

Short report

Prism adaptation in Parkinson's disease

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SUMMARY Prism adaptation is impaired by lesions in the basal ganglia in non-human primates, suggesting that this area is involved in this form of visuomotor learning. We investigated the ability of patients with Parkinson's disease to prism adapt. Patients and controls wore prisms which deflected vision laterally by 11° . After baseline testing with a localisation task that permitted no feedback about performance accuracy, prism adaptation was tested at 4 minute intervals over a 28 minute trial. All subjects erred initially, reaching too far to the left of the target, but a separate pointing task encouraged adaptation and reaching error decreased at a similar rate in Parkinsonians and controls. Immediately after the prisms were removed, all subjects reached to the right of the target. This negative after effect was present in controls but not patients when assessed 4 minutes later, suggesting that the patients could not maintain the new sensorimotor relationship imposed by the prisms after their removal. This is similar to performance on visuospatial and executive tasks in Parkinsonians, where ongoing behaviour cannot be modulated without external guidance.

Prism adaptation is disrupted by lesions in the basal ganglia in non-human primates.^{1,2} To explore further the role of the basal ganglia in this form of visuomotor learning, we investigated the relative ability of patients with Parkinson's disease and controls to adapt to laterally displacing prisms.

In prism adaptation, the subject wears prisms that displace the visual field in some regular fashion. Initially the subject errs in directed movements by reaching toward the displaced view of an object. Over time, adaptation occurs, allowing movements to be directed to the object's true position. After the prisms are removed, the subject errs by reaching in the direction opposite to that of the prisms' original displacement. This "negative after effect" indicates that some central correction for the prisms' distortion has been established. A period of readaptation is then required before reaching becomes accurate again.

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Methods and subjects

Fourteen patients with idiopathic Parkinson's disease participated in this study. All were receiving dopamine agonists and were considered to be optimally treated when tested. Mean patient age was 60.6 yr (SD 8.0) and education was 11.8 yr (SD 3.9). Thirteen healthy elderly people served as controls. Their mean age was 66.7 yr (SD 2.5) and education was 11.9 yr (SD 1.7). All of the subjects were right-handed and none were demented.

Localisation apparatus and testing The subject sat in front of a box whose top reached approximately to shoulder level. A cloth cover extended from the top of the box to the subject's shoulders to obscure sight of the arms. A wooden dowel (approx 24 cm high) extended vertically from the top of the box at a point 35 cm in front of the patient and 2.5 cm to the right of midline. The subject was asked to reach into the box and, with a pen, touch the undersurface of the box top at a point directly beneath the dowel. Responses were recorded on paper attached there. Only the right hand was used in all trials and the subject's hand was always placed at a standard point outside of the box before a response was initiated. A chin rest ensured correct and consistent orientation of the head and body. Inside the box, a board placed 2.5 cm behind the dowel limited localisation to the lateral axis. Each localisation trial consisted of five attempts to localise the dowel.

Table Timeline of prism adaptation procedures

Time (min)	Procedure	Prisms	
- 5	Baseline localisation trial	Off	
- 1	Baseline localisation trial	Off	
0	Prisms On, immediate localisation trial	On	
4	Localisation trial → Adaptation training	↓	
8	Localisation trial → Adaptation training		
12	Localisation trial → Adaptation training		
16	Localisation trial → Adaptation training		
20	Localisation trial → Adaptation training		
24	Localisation trial		
28	Localisation trial		
29	Prisms Off, immediate localisation trial		Off
33	Localisation trial		↓
37	Localisation trial		
41	Localisation trial		

Procedure The procedure is summarised in the table. Two baseline localisation trials were administered: 4 minutes before and immediately before the prisms were worn. After baseline testing, the subject put on goggles fitted with 20 diopter Fresnel prisms that deflected vision 11.3° to the left. Localisation was tested immediately and then at 4 minute intervals for the next 28 minutes. The prisms were then removed and a localisation trial administered immediately. Trials were repeated three more times at 4 minute intervals after the prisms were removed.

Adaptation training To ease adaptation to the prisms, subjects were given controlled experience observing their own guided movements in the period between each localisation trial. A matrix of numbers was placed on a table in front of the subjects and they were asked to point to numbers located at various distances to their left and right. Each adaptation trial was balanced for side and distance of reach. This adaptation training was omitted during the final 4 minute interval in which the prisms were worn (24–28 min), so localisation testing at 28 min was without benefit of this training.

Data analysis Performance of the two groups over time was compared using analysis of variance (ANOVA) for repeated measures. This analysis was applied to crucial time segments of the paradigm to determine changes in performance as prisms were put on and taken off, and when adaptation training was withheld. The adaptation curve of each patient was characterised by slopes using regression analysis and *t* tests were used to compare the adaptation slopes in the two groups.

Results

On the baseline localisation trials, a divergence between the localisation of the controls and Parkinsonians was noted: the Parkinsonians localised the marker further to the left ($p < 0.05$). Changes in localisation while the prisms were worn followed consistently from each group's baseline (fig).

ANOVA showed that the initial prism effect was similar in both groups (that is, comparison of times -5 and -1 to time 0) and adaptation to the prisms, as indicated by the slow return of localisation error to baseline levels, occurred at a similar rate (that is, comparison of times 0 to 24) (fig). Withdrawal of adaptation training, measured at the 28 minute interval, decreased the level of adaptation from that seen at 24 minutes similarly in both groups (fig).

Since each localisation trial consisted of five attempts, the variability of a subject's response at each time point could be calculated. There were no significant differences in variability of performance as a result of transition from one component of the paradigm to another (that is, putting on prisms, withdrawal of adaptation training or taking off prisms) nor were there differences between patients and controls.

Localisation data from 0 to 24 minutes for each subject was subjected to a regression analysis which derived a slope that characterised the rate of adaptation. Mean slopes for patients and controls were compared using a *t* test; they did not differ significantly.

Both groups showed significant negative after effect when the prisms were removed at 29 minutes and localisation compared with that seen at -5 and 0 minutes ($p < 0.05$). However, comparison of performance at 29, 33 and 37 minutes revealed that negative after effect lasted significantly longer in the control group. This is seen at 33 minutes where the Parkinson's disease group has returned to baseline while the controls still show negative after effect ($p < 0.05$).

Discussion

Prisms produce a lack of correlation between intended and actual movement. A new sensorimotor relation-

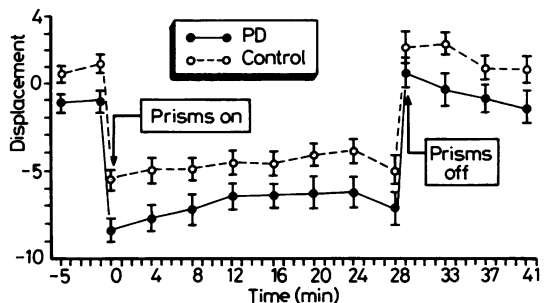


Fig Displacement (in cm) of localisation attempts from the target marker in Parkinson's disease (PD) and control groups. Each data point represents the group mean value (\pm standard error) of the mean of five localisation attempts per patient at that time. Times at which the prisms were put on and removed are indicated.

ship must then be established to compensate for the displacement caused by the prisms. Parkinsonians adapted to the prisms, but could not maintain this modulation of behaviour once the prisms were removed. In contrast, for controls the negative after effect was more persistent; their adaptation was not entirely dependent on the presence of the prisms.

Since localisation was tested at 4 minute intervals, we could not see how quickly the negative after effect actually dissipated in each group. It was present immediately after the prisms were removed in the Parkinson's disease group, but not 4 minutes later. It is possible that it was not maintained at all without the prisms. In contrast, we know that this effect was present in the controls for at least 4 minutes. Future studies should concentrate in more detail on temporal changes after the prisms are removed.

Weiner *et al.*³ found no difference between prism adaptation in Parkinson's disease and controls. However, their paradigm differed in two significant respects: (1) In that study, the patient was given the opportunity to determine the accuracy of each localisation attempt by observing his hand placement. This allowed patients to modify their attempts based on where they thought the target ought to be as opposed to where they actually saw it. In the present study, patients never received any feedback about the accuracy of their attempts, so the actual process visuomotor adaptation was more directly monitored. (2) The previous study also did not assess adaptation at frequent intervals, and it is precisely in this form of analysis that group differences emerged here.

It is not clear why the baseline of patients and controls differed. Further studies using both hands and varying marker locations would be useful for clarifying this issue. The variability of localisation in the two groups was similar and performance in each group was consistent over time. It is logical then to analyse each group's performance from its own baseline.

Prism adaptation requires experiencing the outcome of intended movements; passive movement will not result in prism adaptation. Adaptation is also impeded when movements are not fully under the subject's control and he cannot produce the movement that he intended to produce.⁴ Poor prism adaptation has been reported in patients and animals with cerebellar lesions, perhaps because their dysmetria affects the adaptation process in this manner.^{3,5} It was possible the tremor and bradykinesia of Parkinson's disease would impede prism adaptation in a similar manner. However, the present data indicate that Parkinsonians are capable of adapting as rapidly as controls.

Prism adaptation does not occur in primates with basal ganglia lesions¹² and was impaired in a patient

with a unilateral caudate lesion.⁶ Since it is not simply that the motor disorders of Parkinson's disease disrupt prism adaptation, the basal ganglia must play a more central role in the adaptation process. The hypothetical processes of corollary discharge⁷ and efference copy⁸ have been put forward to suggest that the basal ganglia assist in the correlation and recalibration of sensory and motor information. However, these concepts would predict that basal ganglia dysfunction results in defective prism adaptation as opposed to the lack of persistence of negative after effect.

It is also possible that the basal ganglia are part of a corticostriatal system that aids in planning and modulating ongoing activity (either motor or cognitive) in the absence of external guidance. For example, patients with Parkinson's disease perform poorly on tracking and tracing tasks in which they must shift from one movement to another without external guidance;⁹⁻¹² they are unable to systematically shift mental set in cognitive tasks;^{13,14} and have difficulty with constructional tasks which involve planning and checking the constructive activity. In the present study as well, removal of a condition in the external environment that prompted a modification of behaviour resulted in the rapid cessation of that modification.

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References

- 1 Bossom J. The effect of brain lesions on prism adaptation in monkey. *Psychonomic Science* 1965;2:45-46.
- 2 Bossom J, Ommaya AK. Visuo-motor adaptation (to prismatic transformation of the retinal image) in monkeys with bilateral dorsal rhizotomy. *Brain* 1968; 91:161-72.
- 3 Weiner MJ, Hallet M, Funkenstein HH. Adaptation to lateral displacement of vision in patients with lesions of the central nervous system. *Neurology* 1983;33:766-72.
- 4 Held R, Freedman ST. Plasticity in human sensorimotor control. *Science* 1963;142:455-61.
- 5 Baizer JS, Glickstein M. Role of cerebellum in prism adaptation. *J Physiol (Lond)* 1974;24:34P-5P.
- 6 Potegal M. The caudate nucleus egocentric localization system. *Acta Neurobiol Exp* 1972;32:479-94.
- 7 Teuber H-L. Complex functions of the basal ganglia. In:

- Yahr MD, ed. *The Basal Ganglia*. New York: Raven Press, 1976:151–68.
- 8 Angel RW. Efference copy in the control of movement. *Neurology* 1976;**26**:1164–8.
 - 9 Stern Y, Mayeux R, Rosen J, IJson J. Perceptual motor dysfunction in Parkinson's disease: a deficit in sequential and predictive movement. *J Neurol Neurosurg Psychiatry* 1983;**46**:145–51.
 - 10 Stern Y, Mayeux R, Rosen J. Contribution of perceptual motor dysfunction to construction and tracing disturbances in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1984;**47**:983–9.
 - 11 Flowers K. Lack of prediction in the motor behaviour of Parkinsonism. *Brain* 1978;**101**:35–52.
 - 12 Bloxham CA, Mindel TA, Frith CD. Initiation and execution of predictable and unpredictable movements in Parkinson's disease. *Brain* 1984;**107**:371–84.
 - 13 Lees AJ, Smith E. Cognitive deficits in the early stages of Parkinson's disease. *Brain* 1983;**106**:257–70.
 - 14 Flowers KA, Robertson C. The effect of Parkinson's disease on the ability to maintain a mental set. *J Neurol Neurosurg Psychiatry* 1985;**48**:517–29.