

Visual control of locomotion in Parkinson's disease

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Summary

The effect of placing parallel lines on the walking surface on parkinsonian gait was evaluated. To identify the kind of visual cues (static or dynamic) required for the control of locomotion, we tested two visual conditions: normal lighting and stroboscopic illumination (three flashes/s), the latter acting to suppress dynamic visual cues completely. Sixteen subjects with idiopathic Parkinson's disease (nine males, seven females; mean age 68.8 years) and the same number of age-matched controls (seven males; nine females, mean age 67.5 years) were studied. During the baseline phase, Parkinson's disease patients walked with a short-stepped, slow velocity pattern. The double limb support duration was increased and the step cadence was reduced relative to normal. Under normal lighting, visual cues from the lines on the walking surface induced a significant improvement in gait velocity and stride length

in Parkinson's disease patients. With stroboscopic illumination and without lines, both groups reduced their stride length and velocity but the changes were significant only in the Parkinson's disease group, indicating greater dependence on dynamic visual information. When stroboscopic light was used with stripes on the floor, the improvement in gait due to the stripes was suppressed in parkinsonian patients. These results demonstrate that the perceived motion of stripes, induced by the patient's walking, is essential to improve the gait parameters and thus favour the hypothesis of a specific visual-motor pathway which is particularly responsive to rapidly moving targets. Previous studies have proposed a cerebellar circuit, allowing the visual stimuli to by-pass the damaged basal ganglia.

Keywords: Parkinson's disease; locomotion; optic flow

Introduction

Since the original description by Parkinson (1817), Parkinson's disease has been recognized as a motor disorder resulting from neuropathological changes affecting the basal ganglia (Barbeau, 1986). However, sensorial deficits have also been reported (Snider *et al.*, 1976; Koller, 1984). More specifically, visual deficits have been demonstrated with respect to visual evoked potentials (Bodis-Wollner and Yahr, 1978; Onofrij *et al.*, 1986) and spatiotemporal contrast sensitivity (Bulens *et al.*, 1986; Tagliati *et al.*, 1992). The interest in the visual defect in parkinsonism is enhanced by the possible relationships between gait disorders and visual perception, inasmuch as gait problems such as festination and the freezing phenomenon are strongly influenced by visual stimulation (Mestre *et al.*, 1992).

The earliest detailed analysis of gait in parkinsonism was performed by Martin (1967), who described mainly the consequences on gait of encephalitis lethargica. Martin was also the first to report the effectiveness of utilizing vision to facilitate locomotor activity. Moreover, he showed that only certain visual stimuli were effective in improving gait:

transverse lines, an inch or more wide, 18 inches or so apart, and of a colour contrasting with that of the floor (white lines on a dark ground). Zigzag lines, lines parallel to the line of movement, very narrow lines, lines wider than 6 feet or stripes without contrast of colour had no influence. Later studies confirmed the positive influence of visual guidance on gait movements in Parkinson's disease patients (Forssberg *et al.*, 1984; Azulay *et al.*, 1996). Other sensory cues may also improve parkinsonian gait, e.g. rhythmic auditory cues (Richards *et al.*, 1992; Thaut *et al.*, 1996; McIntosh *et al.*, 1997). While the influence of vision on gait control in parkinsonian patients has been established, the questions regarding the mechanisms of action of the visual cues are still controversial (Morris *et al.*, 1996). One suggestion is that stripes on the floor improve gait by drawing attention to the stepping process. Another is that each stripe may trigger a step during locomotion. A third is that, when patients walk, the stripes move downward in the visual field and induce specific dynamic visual stimuli that may improve motor performance. Dynamic visual cues have been shown to

provide an important contribution to body balance, in standing (Amblard *et al.*, 1985; Crémieux et Mesure, 1994) as well as in walking (Assaiante *et al.*, 1989) in healthy adults. The aim of the present study was to evaluate the type of visual cues (static or dynamic) required for the control of locomotion in parkinsonian patients. With this aim, we tested two visual conditions: normal lighting and stroboscopic illumination, the latter serving to suppress dynamic visual cues completely (Amblard *et al.*, 1985; Assaiante *et al.*, 1989). The comparison of locomotor performance observed with normal vision with that obtained under stroboscopic illumination allowed us to determine the specific contribution of dynamic visual cues in parkinsonian gait. We also evaluated the benefit of placing parallel lines on the walking surface in both visual conditions. This was done to determine whether motion of the lines is necessary to improve locomotor performance in Parkinson's disease patients, i.e. if transverse lines induce an improvement in gait performance under normal lighting, the persistence or removal of the effect under stroboscopic illumination would determine whether or not it is linked to the perceived motion of the lines.

Methods

Subjects

Thirty-two subjects were included in the study: 16 Parkinson's disease patients (nine males, seven females; mean age 68.8 ± 4 years) and the same number of age-matched normal controls (seven males, nine females; mean age 67.5 ± 5 years). All patients were clinically diagnosed as having 'idiopathic' Parkinson's disease according to the UK Brain Bank diagnostic criteria (Gibb and Lees, 1988), and had a sustained improvement with dopaminergic treatments.

Eleven parkinsonian patients were Hoehn and Yahr stage II and five were Hoehn and Yahr stage III. The mean disease duration was 6.3 years. All patients and controls had a visual acuity of 20/20, with correction if necessary. The recordings were carried out at the same hour in the morning. Parkinson's disease patients had fasted overnight and were without treatment for at least 12 h. All patients and controls gave informed consent according to the declaration of Helsinki and the protocol was approved by the ethics committee of The University of Marseille.

Procedures of gait analysis

All observations were performed on a 12 m walkway. Kinematic gait analysis was performed with a commercially available automatic motion analyser (Ferrigno and Pedotti, 1985), with four cameras at a sampling frequency of 100 Hz and with two high-resolution force platforms which measure the timing amplitude, direction and location of the force exerted on the support level. Nine markers were placed symmetrically on the subjects at the following sites: fifth metatarsal joint, external malleolus, tibial plate and

posterosuperior iliac crest, with the last marker being placed on the sacrum to determine hip, knee and ankle movements. In the present study, only some of the gait parameters (velocity, stride length, cadence, double limb support duration) were analysed.

Subjects were instructed to perform three consecutive walks, either on a uniformly grey flat surface or on the same support with parallel transverse high contrasting white lines (5 cm wide) spaced at 45 cm intervals, this having been demonstrated previously to be the most effective pattern (Azulay *et al.*, 1996). They were instructed to walk at their natural speed, looking straight ahead without any specification regarding foot positioning. The analysis started after walking a distance of 4 m.

Two visual conditions were tested: normal lighting and stroboscopic (electronic) illumination at 3 Hz (flash duration <0.2 ms, flash energy: 0.3 J). Both types of lighting conditions were provided by the same sources and were adjusted to be perceived as having an equivalent brightness. All the light sources were placed on the ceiling of the experimental room to avoid any dazzle. The first three trials under stroboscopic illumination had the aim of familiarizing the subjects with the testing conditions. In each experiment, each of the support situations (grey floor or transverse lines) combined with each of the two illumination situations was presented according to a pseudo-random design. The results of the three trials in each situation were averaged.

Statistical analysis

ANOVA (analysis of variance) was used to compare the different parameters (stride length, velocity, cadence) between the two groups (patients and control subjects) and between the different conditions (with and without stripes, normal and stroboscopic light). The minimum 0.05 level of significance was adopted throughout the data analysis. Results are expressed as mean \pm SD.

Results

Analysis of gait parameters (Table 1)

During the control situation (normal light, normal ground), the mean gait velocity of patients with Parkinson's disease (0.76 ± 0.2 m/s) was slower than that of controls (1.13 ± 0.2 m/s) [$F(1,30) = 27.75$; $P < 0.001$]; the mean stride length was shorter (925 ± 176 mm versus 1172 ± 193 mm, respectively) [$F(1,30) = 14.48$; $P < 0.001$]; the cadence was reduced (99 ± 11.3 steps/min versus 116 ± 10.7 , respectively) [$F(1,30) = 19.79$; $P < 0.001$]; and the relative double limb support duration was greater ($13 \pm 2.3\%$ versus $9 \pm 1.4\%$, respectively) [$F(1,30) = 26.63$; $P < 0.001$]. These results were obtained for patients and controls walking at their preferred speed.

Analysis of the main effects

Before analysing the effects of stripes and illumination in each group separately, we analysed these conditions in

Table 1 Gait parameters for 16 Parkinson's disease subjects and 16 controls (mean of three trials and SD)

	Patients		Controls	
	Mean	SD	Mean	SD
Stance duration (ms)	779	110	622	55.8
Swing duration (ms)	457	56.5	424	39.8
Stance (%)	63	2.6	59	1.2
Swing (%)	37	2.6	41	1.2
Double support duration (ms)	158	34.7	98	16.4
Double support (%)	13	2.3	9	1.4
Anterior step length (mm)	468	85.2	590	99.2
Swing velocity (m/s)	1.81	0.4	2.47	0.4
Stride duration (ms)	1237	156	1046	93
Cadence (steps/min)	99	11.3	116	10.7
Stride length (mm)	925	176	1172	193
Step width (mm)	116	63.3	99	35.8
Mean velocity (m/s)	0.76	0.2	1.13	0.2

Mean and standard deviations for each locomotor parameter obtained for 16 Parkinson's disease subjects and 16 age-matched controls in the control situation (normal floor, normal light). Mean values of three trials.

both groups together. We considered first the gait velocity parameter and used a three-way ANOVA with fixed effects [groups (Parkinson's disease patients versus controls) × floor (normal floor versus stripes on the floor) × light (stroboscopic versus normal light)]. We found a significant main effect of the stripes in the whole population [$F(1,28) = 8.76; P < 0.01$], a significant main effect of the illumination [$F(1,28) = 29.27; P < 0.001$] and a significant main effect of the group [$F(1,28) = 30.69; P < 0.001$]. For the stride length parameter, there was a significant main effect of the stripes in the whole population [$F(1,28) = 5.71; P < 0.05$], a significant main effect of the illumination [$F(1,28) = 22.21; P < 0.001$] and also a significant main effect of the group [$F(1,28) = 15.40; P < 0.001$]. Finally, there was a significant interaction between vision and floor [$F(1,28) = 5.05; P < 0.05$].

Effect of the stripes

When stripes were placed on the floor, patients with Parkinson's disease and the controls were required simply to walk across the floor, looking straight ahead without any instructions about the stripes. Gait velocity was analysed using a two-way ANOVA (normal light versus stroboscopic light and normal floor versus stripes on the floor) in each group, which revealed a significant effect of the stripes only in patients with Parkinson's disease [$F(1,15) = 28.43; P < 0.001$]. Also, the parkinsonian patients walked significantly faster with stripes on the floor (0.82 ± 0.19 m/s) than without (0.76 ± 0.2 m/s), but only with normal light [$F(1,15) = 13.86; P < 0.01$] (Fig. 1). In all patients, there was no change in cadence with stripes (99 ± 11.3 steps/min and 101 ± 10.3 steps/min with and without stripes, respectively). However, we also found the same overall significant effects of the stripes in the Parkinson's disease group on the stride length

as on the velocity [$F(1,15) = 24.42; P < 0.001$]. The patients had a significantly longer stride length with stripes (970 ± 180 mm) than without stripes (930 ± 180 mm) with normal light [$F(1,15) = 23.71; P < 0.001$] (Fig. 2). Despite the improvement of their gait due to the stripes, Parkinson's disease patients remained significantly less skilful than the healthy subjects. In the normal ageing subjects, the results obtained with and without stripes did not differ, whichever of the parameters were considered. In the Parkinson's disease group, individual responses were found to vary greatly. Figure 3 shows that in five Parkinson's disease patients, absolutely no change occurred with markers on the floor (patients 2, 4, 6, 9 and 12), whereas eight patients showed an improvement of >10%. In one case (patient 14), the velocity was 32% higher with than without visual cues. Overall, a mean improvement of 9.4% was obtained in the Parkinson's disease group and was associated with an increase in stride length. The analysis of the results showed that stride length was consistently different from the space between two or three floor markers (45 or 90 cm), proving that patients with Parkinson's disease did not use the markers to regulate their stride length (Fig. 4). No correlations were found between the sensitivity to the stripes and disease duration, the Hoehn and Yahr stages or patients' ages.

Effect of the stroboscopic light

Stroboscopic illumination at 3 Hz was used to suppress dynamic visual cues for the subjects walking over both types of support (with and without stripes). In the situation without stripes, our purpose was to specify the type of visual cues (static or dynamic) which play a role in locomotor control in Parkinson's disease patients. In the situation with stripes on the floor, our aim was to determine whether or not the

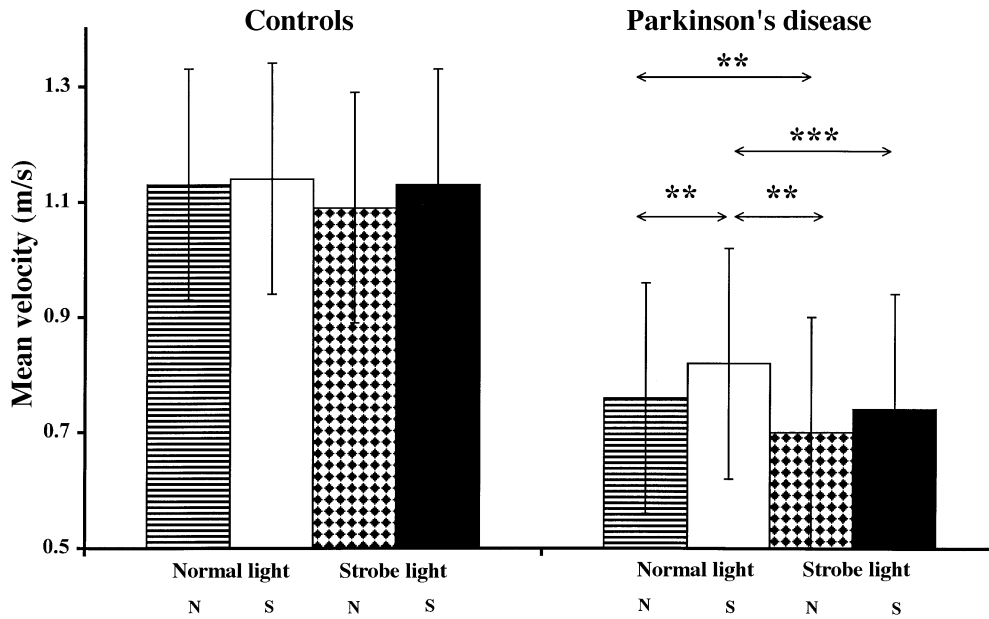


Fig. 1 Comparison of the mean values and standard deviations for velocity obtained for 16 Parkinson's disease subjects (right) and 16 controls (left) with stripes (S) and without stripes (N) on the floor combined with both conditions of illumination: normal and stroboscopic light. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

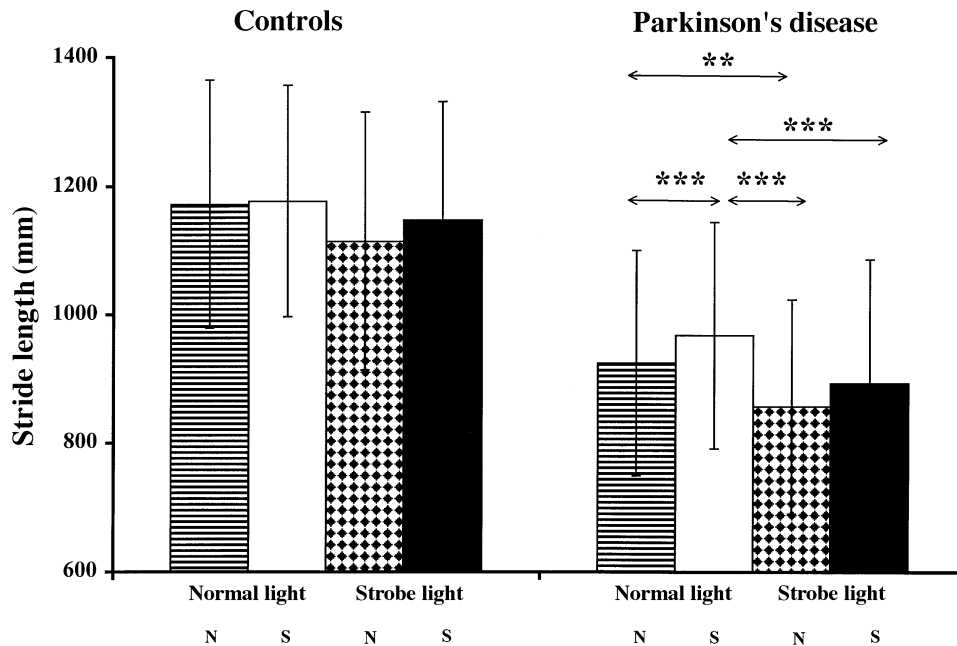


Fig. 2 Comparison of the mean values and standard deviations for stride length obtained for 16 Parkinson's disease subjects (right) and 16 controls (left) with stripes (S) and without stripes (N) on the floor combined with both conditions of illumination: normal and stroboscopic light. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

improvement obtained with normal lighting was due to the perception of motion of the lines induced by the patient's walking.

A two-way ANOVA analysis (normal light versus stroboscopic light and normal floor versus stripes on floor),

performed for each group, found a significant effect of the stroboscopic light only for the patient group [$F(1,15) = 25.72$; $P < 0.001$]. When patients and controls walked with stroboscopic illumination, only patients reduced their velocity significantly compared with normal lighting both on the

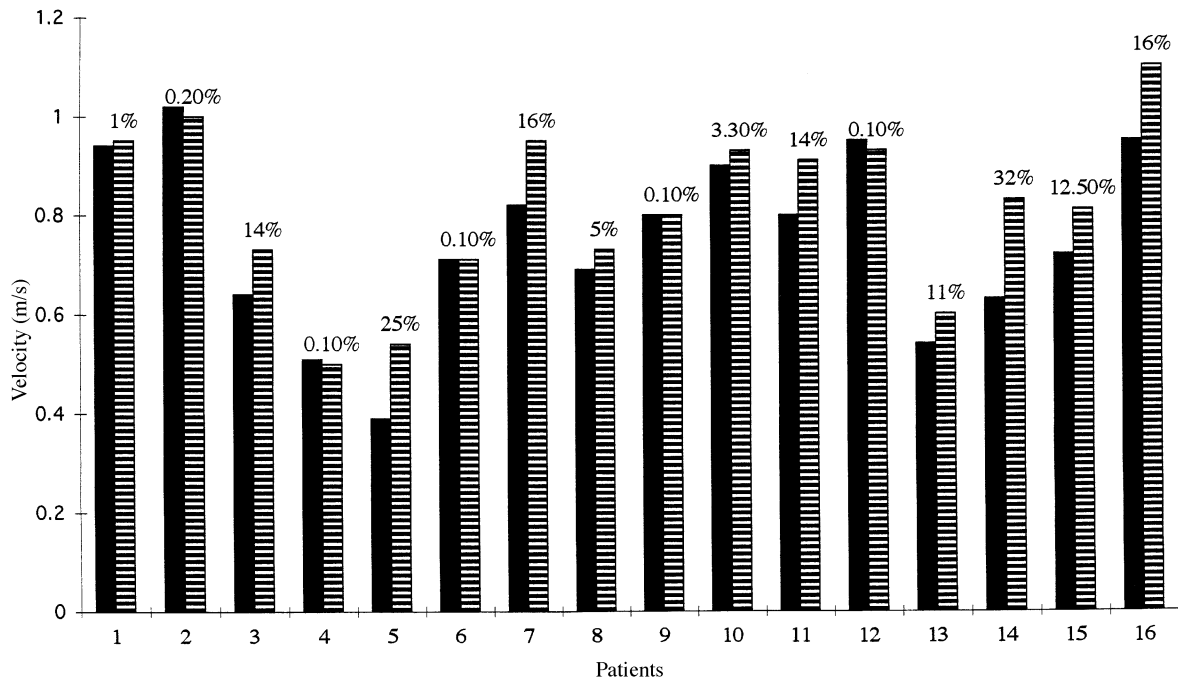


Fig. 3 Individual values of the mean gait velocity obtained with stripes (striped bars) and without stripes (solid bars) and normal light obtained for the 16 parkinsonian patients. The variation is expressed as a percentage of the baseline values. Eight patients had a velocity gain of >10%.

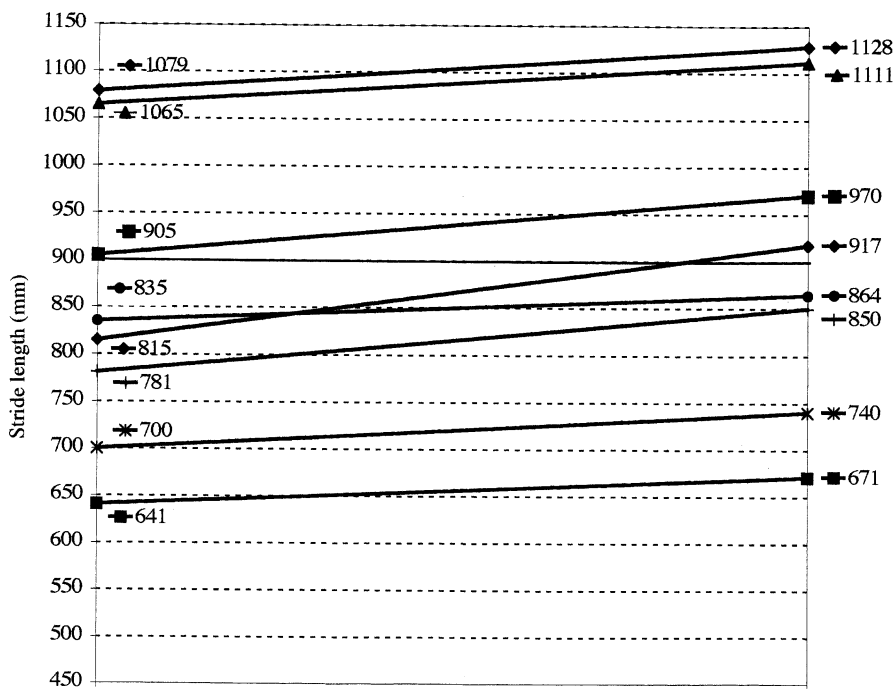


Fig. 4 Increase of the stride length induced by the stripes in the eight parkinsonian patients whose gait velocity increased by >10%. Note that the stride length remained different from the space placed between two or three stripes (45 or 90 cm).

normal floor (0.76 ± 0.2 m/s > 0.70 ± 0.22) [$F(1,15) = 17.67$; $P < 0.001$] and with stripes on floor (0.82 ± 0.2 m/s > 0.74 ± 0.2) [$F(1,15) = 13.50$; $P < 0.01$] (Fig.

1). Similarly, the stride length was significantly reduced in the Parkinson's disease group by stroboscopic light whatever the type of support [$F(1,15) = 19.44$; $P < 0.001$]. The

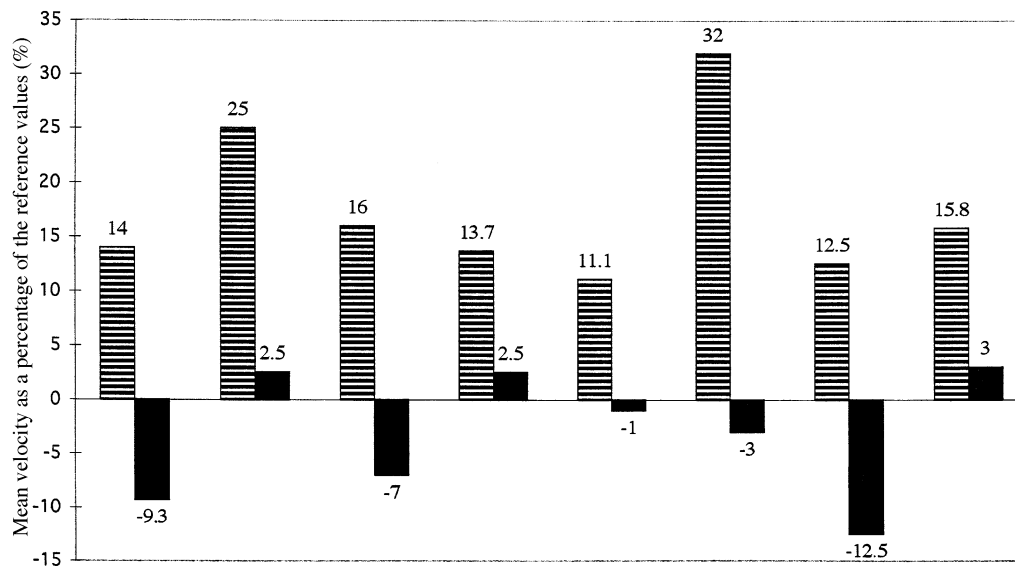


Fig. 5 Individual variations of the gait velocity obtained for the eight Parkinson's disease patients whose velocity increased by $>10\%$ with the stripes, expressed as a percentage of the baseline values. Comparison of the results obtained with stripes and normal light (striped bars) and with stripes and stroboscopic light (solid bars).

patients reduced their stride length when stroboscopic light was used both without stripes (from 925 ± 175 mm to 857 ± 166 mm) [$F(1,15) = 24.54$; $P < 0.001$] and with stripes (from 968 ± 176 mm to 893 ± 193 mm) [$F(1,15) = 11.24$; $P < 0.001$] (Fig. 2). The cadence and the double limb support time for Parkinson's disease patients and controls remained unchanged. These results suggest that the Parkinson's disease group was more dependent than the controls on dynamic visual information. Also, the improvement in velocity and in stride length induced by the transverse lines under normal lighting in Parkinson's disease patients was suppressed by the use of stroboscopic illumination (Fig. 5), and the results obtained under stroboscopic illumination with stripes returned to the baseline values. These results demonstrate that the Parkinson's disease patients were no longer able to use the visual information provided by the stripes to improve their gait parameters when dynamic visual perception was suppressed.

The healthy subjects, slightly but not significantly, showed a deterioration in their gait parameters under stroboscopic light without floor markers. When they walked under combined conditions (stroboscopic light and stripes), their velocity and stride length returned to the values obtained in the baseline conditions.

Effect of the group

When we considered velocity and stride length, the results of Parkinson's disease patients always remained significantly different from those of the healthy subjects. We found significant differences between the groups using a one-way ANOVA for both the velocity and the stride length. Significant effects were found in the following situations: normal light

without stripes [$F(1,30) = 27.75$; $P < 0.001$] for velocity and [$F(1,30) = 14.48$; $P < 0.001$] for stride length; normal light with stripes [$F(1,30) = 26.10$; $P < 0.001$] for velocity and [$F(1,30) = 12.37$; $P < 0.01$] for stride length; stroboscopic light without stripes [$F(1,30) = 32.45$; $P < 0.001$] for velocity and [$F(1,30) = 16.38$; $P < 0.001$] for stride length; and stroboscopic light without stripes [$F(1,30) = 30.34$; $P < 0.001$] for velocity and [$F(1,30) = 15.47$; $P < 0.001$] for stride length.

Finally, we performed a three-way ANOVA with fixed effects as previously in the section concerning the analysis of the main effects [groups (Parkinson's disease patients versus controls) \times floor (normal floor versus stripes on the floor) \times light (stroboscopic versus normal light)] but restricted to those 50% of Parkinson's disease patients who showed a pronounced effect of stripes on gait velocity, and with stripes and illumination as other main factors. We limited the analysis to just these patients due to the variability of the data and the rather small effect, since it is well known that not all patients are equally sensitive to visual cues. In these conditions, we found a clearly significant two order interaction [$F(3,63) = 5.32$; $P < 0.01$], thus confirming that both groups behaved differently from each other with respect to vision. More precisely, there was a significant interaction between groups and light when the stripes were present [$F(1,22) = 6.04$; $P < 0.05$], indicating that the groups did not respond in the same way to the deprivation of motion vision. Similarly, there was a significant interaction between groups and floor conditions under normal lighting [$F(1,22) = 8.06$; $P < 0.01$], indicating that the groups did not respond in the same way to the presence of stripes.

Discussion

The results of these experiments provide evidence that the visual control of locomotion in Parkinson's disease is quite different from that observed in normal age-matched controls (mean age 67.5 ± 5 years). In Parkinson's disease, dynamic visual perception is required predominantly when patients are walking, whereas the results for normal age-matched controls showed that, similarly to younger healthy subjects (Assaiante *et al.*, 1989), such dynamic visual perception is not required. We showed that some of the parkinsonians (about half) are able to use transverse stripes placed on the floor to improve their gait velocity and stride length, confirming previous reports. However, our study is the first to show that this improvement is conditioned by the perception of motion of the stripes. Our results enhance the validity of previous hypotheses concerning the role of a specific visuomotor pathway, specifically elicited by dynamic visual stimuli.

In the first part of our study, we found that the results of most of the spatiotemporal and kinematic parameters measured for Parkinson's disease patients were different from those obtained for normal age-matched controls, as has been found in previous studies which have quantified the gait abnormalities in Parkinson's disease patients (Knutsson, 1972; Murray *et al.*, 1978; Stern *et al.*, 1983; Blin *et al.*, 1990, 1991; Ferrandez and Blin, 1991; Morris *et al.*, 1994a). Compared with healthy elderly people, Parkinson's disease patients walked more slowly, with shorter strides and longer duration of stance and double support phases; their cadence was also significantly less. However, we did not find a significant reduction of swing duration, which has been reported previously (Blin *et al.*, 1990).

We did not analyse the relationships between cadence, stride length and velocity. In the literature, the results are controversial, as are the hypotheses about the central mechanisms of gait abnormalities in Parkinson's disease. Stern *et al.* (1983) and Blin *et al.* (1990) found the relationships between the kinematic parameters unchanged and, hence, considering that the invariant pattern of gait is not impaired in Parkinson's disease, they concluded that the low velocity they observed is a consequence of the mechanical impairment and posture abnormalities, and results in the other observed changes. Contrary to these reports, Morris *et al.* (1994b) found that, when the effects of walking speed were taken into account, stride length was shorter and the cadence higher in Parkinson's disease patients than in controls. They did not find an increase in the double limb support duration phase. The authors considered that the fundamental deficit in parkinsonian gait is the internal regulation of stride length.

In their experiments, Morris *et al.* (1994a, b, 1996) used visual markers to modify the gait pattern of the subjects. External sensory cueing has been mentioned repeatedly as a good strategy to facilitate locomotor activity in Parkinson's disease, as demonstrated using rhythmic auditory stimulation

(Thaut *et al.*, 1996; McIntosh *et al.*, 1997) or visual stimulation (Martin, 1967; Forssberg *et al.*, 1984; Richards *et al.*, 1992; Morris *et al.*, 1994a; Azulay *et al.*, 1996). For visual cueing, the same pattern was usually used: transverse stripes along the pathway in front of the patient. Martin (1967), who first reported the beneficial effect of these markers, demonstrated that other types of markers were not effective. Forssberg *et al.* (1984) later reported similar results but the authors did not detail their procedure. They placed sheets of white paper on the ground and noted that gait improved with an increase in stride length by $>100\%$. Walking speed also increased, but no quantified data were given. Richards *et al.* (1992) also used transverse stripes separated by a distance equal to 40% of the patient's height, and confirmed a beneficial effect. Fifteen Parkinson's disease patients were studied and were found to walk faster with visual cues (86.1 m/s) than without (72.3 m/s). This improvement was due to an increase in stride length associated with a slower cadence. The modifications obtained by Richards *et al.* (1992) were considerably smaller than those reported by Forssberg *et al.* (1984), but very similar to ours. In a previous study (Azulay *et al.*, 1996), as well as in this experiment, we found that the stripes induced a significant increase in velocity and stride length, while cadence remained unchanged. The overall improvement in velocity was not very large when we considered the mean value of the Parkinson's disease group ($\sim 10\%$) but was explained by important variations in individual responses: some patients did not modify their gait parameters with the stripes whereas others, for example, increased their speed up to 32%.

A major factor concerning the visual cues is the instructions given to the patients. In the studies performed by Morris *et al.* (1994a, b, 1996), the visual markers were spaced at the mean stride length of the Parkinson's disease subjects or their age-matched controls and the patients were instructed to step over the markers. The aim of these studies was to evaluate the ability of parkinsonians to regulate their stride length, and the investigators showed that Parkinson's disease subjects were still able to achieve a normal stride length with visual cues, whereas stride length could not be modulated by internal control mechanisms. In their latest paper, Morris *et al.* (1996) also discussed the mechanism of action of visual cue training. They found that the effects of visual cueing persisted for 2 h after the removal of the markers when the subjects were trained for 20 min. The investigators considered that this result favoured the hypothesis of an attentional strategy more than an enhancement of the locomotor pattern (the visual cues triggering each step).

Our experimental paradigm focused on another aspect of the control of locomotion. We hypothesized that the highly specific pattern of visual cues which is effective does not support the idea of an attentional process focused on stride length as a single explanation. Moreover, we did not provide subjects with any instructions about the stripes. They needed only to walk as normally as possible looking straight ahead. The analysis of the stride length obtained with the stripes

clearly confirmed that the patients did not use the stripes as a target for foot positioning. Furthermore, the random order of the different situations and the small number of recordings render the hypothesis of a training effect unlikely. In these conditions, we evaluated the role of the motion of stripes as produced by the patient's own movement. During locomotion, it is possible to differentiate static visual cues that are available within a single flash of stroboscopic light, namely position and orientation visual cues, from dynamic visual cues that are perceptible under permanent illumination and are involved in the visual perception of movement produced by the subject's own actions (Assaiante *et al.*, 1989), also called optic flow. The role of dynamic visual cues in visually guided locomotion in normal young adults appears essential only when the conditions of equilibrium are compromised (Assaiante *et al.*, 1989), whereas we found that in Parkinson's disease patients, and not in the normal age-matched controls, stroboscopic light produced a deterioration of the gait velocity and the stride length even in unperturbed conditions of equilibrium, suggesting that the patients were highly dependent on dynamic visual information for the control of their gait velocity. Moreover, when stroboscopic light was used in combination with stripes, Parkinson's disease patients who had improved their gait parameters with the floor markers no longer benefited from the stripes. Considering that we used a methodology which avoided any dazzling effect, this result supports the hypothesis that the stripes generated optic flow which influenced the gait velocity and stride length in patients. Recently, Prokop *et al.* (1997) have shown that optic flow modulates walking velocity in normal subjects and that this effect was related to a modulation of stride length without a modulation in stride frequency, results in line with those we obtained with the Parkinson's disease patients. In their experiments, Prokop *et al.* (1997) used an artificial optic flow which resulted in a mismatch between the leg proprioceptive and the visual velocity information. Their results suggest that the adjustment of the gait velocity is the result of a summation of visual and leg proprioceptive velocity information. The fact that the strategy of Parkinson's disease patients to control their walking velocity relies more on information originating from dynamic visual cues than from proprioceptive feed-back may be due to a reduced kinesthetic feed-back which has been established recently by Demirci *et al.* (1997). We can therefore suggest the hypothesis that the visual dependence may be the consequence of an adaptative process, in a long-standing degenerative disease such as Parkinson's disease, to compensate for an impaired kinaesthetic feed-back.

It is well established that movements driven by external stimuli employ different pathways from those driven by internal decisions (Goldberg, 1985; Passingham *et al.*, 1989). Marsden and Obeso (1994) proposed that the cerebellum may be used in Parkinson's disease to compensate for the basal ganglia deficit. Glickstein and Stein (1991) hypothesized that information concerning the motion of stripes may use a specific visuomotor pathway, relaying through the cerebellum

and thus by-passing the damaged basal ganglia. Their speculation derived from results obtained in animal models (Glickstein *et al.*, 1985) showing that cells in the cerebral cortex which are especially sensitive to moving targets provide the cerebellum with the major visual input by way of cells of the pontine nuclei. The suspected role of cerebellar pathways as an alternative motor pathway was confirmed by Rascol *et al.* (1997) in another motor task. Using single photon emission tomography, they recently reported that patients not on medication exhibited an overactivation in the ipsilateral cerebellar hemisphere during a finger-to-thumb motor task. This task was performed under sensory deprivation conditions (eyes closed), suggesting that visual information, especially concerning moving targets, may be the most powerful but not the sole input involving the cerebellar pathway.

In our study, only half of the patients improved their gait parameters using stripes. We were not able to correlate the responsiveness to the floor markers with the characteristics of the patients (age, sex) or the disease (duration, severity). The interactions between a perceptive visual field dependence–independence and the visual contribution to postural control were demonstrated recently (Isableu *et al.*, 1997). They should be addressed in the visual control of locomotion as well.

In conclusion, the results presented here suggest that in Parkinson's disease, visual cueing can facilitate locomotion and that this facilitation is linked to the visual perception of motion rather than to position or orientation. We do not know whether or not conscious perception of visual motion is necessary for the effects to occur. In fact, the dynamic visual effects may occur subconsciously perhaps using mechanisms similar to that which controls velocity in pursuit eye movements. The fact that only some Parkinson's disease patients improved their gait under visual cueing seems more likely to be attributable to differences at the perceptual than at the motor level.

Acknowledgements

We wish to thank Dr J. Massion for critically reviewing the manuscript, Dr C. Francklin-Mestre for revising the English and Mr F. Dumaine for technical assistance. This study was supported by a grant from the French Ministry of Health (Programme Hospitalier de Recherche Clinique 1996).

References

- Amblard B, Crémieux J, Marchand AR, Carblanc A. Lateral orientation and stabilization of human stance: static versus dynamic visual cues. *Exp Brain Res* 1985; 61: 21–37.
- Assaiante C, Marchand AR, Amblard B. Discrete visual samples may control locomotor equilibrium and foot positioning in man. *J Mot Behav* 1989; 21: 72–91.

- Azulay JP, Van den Brand C, Mestre D, Blin O, Sangla I, Pouget J, et al. Analyse cinématique de la marche du parkinsonien: effets de la levodopa et de stimulations visuelles. *Rev Neurol (Paris)* 1996; 152: 128–34.
- Barbeau A. Parkinson's disease: clinical features and etiopathology. In: Vinken PJ, Bruyn GW, Klawans HL, editors. *Handbook of clinical neurology*, Vol. 49. Amsterdam: Elsevier; 1986. p. 87–152.
- Blin O, Ferrandez AM, Serratrice G. Quantitative analysis of gait in Parkinson patients: increased variability of stride length. *J Neurol Sci* 1990; 98: 91–7.
- Blin O, Ferrandez AM, Pailhous J, Serratrice G. Dopa-sensitive and dopa-resistant gait parameters in Parkinson's disease. *J Neurol Sci* 1991; 103: 51–4.
- Bodis-Wollner I, Yahr MD. Measurements of visual evoked potentials in Parkinson's disease. *Brain* 1978; 101: 661–71.
- Bronstein AM. Suppression of visually evoked postural responses. *Exp Brain Res* 1986; 63: 655–8.
- Bulens C, Meerwaldt JD, van der Wildt GJ, Keemink CJ. Contrast sensitivity in Parkinson's disease. *Neurology* 1986; 36: 1121–5.
- Crémieux J, Mesure S. Differential sensitivity to static visual cues in the control of postural equilibrium in man. *Percept Motor Skills* 1994; 78: 67–74.
- Demirci M, Grill S, McShane L, Hallett M. A mismatch between kinesthetic and visual perception in Parkinson's disease. *Brain* 1997; 41: 781–8.
- Dichgans J, Mauritz KH, Allum JH, Brandt T. Postural sway in normals and atactic patients: analysis of the stabilizing and destabilizing effects of vision. *Agressologie* 1976; 17 (C Spec No): 15–24.
- Ferrandez AM, Blin O. A comparison between the effect of intentional modulations and the action of L-dopa on gait in Parkinson's disease. *Behav Brain Res* 1991; 45: 177–83.
- Ferrigno G, Pedotti A. ELITE: a digital dedicated hardware system for movement analysis via real-time TV signal processing. *IEEE Trans Biomed Eng* 1985; 32: 943–50.
- Forssberg H, Johnels B, Steg G. Is parkinsonian gait caused by a regression to an immature walking pattern? In: Hasller RG, Christ JF, editors. *Parkinson-specific motor and mental disorders*. *Adv Neurol*, Vol. 40. New York: Raven Press; 1984. p. 375–9.
- Gibb WR, Lees AJ. The relevance of the Lewy body to the pathogenesis of idiopathic Parkinson's disease. [Review]. *J Neurol Neurosurg Psychiatry* 1988; 51: 745–52.
- Glickstein M, Stein J. Paradoxical movement in Parkinson's disease. [Review]. *Trends Neurosci* 1991; 14: 480–2.
- Glickstein M, May JG 3rd, Mercier BE. Corticopontine projection in the macaque: the distribution of the labelled cortical cells after large injections of horseradish peroxidase in the pontine nuclei. *J Comp Neurol* 1985; 235: 343–59.
- Goldberg G. Supplementary motor area: structure and function: review and hypotheses. *Behav Brain Sci* 1985; 8:567–615.
- Isableu B, Ohlmann Th, Crémieux J, Amblard B. Selection of spatial frame of reference and postural control variability. *Exp Brain Res* 1997; 114: 584–9.
- Knutsson E. An analysis of parkinsonian gait. *Brain* 1972; 95: 475–86.
- Koller WC. Sensory symptoms in Parkinson's disease. *Neurology* 1984; 34: 957–9.
- Lee DN, Aronson E. Visual proprioceptive control of standing in human infants. *Percept Psychophys* 1974; 15: 529–32.
- Marsden CD, Obeso JA. The functions of the basal ganglia and the paradox of stereotaxic surgery in Parkinson's disease [see comments]. [Review]. *Brain* 1994; 117: 877–97. Comment in: *Brain* 1995; 118: 822, Comment in: *Brain* 1995; 118: 1613–7.
- Martin JP. *The basal ganglia and posture*. Philadelphia: Lippincott; 1967.
- McIntosh GC, Brown SH, Rice RR, Thaut MH. Rhythmic auditory-motor facilitation of gait patterns in patients with Parkinson's disease [see comments]. *J Neurol Neurosurg Psychiatry* 1997; 62: 2–26. Comment in: *J Neurol Neurosurg Psychiatry* 1997;63: 556–7.
- Mestre D, Blin O, Serratrice G. Contrast sensitivity is increased in a case of nonparkinsonian freezing gait. *Neurology* 1992; 42: 189–94.
- Morris ME, Ianseck R, Matyas A, Summers JJ. Ability to modulate walking cadence remains intact in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1994a; 57: 1532–4.
- Morris ME, Ianseck R, Matyas TA, Summers JJ. The pathogenesis of gait hypokinesia in Parkinson's disease. *Brain* 1994b; 117: 1169–81.
- Morris ME, Ianseck R, Matyas TA, Summers JJ. Stride length regulation in Parkinson's disease: normalization strategies and underlying mechanisms. *Brain* 1996; 119: 551–68.
- Murray MP, Sepic SB, Gardner GM, Downs WJ. Walking patterns of men with parkinsonism. *Am J Phys Med* 1978; 57: 278–94.
- Onofrij M, Ghilardi MF, Basciani M, Gambi D. Visual evoked potentials in parkinsonism and dopamine blockade reveal a stimulus-dependent dopamine function in humans. *J Neurol Neurosurg Psychiatry* 1986; 49: 1150–9.
- Parkinson J. *An essay on the shaking palsy*. London: Sherwood, Neely, and Jones; 1817.
- Passingham RE, Chen YC, Thaler D. Supplementary motor cortex and self-initiated movement. In: Ito M, editor. *Neural programming*. Basel: Karger; 1989. p. 13–24.
- Prokop T, Schubert M, Berger M. Visual influence on human locomotion. Modulation to changes in optic flow. *Exp Brain Res* 1997; 114: 63–70.
- Rascol O, Sabatini U, Fabre N, Brefel C, Loubinoux I, Celsis P, et al. The ipsilateral cerebellar hemisphere is overactive during hand movements in akinetic parkinsonian patients. *Brain* 1997; 120: 103–10.
- Richards CL, Malouin F, Bédard PJ, Cioni M. Changes induced by L-dopa and sensory cues on the gait of parkinsonian patients. In:

Wollacot M, Horak F, editors. Posture and gait: control mechanisms. Vol. II. Eugene (OR): University of Oregon Books; 1992. p. 126–9.

Snider SR, Fahn S, Isgreen WP, Cote LJ. Primary sensory symptoms in parkinsonism. *Neurology* 1976; 26: 423–9.

Stern GM, Franklyn SE, Imms FJ, Prestidge SP. Quantitative assessments of gait and mobility in Parkinson's disease. *J Neural Transm* 1983; Suppl 19: 210–4.

Tagliati M, Brannan JR, Bodis-Wollner I. Contrast sensitivity in PD

[letter; comment]. *Neurology* 1992; 42: 1126–7. Comment in: *Neurology* 1991; 41: 1200–2.

Thaut MH, McIntosh GC, Rice RR, Miller RR, Rathbun J, Brault JM. Rhythmic auditory stimulation in gait training for Parkinson's disease patients. *Mov Disord* 1996; 11: 193–200.

Received November 10, 1997. Revised August 17, 1998.

Accepted August 27, 1998