and PS experiments. During the HPS sequence (fig. 1), both subjects appear to identify the repetitive pattern and produce a few predictive saccades. During the PS sequence (fig. 2), in which the subjects had been told in advance that the target would move regularly between two points, noticeable differences appeared between the two subjects. The normal subject tended consistently to reduce his saccadic latency or even anticipate the target, whereas the patient only irregularly made anticipatory saccades. Occasionally, a patient would ‘freeze’ and miss a couple of target displacements as shown in the figure. Only 2 patients showed this phenomenon, each on one occasion. It is interesting that it only occurred in the PS run and never in the HPS or RS run, in spite of the fact that RS sequences were of longer duration and included shorter interstimulus intervals than PS sequences.
In fig. 3 a plot showing the evolution of mean saccadic latencies for all the subjects during RS, HPS and PS sequences is presented. The latency is considered positive when the response follows the stimulus and negative when the response occurs before the stimulus (anticipation). During the regular target displacements in the HPS sequence, both groups progressively reduced their latencies. There was a significant correlation between trial (saccade) number and latency (Kendall correlation coefficient: controls, $r = 0.59$, $P < 0.01$; patients, $r = 0.33$, $P < 0.05$).

![Fig. 3. Sequential analysis of mean saccadic response time (latency) for parkinsonian patients (open circles, broken line) and normal controls (filled circles, continuous line) in random (RS), hidden predictive (HPS) and predictive (PS) tests. In the RS the mean latency of patients is slightly prolonged, but during HPS it can be seen that the latency of both groups gradually shortens with successive cycles. In PS the latency rapidly shortens in both groups, but whereas normal controls show a plateau mean response time of approximately $-120$ ms (anticipatory saccades), the patients are unable to improve beyond the response time found in HPS. Vertical bars represent $\pm 1\text{SD}$.](image)

However, the most prominent differences occur during the PS sequence where it can be seen that mean latencies are negative (anticipatory) in controls and positive in patients. There was a significant difference ($z = 1.96$, $P = 0.05$ Mann-Whitney test) between the saccadic mean latencies of patients ($107 \pm 181$ (SD) ms) and controls ($-116 \pm 220$ ms) during the PS sequence. During the predictive sequence in the HPS run the percentage of saccades which are anticipatory is very similar in both groups (controls: 23%, patients: 22%). During the PS run the patients marginally increased this value to 29 per cent, in marked contrast to the controls in whom 60 per cent of saccades became anticipatory; this interaction between conditions (HPS and PS) and subject groups was statistically significant ($F = 5.62$, $P = 0.03$) (fig. 4).

**Smooth Pursuit Movements**

*Turnover velocity analysis.* No significant differences were found between the two groups (fig. 5). Both groups increased their turnover velocity in the predictive mode ($F = 27.93$, $P < 0.001$). In addition it was found that turnover velocity gaze values
were higher than eye alone ($F = 5.00$, $P = 0.04$), but this was made more evident in the predictive mode (interaction eye-gaze/random-predictive: $F = 5.45$, $P = 0.03$). This improvement in the smooth pursuit turnover velocity during the head-free condition is thought to be due to an increased efficiency of the catch-up saccades generated by the latter condition and has been discussed elsewhere (Bronstein and Kennard, 1984).

**Phase lag analysis.** Fig. 6 shows a recording from a normal subject and a patient during random and predictive conditions, in which a few cycles of the target moving at the highest frequency used are shown. Two particular features may be seen; first,
Fig. 6. Examples of eye movements of a normal control and parkinsonian patient during random (upper) and predictive (lower) smooth pursuit. In each pair of traces the target is above and left eye below. The zero velocity points of the target (solid vertical line) and eye (interrupted vertical line) are indicated. The difference in position expressed in deg between the eye and the target indicates the phase error which can be seen to be greater in the parkinsonian patient than the normal control. The phase error is reduced during predictive pursuit compared with random pursuit in both subjects.

The morphology of the eye movement in the two subjects is rather similar. Both show impaired pursuit containing frequent catch-up saccades. Secondly, the main difference between the two subjects' performance is in the phase error, the patient showing phase lags approximately double those of the control. In comparison during the predictive sequence it can be seen that both subjects considerably reduce their phase lag. During head-free tracking (fig. 7), although head movement itself was not delayed in relation to the target, gaze still showed an increased phase lag. The mean and standard deviations of the phase error in these different conditions are plotted in fig. 8. The statistical analysis was performed for eye and gaze together and separately for head. The eye and gaze phase lags are significantly larger in the patients compared to controls ($F = 6.99$, $P = 0.02$). Reduction of phase lag in the predictive condition was statistically significant ($F = 45.88$, $P < 0.001$). However, this reduction was equally evident in the two groups, there being no significant interaction between the random-predictive conditions and groups. The phase lag found at the higher frequency are, as expected, significantly greater in both groups ($F = 106.83$, $P = 0.001$).

The phase lag of the head, in contrast to eye and gaze, showed no significant difference between the two groups. A greater mean phase lag of the head was found at the higher frequency ($F = 33.84$, $P < 0.001$). There was a significant reduction in phase lag during the predictive sequence ($F = 30.02$, $P < 0.001$). This reduction was present in both groups to the same extent but there was no significant random-predictive group interaction.
FIG. 7. The eye, gaze and head movements of a parkinsonian patient during predictive head-free smooth pursuit. The zero velocity points of the target (solid vertical line) and gaze and head (interrupted vertical line) are indicated. There is very little phase error of the head which on some occasions is leading (i.e., anticipating) the target, whereas the gaze still shows a considerable phase lag.

FIG. 8. The mean and standard deviation smooth pursuit phase error of eye, gaze and head for parkinsonian patients (circles) and normal controls (squares) during pursuit of a target of frequency 0.3 and 0.6 Hz. The target moved in the random (filled symbols) or predictive (open symbols) mode.

**DISCUSSION**

The main finding of this study of eye movements in Parkinson’s disease is that under certain predictable conditions there is a reduced tendency to make anticipatory saccades. Surprisingly, however, two earlier studies did not report
abnormalities of prediction in the parkinsonian ocular motor system (Melvill Jones and Mandl, 1976; Flowers and Downing, 1978). This was taken by Flowers and Downing to indicate that the organization of motor activities involving predictive movements may be different for the eye and limbs, each having its own separate predictor mechanism which is differentially affected by Parkinson's disease. This apparent contradiction with our results is partly due to the fact that they based their conclusions solely on smooth pursuit experiments and did not investigate the saccadic system. We have also found an improvement in predictive smooth pursuit in parkinsonian patients shown by the reduction in phase lag and increase in turnover velocity.

It is in the saccadic system that we have shown significant abnormalities in the predictive capacity of parkinsonian patients. Moreover, the experimental design permits us to make some observations upon the nature of this predictive defect. During the hidden predictive saccade sequence both parkinsonian patients and controls are initially unaware of the regularity of the target trajectory but, as shown by the reduction in saccadic latency and by the increasing number of anticipatory saccades, gradually recognize its predictable nature. Despite this, in the predictive saccade (PS) test, parkinsonian patients, when compared with controls, produced far fewer anticipatory saccades and did not further reduce their mean saccadic latency.

Before the PS sequence all the subjects were informed about the regularity of the target displacement, but special care was taken not to suggest whether they should anticipate or follow it. Under similar test conditions to the ones we used, normal subjects were found to have saccadic latencies which were critically influenced by the type of verbal instruction (Polidora et al., 1957). Those subjects instructed to 'anticipate' had negative (anticipatory) mean latencies of about 100 ms and those told to 'follow' the target produced their saccades 100 ms after the target jump. In the absence of any specific instruction given in the PS sequence other than to inform the subjects about the regularity of the target movement, the trend of our normal subjects was to anticipate the target to a degree (60% anticipatory) similar to previously published studies using comparable test conditions (Stark et al., 1962; Horrocks and Stark, 1964). Parkinsonian patients in our experiments tended to 'follow' the target rather than regularly anticipate it, which probably reflects an increased dependence on the visual stimulus. Their mean latency values of about 100 ms during PS are compatible with a predictive process in which reaction times are only reduced to the lower limit. In this situation information contained in the visual target can still induce modifications in the saccadic response (Horrocks and Stark, 1964).

Previous work on manual tracking of a continuously moving target has shown that parkinsonian patients are able to reduce tracking lag when the target path becomes regular and therefore predictable (Flowers, 1978a; Bloxham et al., 1984; Day et al., 1984). Experiments which involve discontinuous (i.e., ballistic) limb movements in tracking a step function target (Flowers, 1976, 1978b), however,
showed a failure of prediction. Similarly, Bloxham et al. (1984) have shown that parkinsonian patients are unable to make use of advance information in performing a reaction time test. These findings, as well as the results in our PS experiment, emphasize the difficulty parkinsonian patients have in making movements of eye or limb in advance of the visual target. This abnormality reflects an increased reliance on visual input which has previously been described (Cooke et al., 1978; Flowers, 1978b; Stern et al., 1983), and is reminiscent of the much improved locomotor function which occurs clinically when such patients are provided with a powerful visual input, for example a striped floor (Martin, 1967).

Two possible pathophysiological explanations for this disturbance of prediction are worthy of consideration. The first arises from the similarity between parkinsonian patients and patients with frontal lobe lesions who show a difficulty in utilizing verbal instructions in the construction of motor programs (Luria, 1969). An example of this in relation to the ocular motor system is shown in experiments on patients with well-defined surgical lesions in the prefrontal region who, after relevant verbal instructions, were unable to generate saccades in a direction opposite to a sudden target displacement (Guitton et al., 1982). These patients therefore followed the visual stimulus rather than the verbal instruction. Several studies of cognitive and motor function in Parkinson's disease have already shown abnormalities similar to those found in patients with frontal lobe lesions (Denny-Brown, 1968; Morel-Maroger, 1977; Figini and Bronstein, 1981; Bowen et al., 1972; Lees and Smith, 1983). These frontal lobe type abnormalities found in Parkinson's disease may be due to dysfunction of the ascending dopaminergic mesocorticolimbic pathway projecting to the frontal cortex (Javoy-Agid and Agid, 1980).

A second possible pathophysiological explanation for the failure of our parkinsonian patients to make anticipatory saccades during the predictive sequence is to be found in recent experimental studies of cells in the pars reticulata of the substantia nigra in primates. This may also explain the other saccadic abnormality we found which was a prolonged latency, previously reported in the ocular (Shibasaki et al., 1979; Teräväinen and Calne, 1980a, b; Shimizu et al., 1981) and somatic motor systems (Kennard et al., 1982; Evarts et al., 1981). These experiments have shown a clear relationship between the basal ganglia and the major saccadic control centres, the frontal eye fields (area 8) (Kunzle and Akert, 1977) and the superior colliculus (Hikosaka and Wurtz, 1983). The dopaminergic cells in the pars compacta of the substantia nigra project to the caudate nucleus and putamen (striatum), which in turn receives a projection from the frontal eye fields, particularly to the posterior caudate nucleus. One of the two major outputs from the striatum is to the pars reticulata of the substantia nigra, some of whose cells project to the intermediate and deep layers of the superior colliculus (Jayaraman et al., 1977). These cells have been found to reduce their discharge rate both in response to an appropriately located visual target and to a remembered target (Hikosaka and Wurtz, 1983). Since they appear to be gabaergic inhibitory cells (Vincent et al.,
a decrease in discharge rate would reduce tonic inhibition, and hence increase the excitability of collicular cells. These cells have been found to exhibit a burst of discharge before saccades of a particular direction and amplitude (Wurtz and Goldberg, 1972). A disturbance of the dopaminergic input to the corpus striatum, as occurs in Parkinson's disease, may lead to an abnormal output to the substantia nigra pars reticulata resulting in reduced facilitation of collicular cells leading to prolonged latencies. Since the substantia nigra pars reticulata cells also discharge prior to saccades to a remembered target, an abnormal input to these cells could lead to an impaired ability to make anticipatory saccades, which are in essence saccades to a remembered target. It is of interest that DeJong and Melvill Jones (1971) showed that patients with Parkinson's disease took a longer time than controls to make saccades back and forth between two targets which are continuously illuminated. In this case the saccade must be initiated by something other than the onset of a visual target, again possibly analogous to saccades to remembered targets.

Examination of the smooth pursuit system in our patients showed an increased phase lag, a feature previously described for hand tracking experiments in Parkinson's disease and in monkeys with caudate lesions (Stark, 1968; Bowen, 1969; Bowen et al., 1975; Flowers, 1978a). Since smooth pursuit operates by continuously detecting and subsequently correcting retinal error (i.e., a closed-loop system), it could be argued that a prolonged reaction time at one or more points during the central processing for such a tracking task may well produce delay (lag) between target and eye. An interesting finding, however, was that the phase lag of the head during smooth pursuit was not significantly increased, which indicates its unique form of motor control. With the head free to move during visual tracking of a target the only important factor in the maintenance of foveation of the target is the retinal or gaze error. Indeed changes in head gain or phase can be enormous and yet gaze gain, phase or accuracy will remain unchanged (Lanman et al., 1978; Collewijn et al., 1982). This is due to stabilization of the eyes on target by the vestibulo-ocular reflex despite the variable head error (Lanman et al., 1978). There therefore appears to be no need for precise detection of head error during head-free smooth pursuit and this is reflected in its large variance for both groups of subjects shown in fig. 8.

Although the absolute value of eye-gaze smooth pursuit phase lag was increased in the parkinsonian patients the percentage of reduction during regular sine wave tracking was similar in both groups. This is in agreement with previous papers (Flowers and Downing, 1978; Melvill Jones and Mandl, 1976) where preserved predictive behaviour in the smooth pursuit system of parkinsonian patients was found.

This apparent difference in predictive capacity between the saccadic and smooth pursuit ocular motor systems in parkinsonian patients requires some explanation. We believe that the normal marked dependence of smooth pursuit on the presence of a visible moving target is one of the clues to this problem. Target anticipation in smooth pursuit (i.e., eye in advance of target) occurs only exceptionally, as reflected
in the constant delay (lags present between target and eye position), even amongst normal subjects (fig. 8) (Lisberger et al., 1981). Moreover, it has long been known that normal smooth pursuit eye movements are exceedingly difficult to generate except in response to a slowly moving target. If, as we have suggested earlier, the predictive deficit seen in the saccadic experiments is at least partly due to an increased reliance on visual input, then we would not expect a similar deficit in the smooth pursuit system since a predictive response does not involve anticipation to the same degree as in the saccadic system. The target is constantly present and is being pursued.

A final point of interest emerging from our experiments is that, apart from the ‘high level’ abnormalities of the ocular motor system concerned with prediction, the metrics of saccadic and smooth pursuit movements were relatively unimpaired. In the patients studied, saccadic peak velocities were normal and the turnover velocity of the smooth pursuit system, although marginally reduced, was not significantly different from the normal controls. This would imply that the peripheral neuromuscular ocular plant, and the brainstem ocular motor nuclear and prenuclear neurons were functioning normally. This relative normality contrasts with some of the earlier literature and is due to at least two factors. First, these studies included severely affected patients (Shibasaki et al., 1979; Corin et al., 1972; Teräväinen and Calne, 1980a, b), some of whom had other associated brain lesions (White et al., 1983), or patients with undefined disease severity (DeJong and Melvill Jones, 1971; Melvill Jones and Mandl, 1976; Shibasaki et al., 1979). Secondly, there was sometimes a disparity between the ages of patients and controls (Shibasaki et al., 1979; Shimisu et al., 1981). It is now generally agreed that performance of the ocular motor system, in particular smooth pursuit, is highly dependent upon age (Sharpe and Sylvester, 1978). Spooner et al. (1980) have recently shown that a group of patients with vertebrobasilar insufficiency who were administered a battery of eye movement tests could be considered either as abnormal, if a control group of unselected age was used, or as normal if compared with an age-matched control group. Consideration of the literature on ocular motor disturbances in Parkinson’s disease in relation to these points results in the conclusion that mild or moderately affected patients, suffering only from idiopathic Parkinson’s disease and compared with a properly age-matched control group, show minimally impaired pursuit or slowed saccades and our results would confirm this view. The possibility that more severely affected patients could show a more severe degree of impairment obviously remains, but in our opinion requires further validation.

In conclusion, this study of eye movements in parkinsonian patients has shown significant abnormalities in the predictive behaviour of the saccadic system which may relate to dopamine deficiency either in the frontal lobe, or as a result of abnormal interactions between the basal ganglia and superior colliculus. As similar disturbances have previously been found in hand tracking experiments, our results do not support the hypothesis that the eye and limb predictive mechanisms are differentially affected by the disease.
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