Oculomotor Function in Patients with Parkinson's Disease

M. S. CORIN, TERESITA S. ELIZAN AND M. B. BENDER

Department of Neurology, Mount Sinai School of Medicine, New York, N.Y. (U.S.A.)

(Received 22 June, 1971)

INTRODUCTION

That patients with Parkinson's disease may have abnormalities of oculomotor function is fairly well known by clinicians. Limitation of gaze, especially in the vertical plane, and inadequate convergence have been described previously (Krebs 1925; Bielschowsky 1935).

The present report is of a systematic investigation of oculomotor dysfunction among patients with this disease to determine: (1) the frequency with which such abnormalities occur among these patients; (2) the nature and severity of these defects; (3) the electrooculographic (EOG) recording of "typical" patients; and (4) the effects of levodopa on oculomotor defects. A preliminary report of the results has already appeared (Corin, Elizan and Siegel 1970).

CASE MATERIAL AND METHODS

Seventy patients with Parkinson's disease seen at the Mount Sinai Hospital, New York City, and 30 age-matched controls were studied specifically for oculomotor function. The 30 controls were hospitalized for general surgical conditions, between the ages of 52 and 79 years, with no evidence of neurologic or ophthalmologic disease.

All patients and controls were examined in a standard fashion, beginning with inspection of eyelids, face, and head in the resting position. Eye movements were then evaluated in the following order: (a) spontaneous eye movements as the patient looked about his surroundings; (b) on command to look up, down, right, and left; (c) on command to look at a specific target (i.e., ceiling, floor, specific object on right and left sides); (d) on command with visual gesture (i.e., giving the command, "Look that way", while simultaneously pointing forcefully in the desired cardinal directions of gaze); (e) on visual pursuit of a moving target, including convergence; (f) on oculocephalic maneuver with visual fixation (with the patient fixating a visual target directly in front of him, his head was passively turned to the extremes in the horizontal...
and vertical planes). On all of these tests, the following factors were evaluated as compared to the expected normal: the fullness of extent of gaze, the speed of initiation and performance of the movement, the smoothness of the movement, the ability to maintain gaze in the desired direction, and the relationship of lid, eye and head position. Optokinetic nystagmus (OKN) was tested in the horizontal and vertical planes, specifically noting its frequency, regularity and amplitude and the eye position during the evoked nystagmus. Associated movements on eyelid closure, and pupillary reflex to light stimuli and on attempted convergence, were also noted.

The performance of patients and controls on each test was rated from 0 (normal) to 3 (severely abnormal). The composite degree of total oculomotor function of each patient was graded as follows: 0 = no oculomotor abnormality; 1 = mild defect(s) on any specific test(s); 2 = moderate to severe defects of gaze on directional command only, on command to look at a specific target, on command with visual gesture, and/or on OKN, but no defects on visual pursuit, oculocephalic maneuver with fixation, or Bell’s phenomenon; 3 = moderate to severe defects as in Grade 2, and on visual pursuit, but not on oculocephalic maneuver or Bell’s phenomenon; 4 = moderate to severe defects as in Grade 3, and on oculocephalic maneuver or Bell’s phenomenon.

For each patient, the degree of oculomotor dysfunction was then compared with the general severity of Parkinson’s disease (Grades I through IV, Mones and Elizan 1969).

A short term (1–8 months) study was conducted of the effect of levodopa on oculo-motor function. Results were compared to any modification of other abnormalities considered secondary to Parkinson’s disease.

Electro-oculograms were recorded on patients with each grade of oculomotor dysfunction noted clinically, as well as on controls. Eye movements were recorded on testing identical to that given above. The technique has been described in detail previously (Atkin and Bender 1968).

RESULTS

Abnormalities of eye movement

The results as tabulated in Table 1 indicate that among the patients in this study the most sensitive test of eye movement disturbance was gaze on command, followed in decreasing order of sensitivity by ocular response on command to look at a specific target, on command to look and directed by a visual stimulus (gesture), optokinetic stimulations, on command to pursue a moving visual target, on eyelid closure (Bell’s phenomenon), head turning with or without visual fixation of a stationary target. There were occasional patients, however, who had jerky or saccadic eye movements (“cog wheeling”) on visual pursuit of a moving target, but performed well on gaze to command. There were a few others who had abnormalities on OKN but did well on other tests, such as gaze to command.

A surprisingly high percentage (75%) of these Parkinsonian patients had some degree of oculomotor dysfunction, ranging from mild, irregular, saccadic eye movements with dissociation in sequences of eye-head movements; the head moved ahead.

OCULOMOTOR FUNCTION IN PATIENTS WITH PARKINSON’S DISEASE

Table 1

SUMMARY OF OCULOMOTOR DEFECTS IN PARKINSONIAN PATIENTS

<table>
<thead>
<tr>
<th>Test</th>
<th>Severe Abnormality</th>
<th>Moderate Abnormality</th>
<th>Mild Abnormality</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Command</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>up</td>
<td>25*</td>
<td>12</td>
<td>15</td>
<td>18</td>
</tr>
<tr>
<td>down</td>
<td>17</td>
<td>19</td>
<td>14</td>
<td>20</td>
</tr>
<tr>
<td>right</td>
<td>5</td>
<td>5</td>
<td>29</td>
<td>31</td>
</tr>
<tr>
<td>left</td>
<td>5</td>
<td>5</td>
<td>28</td>
<td>32</td>
</tr>
<tr>
<td>Command to specific target</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>up</td>
<td>9</td>
<td>15</td>
<td>20</td>
<td>26</td>
</tr>
<tr>
<td>down</td>
<td>6</td>
<td>13</td>
<td>25</td>
<td>26</td>
</tr>
<tr>
<td>right</td>
<td>0</td>
<td>2</td>
<td>22</td>
<td>46</td>
</tr>
<tr>
<td>left</td>
<td>0</td>
<td>2</td>
<td>21</td>
<td>47</td>
</tr>
<tr>
<td>Pursuit</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>up</td>
<td>7</td>
<td>9</td>
<td>22</td>
<td>32</td>
</tr>
<tr>
<td>down</td>
<td>6</td>
<td>5</td>
<td>24</td>
<td>35</td>
</tr>
<tr>
<td>right</td>
<td>0</td>
<td>0</td>
<td>26</td>
<td>44</td>
</tr>
<tr>
<td>left</td>
<td>0</td>
<td>0</td>
<td>26</td>
<td>44</td>
</tr>
<tr>
<td>OKN</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>up</td>
<td>17</td>
<td>4</td>
<td>13</td>
<td>36</td>
</tr>
<tr>
<td>down</td>
<td>6</td>
<td>9</td>
<td>3</td>
<td>52</td>
</tr>
<tr>
<td>right</td>
<td>1</td>
<td>1</td>
<td>6</td>
<td>62</td>
</tr>
<tr>
<td>left</td>
<td>1</td>
<td>1</td>
<td>8</td>
<td>60</td>
</tr>
<tr>
<td>Oculocephalic maneuver</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>up</td>
<td>4</td>
<td>5</td>
<td>13</td>
<td>48</td>
</tr>
<tr>
<td>down</td>
<td>1</td>
<td>3</td>
<td>13</td>
<td>53</td>
</tr>
<tr>
<td>right</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>65</td>
</tr>
<tr>
<td>left</td>
<td>0</td>
<td>0</td>
<td>5</td>
<td>65</td>
</tr>
<tr>
<td>Convergence</td>
<td>12</td>
<td>5</td>
<td>10</td>
<td>43</td>
</tr>
<tr>
<td>Bell’s phenomenon</td>
<td>5</td>
<td>4</td>
<td>5</td>
<td>56</td>
</tr>
<tr>
<td>Pupils</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>near reflex</td>
<td>7</td>
<td>4</td>
<td>8</td>
<td>51</td>
</tr>
<tr>
<td>light reflex</td>
<td>2</td>
<td>0</td>
<td>6</td>
<td>62</td>
</tr>
</tbody>
</table>

* Figures represent No. of patients. Total = 70.

of the eyes (eye movement normally occurs first), to complete absence of gaze in one or more directions.

The most conspicuous defect of conjugate eye movements was impaired vertical gaze, with abnormalities of upward gaze slightly more frequent than abnormalities of downward gaze. Every patient found to have any eye movement disturbance had difficulty with gaze on command to “Look up” (52 of 70, with 37 of these being moderate to severe), except for 1 patient in whom severe limitation of convergence was the only observed defect.

Abnormalities of upward gaze were the most prominent of all tests. On visual
pursuit, for example, 38 of the 70 patients had defects of upward gaze; in 16 of these the defects were moderate to severe. There were, however, 9 patients in whom downward gaze was more impaired than upward gaze; in 4 the difference was rather significant. Horizontal gaze deficits were less in degree and frequency than in the vertical plane. There were, nevertheless, 39 of 70 who had abnormalities of horizontal gaze on command but in only 10 patients were the deficits considered moderate to severe. They were occasionally found only on EOG. Many patients had delayed initiation of gaze in one or more directions, while others had slowing or jerkiness of movements themselves. Often, movements in the vertical plane were slower and jerkier than in the horizontal, even in the presence of full excursions. In some cases, there was difficulty in maintaining conjugate gaze in any one extremely deviated position; there was always a slow drift back toward the midline in these patients.

Approximately half of the patients had abnormalities of optokinetic nystagmus, again most commonly in the vertical plane. Most of these defects were considered to be moderate to severe. This varied from total absence of a response in one or more
OCULOMOTOR FUNCTION IN PATIENTS WITH PARKINSON'S DISEASE

255
directions, including nystagmus and pursuit, to a slight difference in the response in one direction as compared to another.

Twenty-two of the 70 patients showed dysfunction in eye movement on head-turning or bending during optic fixation, although in most instances these abnormalities were mild. The associated upward movement of the globes on eyelid closure ("Bell's phenomenon") was impaired in 14 patients and it was either totally absent or severely abnormal in 9 patients.

Convergence was impaired in 27 of the 70 patients with 17 showing moderate to severe abnormality.

About two-thirds of the patients had some type of eyelid dysfunction. Twenty-six had retraction and 10 had ptosis of the superior eyelid; 50 of the patients had blepharospasm, and in 28 of these it was moderate to severe.

Pupillary abnormalities were noted in 19 patients, all of whom had deficits of reaction on attempted convergence; 8 of these 19 had, in addition, abnormalities of the direct and indirect light reflex.

Abnormalities of oculomotor function among the controls were infrequent and mild in degree. Slight limitation of upward gaze, mild lid retraction and slight dissociation of OKN elicited in the vertical plane were occasionally observed.

The composite degree of total oculomotor dysfunction was compared with the general severity of Parkinson's disease (Mones and Elizan 1969) (Table 2). Oculomotor dysfunction tended to be worse in the more severely affected patients. It should be noted, however, that the oculomotor deficits tended to be more marked than the generalized condition, and that in a number of patients, their degrees of severity were poorly correlated.

<table>
<thead>
<tr>
<th>General dysfunction grading</th>
<th>Oculomotor dysfunction grading</th>
</tr>
</thead>
<tbody>
<tr>
<td>IV</td>
<td>0* 1 3 2 3</td>
</tr>
<tr>
<td>III</td>
<td>2 3 12 1 7</td>
</tr>
<tr>
<td>II</td>
<td>11 4 5 2 5</td>
</tr>
<tr>
<td>I</td>
<td>3 3 1 1 0</td>
</tr>
</tbody>
</table>

* Figures represent number of patients in each category. Total = 70.

The effect of levodopa

The effects of levodopa on oculomotor function were evaluated in 29 patients. The duration of drug treatment varied from 1–8 months. The highest dosage level varied from 1–5 g per day. Six of these patients showed moderate degrees of improvement in their generalized condition, 14 had mild degrees of improvement, 2 slight improvement, and in 7 there was no change. Only 2 of these patients with improvement induced by levodopa medication showed any change whatsoever in their oculomotor function. One had a slight improvement in the degree and facility of upward gaze, in the presence

Fig. 3. Same patient as in Fig. 2 except that vertical eye movements are recorded. Again there is jerkiness of spontaneous and pursuit movements and some inability to maintain upward gaze to command, somewhat more marked than for horizontal gaze.

Fig. 4. EOG of patient with moderate disease. There is slowness of movements to command, somewhat more marked to the left. Extent of gaze is somewhat more full on gaze to a specific target and gaze with visual gesture, but there is jerkiness in performance and an overshoot on gaze with visual gesture, somewhat more noticeable on relaxation. In the upper two tracings, eye movement precedes head movement, but the opposite is true in the lower tracing. At rest there is an oscillatory side-to-side movement induced by holding the head stationary in this patient with a side-to-side head tremor.

of a mild degree of improvement in his general condition. The second patient is of some interest, in that 2 months after the institution of levodopa therapy he showed no change in oculomotor status despite a noticeable improvement in his general condition. After 4 months of levodopa therapy he maintained the same degree of general improvement, and finally showed a definite improvement in oculomotor function.
OCULOMOTOR FUNCTION IN PATIENTS WITH PARKINSON'S DISEASE

"LOOK DOWN"  "LOOK UP"

VERT. O.D.

HEAD

"LOOK AT THE CEILING"  "LOOK AT THE FLOOR"

"LOOK OVER THERE"
(WITH A GESTURE)

Fig. 5. Same patient as in Fig. 4. Note rather markedly impaired gaze to command. The extent is improved on subsequent tests but there is still jerkiness and very slow movement. Head movement precedes eye movement on all tests.

PURSUIT

HORIZ. O.U.

TARGET

VERT. O.D.

TARGET

1 sec

1 sec

Fig. 6. Same patient as in Fig. 4. Note saccadization of pursuit, which is somewhat more marked in the vertical plane where there is also a slight limitation in the extent of gaze.

Electro-oculograms (EOG's) of patients with varying degrees of oculomotor dysfunction (i.e., mild, moderate, or severe) were recorded and the records of 1 patient in each category plus a control, are illustrated in Figs. 1–12. In general, these records demonstrated the signs that were noted clinically, but revealed some abnormalities which were not clinically apparent.

Fig. 7. Same patient as in Fig. 2 (patient with mild disease). Note poor OKN with targets from left to right (nystagmus to left), which is more marked at higher target speed in the lower figure. These abnormalities were not clinically apparent.

Fig. 8. Same patient as in Fig. 4 (patient with moderate disease). Impaired vertical OKN, more marked with targets from up to down (upward nystagmus) and at higher target speed.

Fig. 9. EOG of patient with severe disease. Poor horizontal OKN. There are irregular jerks and some following in the direction of the targets is seen.

OCULOMOTOR FUNCTION IN PATIENTS WITH PARKINSON'S DISEASE

Fig. 10. Same patient as in Fig. 4 (patient with moderate disease). Relatively normal except for "plateau" effect in vertical eye movement testing.

Fig. 11. Lower tracing is Bell's maneuver of patient in Fig. 10. Compare to upsweep in Bell's maneuver of normal in upper tracing.

Fig. 12. Same patient as in Fig. 9 (patient with severe disease). Bell's maneuver elicits no obvious upward movement of eyes.

DISCUSSION

Although the literature on Parkinson’s disease contains many references to abnormalities of oculomotor function, there is little emphasis as to the types of defects, particularly as elicited by the electro-oculogram.

The method employed by us for examination of eye movements follows “hierarchies” proposed by Stenvers (1945), and Feldman and Bender (1970). Bender (1969) lists 10

steps in evaluating defects of conjugate gaze, the most sensitive being gaze to command such as, “Look up”. The least sensitive test is the oculocephalic maneuver or head movement during visual fixation. Our results were in accordance with this. Better performance on pursuit testing than on gaze to command has been previously reported in Parkinsonian patients (Bielschowsky 1935; Slatt, Loeffler, and Hoyt 1966). Bielschowsky (1935) attached localizing significance to this, arguing in favor of the anatomic localization of a lesion in the basal ganglia as the cause of the oculomotor deficit. However, many of our patients in this study had some defects on pursuit in addition to that on head turning which, according to Bielschowsky, would place the lesion nearer to the oculomotor nuclei.

In addition to evaluating the range of eye movements, factors such as smoothness of eye movement, ability to initiate and stop eye movement rapidly and to maintain extremes of deviated ocular gaze were noted. Disorders in these functions have been attributed by some to “basal ganglia disease”, because of their resemblance to phenomena of alteration in tone in other muscles, with tremor, rigidity and bradykinesia (Cogan 1956, 1964; Smith 1963, 1966). This latter view may, however, be incorrect. “Cog-wheeling” or saccadization during pursuit and other eye movements have been reported in conditions other than basal ganglia disease (Cords 1929; Savitsky and Winkelman 1947; Rodin 1964).

Some have stated that Parkinsonian patients do not have defects of gaze, but that they simply have bradykinesia, tremor or rigidity of eye muscles similar to what is found in the peripheral musculature. On this basis Bielschowsky (1935) and Cogan (1956) referred to the oculomotor disturbances in these patients as “pseudo-ophthalmoplegia”. Slatt et al. (1966) reached the same conclusion on the basis of electromyographic (EMG) studies carried out on the medial and lateral recti in Parkinsonian patients. This is, of course, only conjecture, as there is not necessarily any reason to believe that eye muscles are influenced in the same manner as peripheral muscles by basal ganglia lesions or that they may not be influenced by other lesions that may or may not affect peripheral muscles. Alternatively these abnormalities could represent defects of conjugate gaze or spasticity of conjugate gaze (Savitsky and Winkelman 1947; Rodin 1964; Bender 1969), in addition to the true limitation of gaze which we found. Dodge (1903) and Westheimer (1954) found smooth pursuit movements in normal man to be punctuated by corrective saccades of small amplitude. Saccadization of pursuit movements seen in the patients in this study may, therefore, represent an accentuation of a normal phenomenon.

The findings that as many as 75% of patients with Parkinson’s disease have some significant defect of oculomotor function is surprising in view of the lack of attention this phenomenon has received in the literature. Defects in gaze were most common in the vertical plane, upward gaze being involved to a greater extent than downward.

Of interest is that in cases of impairment of vertical gaze, whatever the etiology, upward gaze is much more frequently involved. Defects in downward gaze are rare and when they are present, occur in conjunction with paralysis of upward gaze.

Some have stated that in diffuse diseases which may affect conjugate gaze, vertical gaze is the first to be involved because there is a greater representation of the lateral gaze mechanism (Bielschowsky 1935). Toxic-metabolic disturbances, such as barbitu-
rate intoxication with narcosis or other anesthetics may impair vertical vectors before horizontal but there is no evidence for such a mechanism in Parkinson's disease (Krieger, Wagman, and Bender 1958). On the other hand, it has been noted by several observers that in both animals and man (Bender 1960; Christoff, Anderson, and Bender 1962; Bender and Shanzer 1964; Pasik, Pasik and Bender 1969a; Pasik, Pasik and Bender 1969b), bilateral brain lesions at several levels may result in defective vertical gaze, most commonly in the upward direction. The most prominent and most lasting defects of this type have been found in lesions of the brain stem, notably of the posterior commissure-pretectal region. Lesions in this location that are bilateral cause defective upward gaze, less prominently defects of downward gaze and convergence, eyelid retraction, occasionally ptosis, and abnormalities of pupillary reactions. Loss of downward gaze with impaired convergence and meiosis may result from lesions in the prerubral fields of Forel, red nucleus and ventrolateral thalamus (Nashold and Gills 1966; Nashold, Gills, and Wilson 1967). The former is similar to what has been noted in our Parkinsonian patients. It should be noted, in this regard, that in Parkinson's disease there is a diffuse pathologic process not infrequently found in the pretectal region and throughout the brain stem including the tegmentum (Greenfield and Bosanquet 1953; Forno 1966).

It is, of course, possible that the defects noted here are due solely to "basal ganglia" dysfunction. There is some evidence that pathways from the various parts of the cerebral cortex to the oculomotor nuclei pass through the basal ganglia and that these might be interrupted in Parkinson's disease (Mettler 1935, 1964). Lesions at these sites in animals, however, have produced either no defects or inconsistent defects of oculomotor function (Spiegel and Scala 1937; Mettler and Mettler 1942; Mettler 1945, 1964; Bender and Shanzer 1964; Spiegel and Szekely 1961; Kennard and Ectors 1938; Kennard 1944).

The results of EOG have already been noted above. Defects of gaze were frequently noted on this test which were not apparent clinically. Some other authors have recorded EOGs on Parkinsonian patients. Von Noorden and Preziosi (1966) found prolonged execution of 20° gaze shifts, unsteady fixation on a target, and horizontal pendular nystagmus on pursuit movements. Jones and De Jong (1970) found abnormalities in saccadic eye movements with gaze shifts of 25° and Highstein, Cohen, and Mones (1969) noted difficulties with saccadic eye movements at a variety of amplitudes of gaze shifts, particularly difficulty in acceleration, deceleration and in executing gaze shifts in one saccadic movement. It should be noted, however, that to record from a certain number of patients with Parkinson's disease and to state that there are certain characteristics on EOG typical of this disease may be misleading. As we have demonstrated, there is a broad spectrum of oculomotor defects in these patients and not simply defective saccadic, pursuit or other movements.

Blepharospasm, caused by contractions in facial muscles, and other eyelid abnormalities have been commented upon in patients with Parkinson's disease (Smith 1963, 1966). EMG studies done by Loeffler, Slatt, and Hoyt (1966) on orbicularis oculi and levator palpebrae muscles in these patients did much to elucidate what is going on in the muscles to account for some of the phenomena noted clinically, i.e., lid retraction, ptosis and blepharospasm, but failed to explain the central mechanism.
The responses of Parkinsonian patients to levodopa therapy are of some interest. Effects on oculomotor function have previously been reported, in a few patients with so-called progressive supranuclear palsy (Mendell, Chase and Engel 1970). Highstein et al. (1969) demonstrated improvement in saccadic movements in 5 Parkinsonian patients, by EOG. As noted, we found a distinct difference between the responsiveness of extraocular and peripheral musculature. Of the 2 patients who showed noticeable improvement in oculomotor function, 1 did so 2 months after a response had been observed in his peripheral musculature. This suggests that levodopa may have a totally different action upon eye movements as compared to other movements, or that effects upon the former may occur but after a longer interval. It also raises the question of whether oculomotor abnormalities in Parkinson's disease may be due to differential sensitivities of extraocular and peripheral muscles to similar stimuli.

In the light of our findings it seems appropriate to discuss the relationship of Parkinson's disease to other similar conditions. Abnormalities of oculomotor function are well known in post-encephalitic Parkinsonism (Critchley 1929; Goldbach 1929; Crow 1949). In the earlier French literature there were reports of defective vertical gaze in Parkinson's syndrome, probably of the post-encephalitic variety (Barré 1921; Duverger and Barré 1921; Alajouanine, Delafontaine and Lacan 1926). The presence of oculomotor dysfunction has, in fact, been considered by some to favor the diagnosis of post-encephalitic rather than the idiopathic condition. Although some such abnormalities were found less commonly in our patients (notably loss of convergence and strabismus), limitation of conjugate gaze was actually found much more commonly. To use these clinical findings alone, therefore, to distinguish the two conditions, may be fraught with hazard.

The syndrome of progressive supranuclear palsy, as described by Steele, Richardson and Olszewski (1964), is characterized by limitation of conjugate gaze, mostly in the vertical plane; rigidity, more prominent in axial musculature; pseudobulbar palsy, dysarthria and mild dementia. These authors staunchly held that the condition was both clinically and pathologically distinct from Parkinsonism. However, some of the features they described are seen in Parkinsonism and other authors have included patients under this heading who had more typical Parkinsonian signs (Messert and Van Nuis 1966; David, Mackey, and Smith 1968; Behrmann, Carroll, Janota and Matthews 1969; Mendell et al. 1970). Of interest is that in the pathologic anatomy of these cases there is frequently bilateral involvement of the paramedian zone of the pontine tegmentum and pretectum in addition to the basal ganglia, as is also true in post-encephalitic and idiopathic Parkinson's disease. The results of the present study indicate that limitation of conjugate gaze, thought by many to be the hallmark of progressive supranuclear palsy, cannot be used to differentiate it from Parkinsonism. Indeed, there may be a spectrum of clinical conditions with many variations between "typical" cases of each entity.

ACKNOWLEDGEMENTS

We are grateful to Drs. A. Atkin, M. Feldman and G. Siegel for advice on the performance of this study and in preparation of the manuscript, and to Mr. A. Nadel for assistance in performing the electro-oculograms.
Seventy patients with Parkinson's disease were examined specifically for several oculomotor functions. Representative patients had eye movements recorded electro-oculographically. This demonstrated some defects not clinically apparent and accentuated others.

A surprisingly high percentage (75%) of patients showed some degree of impairment of some form of oculomotor function. There was a wide range of degree of disability, i.e., jerkiness, "cog-wheeling" or "saccadic motion", mild limitation of extent of gaze or dissociation of sequence of motion such as head turning preceding horizontal ocular deviation and limitation or even absence of gaze in various planes. Of the latter the most prominent was impairment of upward gaze which was somewhat more frequently encountered than was abnormality of downward gaze. Lateral gaze and convergence defects were less common. Eyelid abnormalities in the form of blepharospasm, lid retraction and ptosis were frequently encountered. Pupillary abnormalities were uncommon.

Oculomotor dysfunction tended to be more severe in cases where other concomitant features of Parkinsonism were very prominent.

A short-term study of the effects of levodopa therapy was conducted in 29 patients. There was a very poor response of oculomotor dysfunction, despite improvement in other Parkinsonian features in many of the patients.


