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Stride length regulation in Parkinson's disease
Normalization strategies and underlying mechanisms

Meg E. Morris,[1,2] Robert Iansek,[1] Thomas A. Matyas[2] and Jeffery J. Summers[3]

[1]*Geriatric Research Unit, Kingston Centre, Cheltenham, the* [2]*School of Physiotherapy and Behavioural Health Sciences, La Trobe University, Bundoora and the* [3]*Department of Psychology, University of Southern Queensland, Toowoomba, Australia*

Correspondence to: Meg Morris, Geriatric Research Unit, Kingston Centre, Warrigal Road, Cheltenham 3192, Australia

Summary
Results of our previous studies have shown that the slow, shuffling gait of Parkinson's disease patients is due to an inability to generate appropriate stride length and that cadence control is intact and is used as a compensatory mechanism. The reason for the reduced stride length is unclear, although deficient internal cue production or inadequate contribution to cortical motor set by the basal ganglia are two possible explanations. In this study we have examined the latter possibility by comparing the long-lasting effects of visual cues in improving stride length with that of attentional strategies. Computerized stride analysis was used to measure the spatial (distance) and temporal (timing) parameters of the walking pattern in a total of 54 subjects in three separate studies. In each study Parkinson's disease subjects were trained for 20 min by repeated 10 m walks set at control stride length (determined from control subjects matched for age, sex and height), using either visual floor markers or a mental picture of the appropriate stride size. Following training, the gait patterns were monitored (i) every 15 min for 2 h; (ii) whilst interspersing secondary tasks of increasing levels of complexity; (iii) covertly, when subjects were unaware that measurement was taking place. The results demonstrated that training with both visual cues and attentional strategies could maintain normal gait for the maximum recording time of 2 h. Secondary tasks reduced stride length towards baseline values as did covert monitoring. The findings confirm that the ability to generate a normal stepping pattern is not lost in Parkinson's disease and that gait hypokinesia reflects a difficulty in activating the motor control system. Normal stride length can be elicited in Parkinson's disease using attentional strategies and visual cues. Both strategies appear to share the same mechanism of focusing attention on the stride length. The effect of attention appears to require constant vigilance to prevent reverting to more automatic control mechanisms.

Keywords: Parkinson's disease; gait; basal ganglia; attention

Abbreviations: DS = double limb support; SMA = supplementary motor area

Introduction
Decreased stride length during walking is a hallmark of idiopathic Parkinson's disease; however, its pathogenesis and response to treatment are poorly understood. The reduced stride size is a primary determinant of gait hypokinesia (reduced movement) which progressively worsens as the disease advances. Because gait disturbance is associated with an increased risk of falls (Aita *et al.*, 1982) and loss of independence (Schenkman, 1992), considerable effort is directed towards the treatment of walking in Parkinson's disease. Pharmacological therapy is particularly effective in the early stages of the condition; however, with disease progression, patients can experience motor fluctuations (Marsden, 1994) and the short-stepped walking pattern can re-emerge. Physiotherapy has also been advocated for the management of gait disorders in Parkinson's disease (e.g. Banks and Caird, 1989; Formisano *et al.*, 1992; Schenkman, 1992), although the development of optimal rehabilitation strategies has been hampered by an inadequate understanding of the pathogenesis of stride length disturbance. As yet there is no definitive treatment method for assisting patients to regulate the length of the stride throughout the course of the disease.

The results of our previous research on gait hypokinesia (Morris *et al.*, 1994*a*) suggested that Parkinson's disease subjects have particular difficulty with the internal regulation of stride length, even though cadence (steps per minute)

control is intact and is easily modulated for a variety of conditions (Morris *et al.*, 1994*b*). Parkinson's disease subjects do have a higher cadence rate than control subjects for any given velocity; however, the increased cadence is a compensation for reduced step size (Morris *et al.*, 1994*b*).

The reason for the reduced movement amplitude in Parkinson's disease has, however, not yet been fully elucidated. An investigation by Berardelli *et al.* (1986) showed that upper limb movements in Parkinson's disease were hypometric and that reduced movement amplitude was related to an abnormally low initial force of muscle contraction. However, they noted that patients could intentionally match the required force in order to achieve the desired movement amplitude, provided that sufficient time was allowed. In another study on upper limb movement, Sheridan *et al.* (1987) found that Parkinson's disease subjects could improve their movement amplitude and speed to normal values, although this occurred at the expense of movement accuracy. Martin *et al.* (1994) also demonstrated that Parkinson's disease subjects could perform arm movements at control amplitude if appropriately trained and could maintain control values for each submovement in a sequence of movements, irrespective of external cues. The common finding in all of these investigations was that normal movement size could be achieved in Parkinson's disease given the appropriate conditions. In agreement with this suggestion, our own experiments have shown that, although Parkinson's disease patients typically walk with smaller steps, they can achieve the desired stride amplitude when provided with visual cues (Morris *et al.*, 1994*a*). Therefore, the normal foot step pattern is not lost in Parkinson's disease, rather there is a problem in activating the correct stepping response for a given context.

In attempting to understand the reasons for the difficulty in generating appropriate stride length in Parkinson's disease, it is useful to consider recent investigations on the pathogenesis of hypokinesia. Studies on primates (Brotchie *et al.*, 1991*a, b*) and individuals with Parkinson's disease (Georgiou *et al.*, 1993, 1994; Phillips *et al.*, 1994; Cunnington *et al.*, 1995) suggest that in hypokinesia the interaction between the basal ganglia and supplementary motor area (SMA) is disrupted during movement performance. The SMA normally prepares for a forthcoming predictable movement with a steady increase in neuronal activity during the premovement period. Once the external signal to move occurs, then neuronal activity in the SMA abruptly ceases (Mushiake *et al.*, 1990). Brotchie *et al.* (1991*a, b*) found that, in primates, the basal ganglia discharge with brief bursts of phasic activity at the end of submovements performed in a sequence. They suggested that this activity represents an internal cue which triggers the rapid drop in SMA neuronal activity. The internal cue appears to be timed to cease premovement activity in the SMA for each submovement in well-learned movement sequences; however, the phasic cue is not observed for novel or complex tasks (Brotchie *et al.*, 1991*a, b*). Thus it appears that the basal ganglia interact with the SMA for only learned movement sequences and it is these sequences that require an internal cue to string the components together. If the basal ganglia cue was absent or disturbed, as in Parkinson's disease, then it is possible that the SMA preparatory activity would be disturbed, leading to an abnormally executed movement. In this situation the abnormal internal cue would impair preparation for each submovement in the sequence and the abnormal submovement preparation would result in an abnormally executed movement. A normal cue or substitute cue delivered at the appropriate time could theoretically enhance preparatory process and result in a more normal movement.

As well as a disorder of submovement preparation, the reduced amplitude of movement in Parkinson's disease hypokinesia could result from a disorder of motor set for the entire movement sequence. A number of investigators have demonstrated the presence of set related activity within the basal ganglia nuclei (e.g. Mushiake *et al.*, 1990; Brotchie *et al.*, 1991*a, b*; Kimura *et al.*, 1992). Neural network modelling also indicates that set related activity is necessary for running of movement sequences in addition to the provision of internal phasic cues (Brotchie *et al.*, 1991*c*). In the SMA, set related activity occurs in some neurons prior to predictable sequences of movement (Mushiake *et al.*, 1990). By contrast, other neurons discharge prior to each submovement during the sequence. It has been proposed that set related activity in the basal ganglia contributes to set related activity in the SMA occurring for whole movement sequences so as to maintain the entire sequence in preparedness for running and execution (Iansek *et al.*, 1995). This function could explain why individual movements in Parkinson's disease are slow and underestimated in amplitude (e.g. Hallett and Khoshbin, 1980), as a deficient preparatory process exists in Parkinson's disease due to the deficient contribution from the basal ganglia.

This model of basal ganglia function suggests that gait hypokinesia in Parkinson's disease could relate to either a disturbance of motor set for whole movement sequences, or to disruption of the internal cue leading to difficulty in stringing each submovement together as a result of defective submovement preparation. The motor cortex plays a major role in execution of movements and abnormal motor set for a whole gait sequence could affect the ability to generate sufficient force to elicit a normal step for each component of the sequence. On the other hand, absence of a phasic cue to move could result in impaired motor preparation, resulting in a shorter step, possibly because the preparation for the step is not terminated at the appropriate locus of the gait cycle.

In considering which of these two motor control mechanisms underpin gait hypokinesia, it is important to recount that Parkinson's disease patients have a fundamental disturbance in stride length regulation and that cadence control remains intact (Morris *et al.*, 1994 *a, b*). It becomes difficult to implicate abnormal cue production by the basal ganglia as the cause of reduced stride length because the function of the internal cue is to string together submovements in a sequence precisely

(Iansek *et al.*, 1995). In parkinsonian gait the submovements appear to be strung together normally, as reflected by the finding that patients retain the ability to modulate walking cadence (Morris *et al.*, 1994*b*). We are therefore left with the hypothesis that reduced stride length in Parkinson's disease may be due to inadequate motor set for gait.

Clinically, a number of strategies have been used to enhance walking in Parkinson's disease. The most well-known method is the use of visual cues placed on the floor at the desired step length to assist with gait initiation and execution (Quintyn and Cross, 1986; Bagley *et al.*, 1991). According to Purdon Martin (1967), visual cues are necessary to elicit the normal stepping response in Parkinson's disease and 'without seeing them [his] voluntary effort is of no avail'. External cues may thus have access to motor control mechanisms which bypass the basal ganglia–SMA loop to enhance movement preparation for each step within the sequence. Alternatively, the cues might simply act to enhance set for large strides by focusing the patients attention on walking with criterion step length. From this viewpoint, other clinicians have noted that visual cues are not always necessary to facilitate movement and that attentional strategies such as mentally rehearsing the forthcoming movement sequence before it is performed, concentrating on the movement during execution, and avoiding dual task performance also improve the gait pattern in Parkinson's disease (Quintyn and Cross, 1986; Yekutiel *et al.*, 1991; Morris *et al.*, 1995; Morris and Iansek, 1996). It is possible that attentional strategies facilitate a more normal locomotor pattern by enhancing motor set for the entire gait sequence.

Because it is not yet clear whether stride length decrements in Parkinson's disease are due to a disorder of motor set or defective internal cueing, it has been difficult to structure rehabilitation strategies for gait hypokinesia on a rational basis. This problem is compounded by the paucity of controlled clinical trials to evaluate the immediate and longer-term effects of gait training in Parkinson's disease. Evaluations of attentional strategies (e.g. Quintyn and Cross, 1986; Yekutiel *et al.*, 1991; Morris *et al.*, 1995) and cueing techniques (Martin, 1967; Forssberg *et al.*, 1984; Bagley *et al.*, 1991; Weissenborn, 1993) amount to only a handful of anecdotal reports and single session studies.

The purpose of this series of experiments was to examine the influence of motor set for the entire gait sequence on the regulation of stride length in Parkinson's disease by comparing the effects of two training strategies on the spatial and temporal parameters of gait. In the first study, we compared the effects of external cues with a specific attentional strategy to ascertain whether these two methods used similar or different motor control mechanisms to normalize stride length. The degree to which Parkinson's disease subjects could be trained to walk to control parameters using each of the strategies was determined, and then we examined the duration of normal gait once the strategies were removed. In the second study we examined the effects of a secondary task performance on the gait pattern following training with either visual cues or the attentional strategy. A series of secondary verbal-cognitive tasks of increasing levels of complexity were used to clarify whether the strategies used attentional or non-attentional mechanisms in the maintenance of normal gait. In the final study, covert monitoring of the gait pattern enabled further examination of the extent to which attentional resources and social motivation provided by supervision enabled the gains from visual cue training to be retained. Collectively, the results suggested that maintenance of normal gait is possible in Parkinson's disease for short time periods and the underlying basis for the sustained improvement is by the use of motor control mechanisms which utilize attentional resources. These control mechanisms appear to bypass the basal ganglia–SMA interaction in order to enhance motor set for large strides for the entire gait sequence.

Method

Subjects

A total of 54 subjects was recruited for the three studies. Twenty-seven subjects with idiopathic Parkinson's disease were recruited from the Kingston Centre, Australia and the same number of age-, sex- and height-matched controls were recruited from senior citizens groups in the Melbourne metropolitan region. In the first and second studies there were 16 Parkinson's disease subjects and 16 control subjects and in the third study there were eight Parkinson's disease subjects and eight control subjects. Some of the subjects in Studies Two and Three had also participated in Study One. The three studies were conducted at 2 month intervals. Subjects were required to provide informed consent according to the Helsinki declaration (1964). To be included, subjects also had to be able to walk 12 m up to 18 times over a 2 h period without a walking aid or assistance. Parkinson's disease subjects were only included if their diagnosis had been confirmed by a neurologist.

Subjects were excluded if they had a past history of neurological conditions other than Parkinson's disease or cardiorespiratory, orthopaedic or visual disturbance that impaired walking ability. Parkinson's disease subjects were also excluded if they were on major tranquilizers, scored <24 out of 38 on the Short Test of Mental Status dementia test (Kokeman *et al.*, 1987) or if they had dyskinesia, as determined by the neurologist.

The final sample comprised 33 males and 21 females with a mean age of 73.4 years and an age range of 59–82 years. For all experiments, testing of the Parkinson's disease subjects commenced 45–75 min post-Parkinson's disease medication, when they were judged by a neurologist or physiotherapist to be in the 'on' state. One subject (S.I.) could not complete all of the trials in Study One due to fatigue. The clinical characteristics of the Parkinson's disease subjects as well as their medication are summarized in Tables 1–3.

The control group for Study One consisted of nine men and seven women, with a mean age of 72.7 years (range 63–

Table 1 *Parkinson's disease subject characteristics for Study One*

Subject	Age (years)	Sex	Height (m)	Weight (kg)	Webster ratings	STMS*	Medication	(mg day^{-1})
1	81	M	1.75	82	16	32	Sinemet	600/150
2	64	F	1.60	53	10	36	Madopar	500/125
3	64	M	1.78	81	12	23	Madopar M	100/25
							Madopar HBS	100/25
							Parlodel	15
4	67	M	1.76	79	9	33	Sinemet	1000/250
							Sinemet CR	200/50
5	79	F	1.58	60	18	31	Sinemet	1000/250
							Madopar HBS	300/75
6	76	M	1.71	68	24	34	Madopar M	300/75
							Domperidone	20
7	71	F	1.54	49	14	33	Sinemet	1200/300
							Parlodel	7.5
							Motilium	60
8	70	M	1.73	70	16	24	Sinemet	500/125
							Parlodel	12.5
9	75	M	1.69	65	20	24	Madopar	800/200
							Sinemet CR	2000/500
10	78	M	1.66	66	11	26	Madopar	900/225
							Sinemet CR	600/150
							Eldepryl	5
11	73	F	1.61	64	16	29	Parlodel	5
							Madopar	300/75
12	73	F	1.55	60	15	33	Sinemet	200/50
							Sinemet CR	1200/300
13	77	F	1.54	57	12	25	Sinemet	100/25
							Sinemet CR	1800/450
14	69	M	1.70	70	13	21	Sinemet	300/75
							Parlodel	7.5
15	75	F	1.57	56	12	33	Madopar HBS	400/100
							Parlodel	15
							Eldepryl	5
16	72	M	1.68	70	20	28	Madopar	1250/312.5
							Sinemet CR	200/50
							Motilium	30

*STMS = Short Test of Mental Status.

82 years), a mean height of 1.65 m (range 1.51–1.8 m) and mean weight 68 kg (range 49–87 kg). The 16 control subjects for Study Two comprised nine men and seven women of age range 63–81 years (mean 73.1 years), height range 1.45–1.8 m (mean 1.66 m) and weight range 45–90 kg (mean 69 kg). For Study Three the control subjects comprised four men and four women of age range 59–77 years (mean 70.1 years), height range 1.53–1.82 m (mean 1.66 m) and weight range 47–99.2 kg (mean 70.4 kg). Control subjects were healthy and had no history of neurological, orthopaedic or cardiorespiratory disorders.

Apparatus

In each of the three studies a computerized clinical stride analyser (CSA) (B and L Engineering, Santa Fe Springs, Calif., USA) was used to measure the temporal (timing) and spatial (distance) parameters of the footstep pattern. The system consists of a set of footswitches worn as innersoles inside the shoes, a microcomputer backpack for temporary data storage and an IBM compatible personal computer. Details of the apparatus have been documented previously (Morris *et al.*, *1994a*). Data was collected on a 12 m gait walkway in the Geriatric Research Unit at the Kingston Centre, Australia. The walkway was tiled in grey linoleum and purposely included 5 m of open space at either end and on each side so that Parkinson's disease subjects did not feel that they were walking in a confined space, which can trigger motor blocks. Visual cues used in each of the experiments consisted of white strips of cardboard which measured 1 mm×50 mm×500 mm. The visual cues were placed on the gait walkway at a distance equivalent to the step length for each Parkinson's disease subject's age-, sex- and height-matched control.

Procedure

The overall procedure for gait analysis was the same for each of the experiments. Footswitches were placed inside the sole of each shoe and attached to the microcomputer backpack

Table 2 *Parkinson's disease subject characteristics for Study Two*

Subject	Age (years)	Sex	Height (m)	Weight (kg)	Webster ratings	STMS*	Medication	(mg day^{-1})
1	76	M	1.71	68	24	34	Madopar M	300/75
							Motilium	60
2	76	M	1.70	80	16	28	Madopar	900/225
3	80	F	1.51	58	11	30	Sinemet M	100/10
							Madopar HBS	400/100
							Eldepryl	5
4	65	M	1.52	62	21	26	Parlodel	15
							Sinemet CR	800/200
							Sinemet M	100/10
5	77	F	1.54	57	17	25	Parlodel	15
							Sinemet	250/25
							Sinemet CR	500/125
6	71	M	1.68	85	7	30	Sinemet M	500/50
7	81	M	1.70	80	13	32	Sinemet CR	800/200
							Sinemet	150/37.5
8	82	F	1.60	57	10	28	Madopar	450/112.5
							Motilium	20
9	70	M	1.73	70	16	24	Sinemet CR	500/125
							Parlodel	12.5
10	68	M	1.75	82	9	30	Madopar	600/150
							Sinemet CR	200/50
							Eldepryl	10
11	75	F	1.57	56	11	33	Sinemet CR	800/200
							Sinemet CR	300/75
							Eldepryl	10
							Madopar HBS	400/100
							Pergolide	100 mcg
12	81	M	1.69	72	12	32	Sinemet	250/25
							Sinemet CR	800/200
							Pergolide	375 mcg
13	71	F	1.54	49	13	33	Sinemet	1250/125
							Parlodel	7.5
							Motilium	60
14	72	M	1.68	70	17	28	Sinemet CR	200/50
							Madopar	600/150
15	64	M	1.78	81	14	23	Madopar M	100/25
							Madopar HBS	300/75
							Parlodel	15
16	76	F	1.69	70	14	26	Madopar Q	150/37.5

*STMS = Short Test of Mental Status.

storage unit, worn on a belt at the waist. A hand-held trigger which was used to signal the start and end of each trial was then attached to the backpack. For each gait, trial subjects walked the full length of the 12 m walkway and data was collected for the final 10 m of each walk. After each trial the data was down loaded onto an IBM compatible PC and analysed using CSA software version 6 (B and L Engineering).

The primary dependent variables analysed were the gait velocity (m min^{-1}), walking cadence (steps min^{-1}), mean stride length (m) which is equal to the length of two consecutive steps, and the percentage of the gait cycle spent in the double limb support (DS) phase during which both feet are in contact with the ground. Although Parkinson's disease subjects can sometimes exhibit a forward flexed posture and a plantargrade gait, these disorders were not present in our subjects as reflected by the finding that none of the subjects had a score >2 on the posture or gait items of the Webster (1968) scale.

The procedure, statistical analysis, results and a brief discussion of the results of each study are next presented separately, followed by a general discussion at the end of the paper.

Study One

Aim and procedure

The primary aim of the first study was to explore whether the effect of training Parkinson's disease subjects to walk to normal gait parameters with external cues utilized a different or similar mechanism to training Parkinson's disease subjects

Table 3 *Parkinson's disease subject characteristics for Study Three*

Subject	Age (years)	Sex	Height (m)	Weight (kg)	Webster ratings	Medication	(mg day^{-1})
1	68	M	1.75	82	9	Madopar	600/150
						Sinemet CR	200/50
						Eldepryl	10
2	71	F	1.54	49	13	Sinemet	1200/300
						Parlodel	7.5
						Motilium	60
3	76	F	1.69	70	14	Madopar Q	150/37.5
4	69	M	1.68	70	13	Sinemet	300/75
						Parlodel	7.5
5	75	M	1.68	65	20	Madopar	800/200
						Sinemet CR	2000/500
6	75	F	1.57	56	12	Madopar HBS	400/100
						Parlodel	15
						Eldepryl	5
7	64	M	1.78	81	14	Madopar M	100/25
						Madopar HBS	300/75
						Parlodel	15
8	70	M	1.73	70	16	Sinemet	500/125
						Parlodel	12.5

to walk to normal gait parameters with a specific attentional strategy. This was addressed by measuring the extent to which normal gait parameters were attained with the two training strategies and by measuring the duration of maintenance of normal gait once the training strategies were removed. The external cues were visual cues set on the floor at a distance equivalent to the mean stride length of each Parkinson's disease subject's age-, sex- and height-matched control. These floor markers were laminated strips of white cardboard that measured 1 mm×50 mm×500 mm. For the attentional strategy, subjects were trained to focus their attention consciously on walking with steps of the same size as their matched control. They were shown the optimal step length by placing a tape measure and two white lines on the floor at the desired distance and were instructed to 'try to walk with steps this size'. They were also instructed to stand next to the lines and to 'measure up' their step length prior to each trial until they had developed 'a mental picture of the correct step size'.

Prior to measurement of Parkinson's disease subjects, baseline data for the preferred gait pattern of control subjects were collected for two trials spaced 2 min apart. The mean data for each of the parameters was used as a criterion against which Parkinson's disease performance could later be compared. These criterion parameters are represented as a dotted horizontal line on Figs 1–3. The criterion was obtained in order to identify a normative standard against which parkinsonian gait data could be interpreted. In concert with the literature on human locomotion (e.g. Winter, 1991) we assumed that normal gait parameters in control subjects would remain stable over brief durations, such as the experimental 3 h period.

Study One comprised three experiments which documented the effects of visual cue training, attentional strategy training

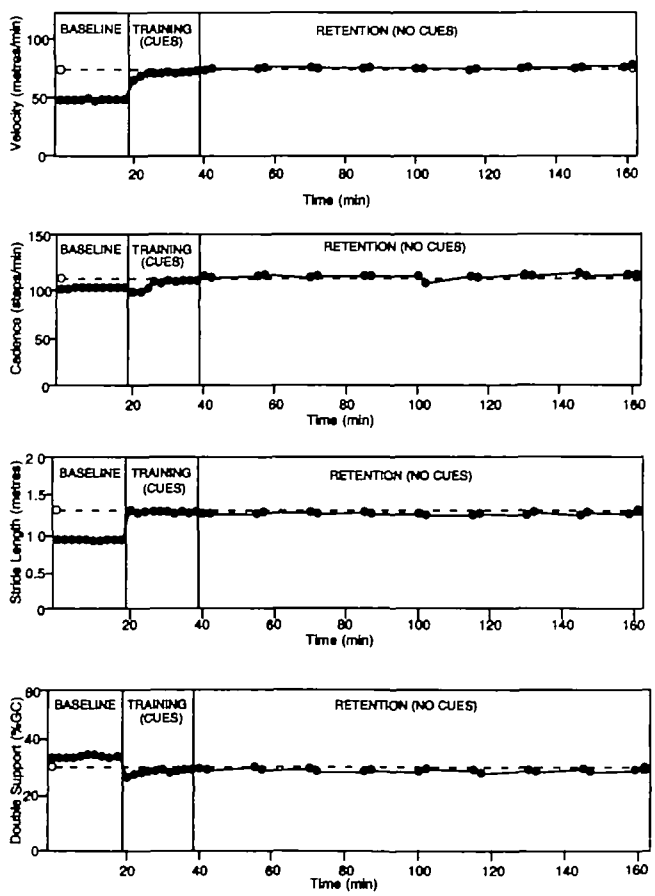

Fig. 1 Mean stride length, cadence, velocity and DS duration for the Parkinson's disease group (closed circles) for baseline, visual cue training (cues) and retention phases, compared with the baseline control values (open circle).

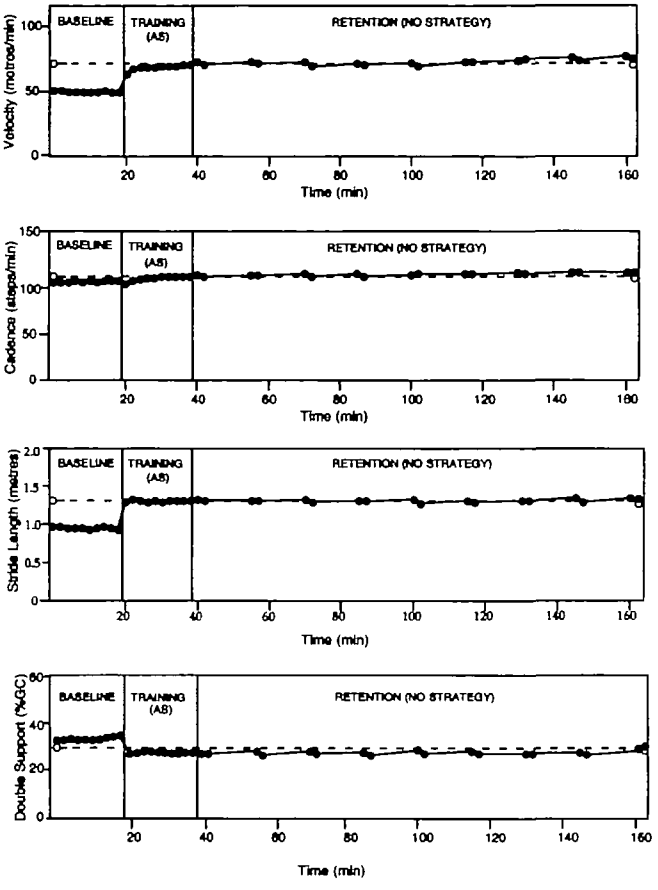

Fig. 2 Mean stride length, cadence, velocity and DS duration for the Parkinson's disease group (closed circles) for baseline, attentional strategy training and retention phases, compared with the baseline control values (open circle).

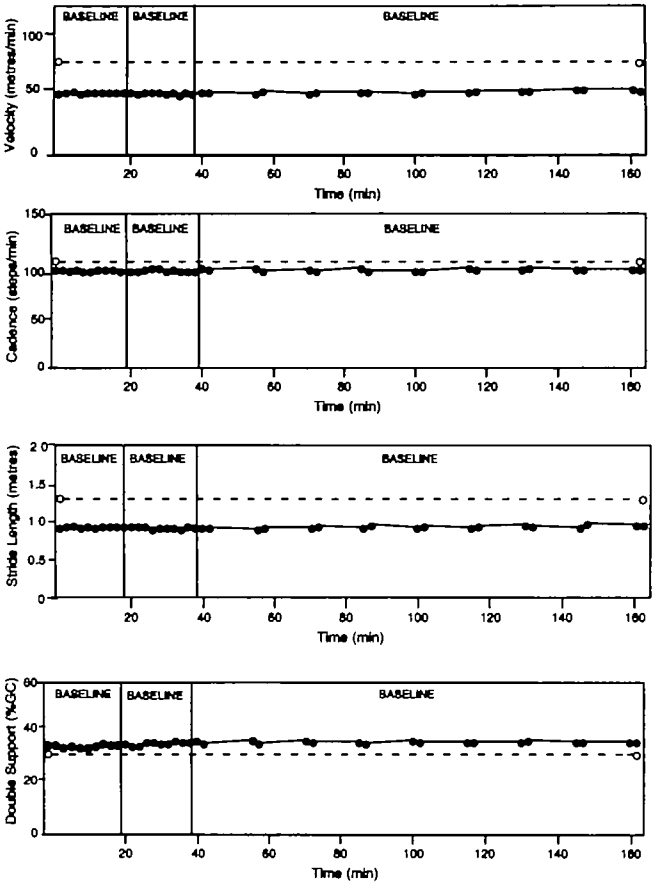

Fig. 3 Mean stride length, cadence, velocity and DS duration for the Parkinson's disease group (closed circles) and the control group (open circle) for baseline conditions.

and an extended baseline condition without training. The three experiments were conducted on separate days over a 9-week period, with average intervals of 3 weeks between each experiment. To control for series effects, the order of testing for the three experiments was randomized for all of the subjects. For the visual cue condition, baseline data were first collected every 2 min for a 20-min period. The instructions for baseline measurement trials were simply to 'Walk to the end of the walkway'. Visual cues spaced at the mean stride length of each Parkinson's disease subject's assigned control were then placed on 10 m of the walkway and subjects were instructed to 'Step over the markers and walk to the end of the walkway'. Trials of visual cue walking were performed at 2-min intervals for 20 min to complete the training programme and measures of gait performance were obtained simultaneously. Finally, in the retention phase subjects performed two gait trials every 15 min for 2 h. Subjects were instructed to 'Walk to the end of the walkway'. Between trials subjects sat in a chair and rested.

For the attentional strategy condition Parkinson's disease subjects were again first tested under baseline conditions every 2 min for 20 min. Then they were familiarized with the target stride length using the attentional strategy outlined above. Parkinson's disease subjects performed gait trials every 2 min for 20 min during which they were instructed to focus their attention on walking with the criterion stride length. Between each of the intervention trials they were re-familiarized with the criterion. To determine retention of training, subjects were then tested twice every 15 min for 2 h without re-exposure to the criterion stride length. Between trials, subjects sat in a chair and rested.

One final condition, an extended baseline, was incorporated into Study One to investigate the temporal stability of the gait pattern in Parkinson's disease. The spatial and temporal parameters of gait were measured every 2 min for 40 min and then twice every 15 min for 2 h. During these 10 m gait trials subjects were instructed to 'Walk to the end of the walkway' and between each trial they were instructed to sit down and rest.

Statistical analysis

Time series analysis was used to test for change in Parkinson's disease gait parameters across each condition, given that the data could be fitted using a linear model. Regression analyses and confidence intervals were used to examine for statistically significant differences between gait parameters for baseline,

Table 4 *Means and 95% confidence intervals (in brackets) for gait velocity, stride length, cadence and DS duration for preferred walking in control subjects and for baseline, visual cue training and retention conditions in Parkinson's disease subjects*

		Parkinson's disease		
Parameter	Control	Baseline	Visual cues	Retention
Velocity (m min^{-1})	73.7 (67.8–79.5)	49.7 (49.4–50.0)	72.2 (70.7–73.7)	76.2 (75.8–76.7)
Stride length (m)	1.31 (1.23–1.39)	0.96 (0.95–0.97)	1.30 (1.30–1.31)	1.30 (1.29–1.30)
Cadence (steps min^{-1})	112.2 (107.8–116.6)	104 4 (103.8–105.0)	108.5 (104.9–112.0)	116.9 (115.8–117.9)
DS (% gait cycle)	31.7 (29.7–33.8)	35.9 (35.6–36.2)	30.2 (29.5–30.9)	31.0 (30.8–31.3)

intervention and retention phases. After linear fits of each phase, the slope and phase transition intercepts were used in a series of planned comparisons using the *t* test for correlated samples. Because the linear models in each condition were consistently found to have zero slope, and because there was no evidence of serial dependence on computing individual autocorrelograms for lags 1–5 (Box and Jenkins, 1976), the mean performance for each of the phases could be compared by computing 95% confidence intervals. The presence of different inter-trial intervals from trials 23 onwards was accommodated in the linear regression by using exact starting times for the abscissa and by confining the estimation of autocorrelograms to the first 20 points which were spaced at even inter-trial intervals.

Results

Figure 1 illustrates the mean time series for baseline stride length, cadence, velocity and double limb support duration for preferred walking, visual cue gait and retention trials in the Parkinson's disease group, compared with the criterion gait pattern for preferred walking in the control group. The figure shows that during the baseline phase Parkinson's disease subjects walked with a short-stepped, slow velocity gait pattern and the percentage of the gait cycle spent in double limb support duration was increased relative to normal. Analysis of the residuals from the linear model for the baseline data showed no statistically significant autocorrelation for any of the Parkinson's disease subjects, indicating stability of performance. Figure 1 also shows that visual cue training led to normalization of the gait pattern in Parkinson's disease and the effects of training were retained for a 2 h period.

Table 4 summarizes the 95% confidence intervals for each of the gait parameters for preferred walking in the control group as well as the 95% confidence intervals for baseline, visual cue walking and retention trials in the Parkinson's disease group. Planned comparisons revealed a significant difference in Parkinson's disease stride length, cadence, velocity and DS from baseline to intervention phases, yet no significant change in the walking pattern from intervention to retention phases. For stride length, the mean intercept for Parkinson's disease baseline performance (0.96) obtained on regression analysis was significantly different at the phase transition from the intervention intercept (1.33) [$t(14) = 13.2$, $P = 0.0001$] even though the difference in slopes across phases was not statistically significant (–0.0005 compared with –0.0008). The retention phase stride length intercept (1.30) and slope (–0.0001) were not significantly different from the intervention phase intercept and slope. The results for Parkinson's disease velocity and DS closely mirrored the trends for stride length. There was a significant increase in velocity intercept from baseline (49.8) to intervention phases (63.8) [$t(14) = 4.7$, $P = 0.001$] and a significant decrease in DS intercept from baseline (35.7) to intervention phases (27.4) [$t(14) = -4.7$, $P = 0.001$), although no significant change in the parameters from intervention to retention phases occurred. In contrast, the differences in cadence slope and intercepts from baseline to intervention phases and from intervention to retention phases were not statistically significant.

Figure 1 also illustrates that Parkinson's disease stride length for the baseline phase was significantly less than for the control group [$t(14) = -12.82$, $P = 0.0001$], as was velocity [$t(14) = -8.99$, $P = 0.0001$]. However, the difference between groups for cadence and DS at baseline was not statistically significant. Visual cues elicited normal gait velocity, cadence, stride length and DS duration in the majority of subjects. As illustrated by Fig. 1, none of the parameters were significantly different from normal under visual cue conditions. The figure also shows that during the retention phase, these parameters were not statistically different from criterion.

One of the unexpected findings of Study One was that gait training using an attentional strategy produced similar results to visual cue training (Fig. 2). This finding is detailed in Table 5 which lists 95% confidence intervals for each of the gait parameters for preferred walking in the control group compared with the 95% confidence intervals for baseline walking, attentional strategy walking and retention trials in the Parkinson's disease group. On regression analysis it was apparent that the baseline Parkinson's disease stride length intercept (0.98) was significantly different from the intervention intercept (1.32) [$t(14) = 5.5$, $P = 0.001$] even though the difference in slopes across phases was not statistically significant [–0.001 compared with 0.0003). The retention stride length intercept (1.33) and slope (–0.0002)

Table 5 *Means and 95% confidence intervals (in brackets) for gait velocity, stride length, cadence and DS duration for preferred walking in control subjects and for baseline, attentional strategy training and retention conditions in Parkinson's disease subjects*

		Parkinson's disease		
Parameter	Control	Baseline	Attentional strategy	Retention
Velocity (m min^{-1})	73.7 (67.8–79.5)	52.3 (51.9–52.6)	71.4 (70.1–72.7)	75.5 (74.3–76.7)
Stride length (m)	1.31 (1.23–1.39)	0.97 (0.96–0.98)	1.32 (1.32–1.33)	1.33 (1.32–1.34)
Cadence (steps min^{-1})	112.2 (107.8–116.6)	107.4 (106.9–108)	110.5 (108.7–112.4)	115 (114.3–115.8)
DS (% gait cycle)	31.7 (29.7–33.8)	35.9 (35.6–36.3)	29.9 (29.6–30.3)	29.7 (29.4–30.1)

were not significantly different from intervention. Similarly, there was a statistically significant increase in the Parkinson's disease velocity slope from baseline (52.4) to intervention (64.1) [$t(14) = 3.9, P = 0.001$] and a decrease in DS intercept from baseline (35.6) to intervention trials (30.6) [$t(12) = -3.68, P = 0.01$], although no significant difference from intervention to retention trials was evident. For cadence, the differences in slope and intercept from baseline to intervention and from intervention to retention phases were not statistically significant.

Figure 2 also illustrates that the Parkinson's disease stride length for the baseline phase was significantly less than the control group [$t(14) = 10.2, P = 0.0001$], as was velocity [$t(14) = -6.2, P = 0.0001$]. Moreover, DS was significantly greater in the Parkinson's disease group than the control group in the baseline phase [$t(12) = 2.2, P = 0.05$]. However, the difference between groups for cadence at baseline was not statistically significant. The attentional strategy produced normal gait velocity, cadence, stride length and DS duration in the majority of subjects. As shown by Fig. 2, during attentional strategy training the Parkinson's disease stride length, velocity, cadence and DS were not significantly different from normal. Figure 2 also shows that during the retention phase these parameters were not statistically different from criterion.

Figure 3 depicts gait performance for the extended baseline condition. The time series shows considerable stability over the testing period and the data appeared to fit a linear model well. Analysis of the residuals from the linear model showed no statistically significant autocorrelation for any of the individual Parkinson's disease subjects. In addition the mean slope and intercept values from regression analyses for the Parkinson's disease group showed no significant changes between baseline and intervention phases or between intervention and retention conditions. Across all three phases, the mean intercept for Parkinson's disease stride length was 0.95 and the mean slope was 0.004. The mean intercept for Parkinson's disease cadence was 103.3 and the slope was 0.002. The mean intercept for Parkinson's disease velocity was 48.27 and the slope was 0.02. Finally the mean intercept for Parkinson's disease DS was 35.36 and the mean slope was 0.004.

For the extended baseline condition, group comparisons revealed statistically significant differences between the Parkinson's disease and control group for all four dependent variables in each of the three phases. The 95% confidence intervals for the complete time series for stride length was only 0.945–0.963 m in the Parkinson's disease group compared with 1.24–1.39 m in the control group. In addition, the 95% confidence intervals for cadence for the Parkinson's disease group were only 103.2–104.2 steps min^{-1}, compared with 107.4–115.9 steps min^{-1} in the control group. The 95% confidence intervals for mean velocity in the Parkinson's disease group (49.0–50 m min^{-1}) also indicated that velocity was substantially less than for controls (68.2–79.0 m min^{-1}). Mirroring the velocity results, the 95% confidence intervals for DS duration in the Parkinson's disease group (35.7–36.6% gait cycle) showed that DS was greater in Parkinson's disease subjects than controls (29.5–33.5% gait cycle).

Discussion

Three key findings emerged from Study One. The first was that visual cue training was effective in enhancing stride length regulation in Parkinson's disease. This training effect transferred to walking without visual cues and led to normalization of stride length, velocity, cadence and DS duration for at least 2 h. The positive finding in relation to visual cue training is consistent with previous reports in the literature (Martin, 1967; Forssberg *et al.*, 1984; e.g. Bagley *et al.*, 1991; Morris *et al.*, 1994*a*, *b*) which have detailed immediate performance effects with floor markers. This investigation demonstrated that exposure to visual cues produces more lasting changes in the walking pattern which occurred even when the cues were removed.

The second key finding was that training using an attentional strategy, whereby Parkinson's disease subjects developed a mental picture of the criterion stride size and consciously focused on walking with that stride size, produced results that were strikingly similar to visual cue training. The effects of the attentional strategy also lasted for at least 2 h after training ceased. Therefore the use of visual cues is not the only strategy that is a useful adjunct to pharmacological therapy for gait hypokinesia in Parkinson's disease. Specific attentional strategies appear to be useful in the enhancement of gait performance in Parkinson's disease. Given the practical

requirements for visual cue training, particularly the lack of portability of this treatment method, attentional strategies might be a useful alternative for people who do not have severe cognitive impairment, which could limit ability to develop a mental representation of the target stride amplitude.

A third finding was that the gait pattern of medicated Parkinson's disease subjects remained stable over a 2.5 h period (refer to Fig. 3). This finding, which appears to be one of the first published report of Parkinson's disease gait stability over an extended period, seems to be contrary to the commonly held belief that Parkinson's disease subjects exhibit marked motor fluctuations, depending on medication status and duration of Parkinson's disease (Marsden *et al.*, 1982; Klawans, 1986). For example, Wall and Turnbull (1992, pp. 57–8) noted that 'as the disease progresses the relief obtained [from medication] becomes less predictable and walking becomes affected. The gait profile of the patient during this stage is unpredictable; there is considerable variability between patients at similar stages of the disease and in the same patient from hour to hour and day to day'. However, the locomotor stability in our Parkinson's disease sample might have been enhanced by the exclusion criteria. Those with marked dyskinesia were excluded (a total of five potential subjects), and these people might have exhibited more marked fluctuations across time. Moreover, only people who could perform repeated 10 m gait trials were included, and testing occurred when they were in the 'on' phase of medication, rather at the beginning or end of dose.

The results of Study One also raise important questions regarding the mechanism of action of visual cue training. The traditionally held view was that visual cues enhance the locomotor pattern in Parkinson's disease by triggering each step in the sequence (for example, *see* Martin, 1967). However, this explanation does not account for why the effects of training persist for 2 h despite removal of the cues during the retention period. An alternative explanation is that the repeated performance of normal gait with cues enhances the motor set for the gait sequence, which in turn enhances the gain of the motor system for normal stride length. Thus the repeated performance of the same motor pattern might enable the pattern to be maintained after removal of the cues. The other possibility is that external cues may simply draw attention to the correct stride length, thereby increasing the step size. This concept is strengthened by the finding that the attentional strategy also elicited normal gait parameters and enabled subjects to maintain the improvement without re-exposure to the criterion stride size. The similarity in behaviour type and duration of effect post training for visual cues and the attentional strategy suggests that both strategies might share a similar mechanism, which may bypass the basal ganglia–SMA interaction.

In the second study we tested this latter hypothesis by investigating the effects of secondary task performance on maintenance of the gait pattern in the retention period following training. It seems plausible that use of a secondary task removes attentional mechanisms from the most automatic movement (gait) and directs them to the attention requiring task (in this case a cognitive task) (*see* Dalrymple-Alford *et al.*, 1994). The result is that the automatic task would likely use the basal ganglia–SMA interaction and thus gait parameters should deteriorate. In contrast, if visual cues simply act to 'trigger', successive steps within the sequence (i.e. to substitute for the disordered phasic basal ganglia cue) then no deterioration in gait parameters would be expected on performance of a secondary task.

Study Two

In this study we examined whether visual cues training and attentional strategy training had similar or different effects on gait by examining the effects of secondary task performance on the walking pattern during the retention phase following training. The concept of attention is difficult to operationalize, however, for the purposes of this investigation we defined attention as a limited capacity for processing information from either memory or external sources. Schmidt (1983, p. 133) argued that attention can be indexed by 'the degree of interference between two tasks'. In line with this viewpoint, we predicted that if attention was directed towards a secondary task following training, then the stride length would revert to baseline levels. It was also predicted that when the secondary task was withheld, stride length would revert to criterion levels because the patient would be able to direct attention back to the footstep pattern. Finally, we predicted that changes in cadence would not be significantly different from normal because the internal regulation of cadence is not defective in Parkinson's disease (Morris *et al.*, 1994*b*).

Procedure

Two experiments, conducted on consecutive days, were performed for Study Two. In the first experiment, we collected two trials of baseline data for each Parkinson's disease subject and then 10 trials of visual cue training followed by two retention trials where the cues were removed and subjects were instructed to 'walk to the end of the walkway'. Four gait trials were then performed concomitant with a series of secondary cognitive tasks of graded levels of attentional demand. The 10 m gait trials were conducted every 2 min although subjects could request a longer duration rest if required. During secondary task performance, subjects were instructed to walk normally whilst they concentrated on reciting a sentence. The Parkinson's disease data were compared with a normative standard set by control subjects, represented by a horizontal line on the figure. The line was obtained as a criterion level based on the average of two trials of preferred gait, conducted 2 min apart.

In the second experiment in Study Two, which was performed the following morning, the same procedure was used except visual cue training was replaced by training using the attentional strategy previously described. The order

Table 6 *Means and 95% confidence intervals (in brackets) for gait parameters for the control group (criterion) and Parkinson's disease group under each of the secondary task conditions (T1–T4) following visual cue training*

		Parkinson's disease			
Parameter	Criterion	T1	T2	T3	T4
Velocity (m min^{-1})	72.65 (66.9–78.4)	70.8 (65–76.7)	65.4 (59.6–71.2)	58.4 (52.7–63.2)	56.3 (49.5–63.2)
Cadence (steps min^{-1})	112.8 (108.1–117.5)	114.7 (108.9–120.6)	111.3 (105.4–117.1)	108.9 (102.8–114.9)	104.2 (97.6–110.5)
Stride length (m)	1.29 (1.22–1.36)	1.23 (1.16–1.30)	1.18 (1.09–1.26)	1.07 (0.98–1.16)	1.05 (0.95–1.15)
DS (% gait cycle)	32.2 (29.6–34.9)	32.45 (29.3–36.5)	34.4 (32.3–36.5)	35.3 (33.5–37.1)	35.7 (33.4–38)

of experiments (visual cue training or attentional strategy) was counterbalanced using a Latin square design in order to control for series effects. In a similar way, the order of presentation of secondary cognitive tasks in both studies was randomized to control for the effects of fatigue, practice and historical variables.

Further data was collected for eight of the control subjects who walked at their preferred gait and under each of the secondary task conditions. The purpose of collecting this data was to ascertain whether the gait patterns of healthy elderly people remain stable when they perform dual tasks of increasing levels of complexity. These control subjects comprised four men and four women of age range 59–77 years (mean 70.1 years), height range 1.53–1.82 m (mean 1.66 m) and weight range 47–99.2 kg (mean 70.4 kg).

The secondary tasks used in Study Two were three items selected from the Benton and Hamsher (1983) sentence repetition stimuli and one additional cognitive task. The Benton and Hamsher stimuli provide a selection of verbal tasks with increasing levels of complexity that presumably place increasing levels of demand on the attentional system. For the easiest secondary task (T1), subjects were asked to recite repeatedly, 'Where is the child?' as they walked. The second level of complexity (T2) was to recite repeatedly, 'Work in the garden until you have picked all the beans'. The third task (T3) was to recite, 'The members of the committee have agreed to hold their meeting on the first Tuesday of each month'. In the most difficult cognitive task (T4) subjects were requested to repeat the days of the week backwards as they walked.

Statistical analysis

The strategy for data analysis for Study Two was similar to the first study. Visual inspection of the individual data for each subject confirmed that the trends in baseline and intervention stages in Study Two were very similar to the trends in baseline and intervention stages in Study One. Because a linear model was appropriate for the data in each phase, means and 95% confidence intervals for stride length, velocity, cadence and DS duration could be calculated for each of the conditions for the experimental group and then compared with the criterion values established for the control group. A series of planned comparisons employing the *t* statistic for correlated samples was used to address the following key issues: (i) whether there was a statistically significant difference between the Parkinson's disease group and criterion for baseline gait, visual cue gait, retention performance and secondary task performance following visual cue training; (ii) whether there was a statistically significant difference between the Parkinson's disease group and criterion for baseline gait, attentional strategy gait, retention performance and secondary task performance following attentional strategy training.

Results

Figure 4 shows the mean values for stride length, cadence, velocity and DS in the Parkinson's disease group compared with the control group for the visual cue training study. As suggested by the figure, in the baseline phase there was a statistically significant difference between the groups for stride length [$t(14) = 8.9$, $P = 0.0001$], velocity [$t(15) = 8.62$, $P = 0.0001$], cadence [$t\ (15) = 3.09$, $P = 0.007$] and DS duration [$t(15) = -2.67$, $P = 0.017$]. However, for visual cue training and the retention tests (R1, R2, R3) there was no significant difference between groups for any of the dependent variables.

During secondary task performance following visual cue training, significant changes in stride length and velocity were obtained (Table 6). For the easiest secondary task (T1) where subjects simply repeated 'Where is the child' as they walked, the results for stride length for the Parkinson's disease group were not significantly different from the preferred gait of controls. However, the mean stride length for the Parkinson's disease group was less than criterion for T2 [$t(14) = 2.66$, $P = 0.02$], T3 [$t(15) = 5.51$, $P = 0.0001$] and T4 [$t(15) = 5.88$, $P = 0.0001$]. Likewise, the mean velocity for the Parkinson's disease group was significantly less than normal for T2 [$t(14) = 3.11$, $P = 0.008$], T3 [$t(15) = 5.47$, $P = 0.0001$] and T4 [$t(15) = 5.17$, $P = 0.0001$]. In contrast, the mean cadence for the Parkinson's disease group was not significantly different from criterion at T1, T2 or T3, even though it was significantly slower than the control group at T4 [$t(15) = 3.12$, $P = 0.007$].

Of note, the reduction in stride length and velocity over the secondary task conditions was proportional to the complexity of the secondary task. In addition, when

Table 7 *Means and 95% confidence intervals (in brackets) for gait parameters for the control group (criterion) and Parkinson's disease group under each of the secondary task conditions (T1–T4) following attentional strategy training*

		Parkinson's disease			
Parameter	Criterion	T1	T2	T3	T4
Velocity (m min^{-1})	72.7 (66.9–78.4)	73.6 (66–81.2)	70.7 (62.6–78.8)	64 (55.4–66.9)	58.9 (50.9–66.9)
Cadence (steps min^{-1})	112.8 (108.1–117.5)	114.5 (107.4–121.6)	112.6 (105.7–119 6)	111.1 (103.8–118.5)	106 (99 4–112.7)
Stride length (m)	1.29 (1.22–1.36)	1.28 (1.20–1.35)	1.25 (1.16–1.33)	1.15 (1.05–1.24)	1.10 (1.00–1.20)
DS (% gait cycle)	32.2 (29.6–34.9)	31.2 (28.3–34)	31.4 (28.8–34.1)	32.9 (30.2–35.7)	33.4 (30.8–36)

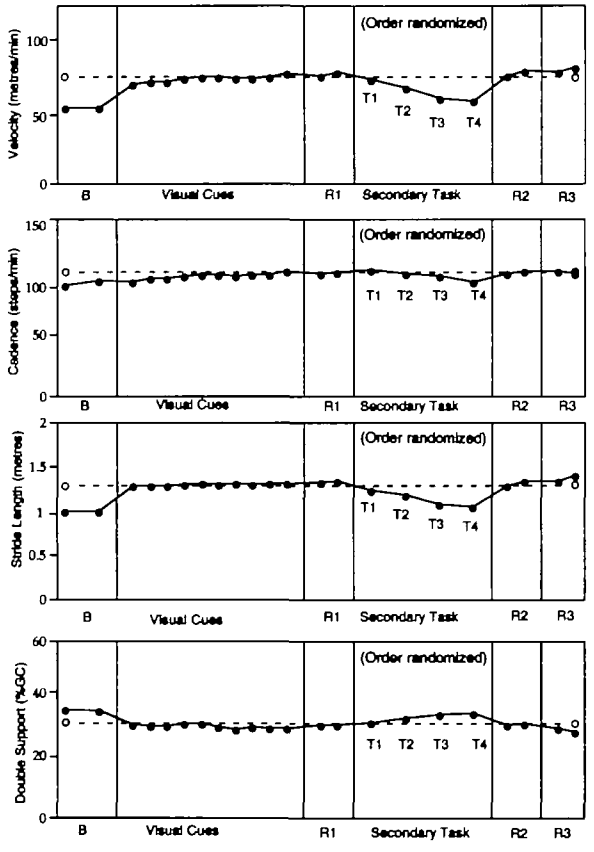

Fig. 4 Mean stride length, cadence, velocity and DS duration for the Parkinson's disease group (closed circles) for baseline, visual cue training, secondary task and retention (R) phases, compared with the baseline control values (open circle).

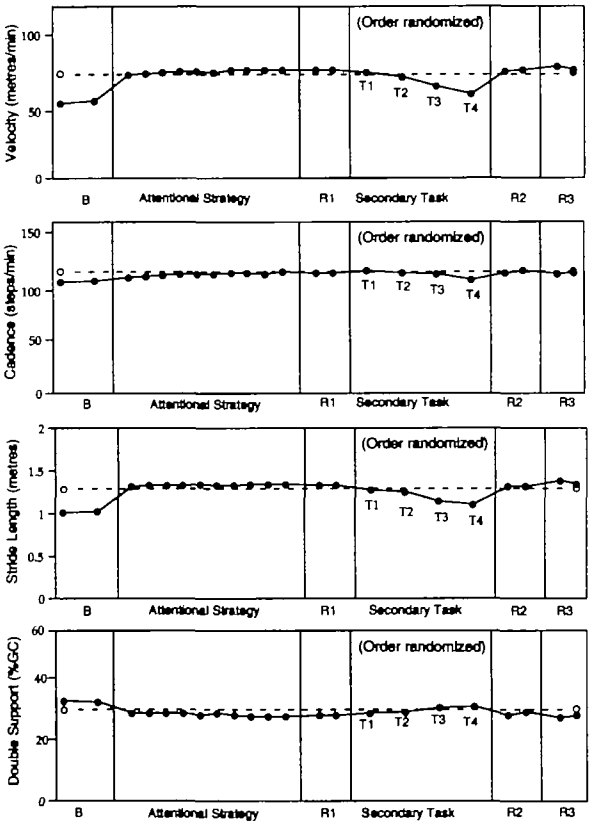

Fig. 5 Mean stride length, cadence, velocity and DS duration for the Parkinson's disease group (closed circles) for baseline, attentional strategy training, secondary task and retention (R) phases, compared with the baseline control values (open circle).

Parkinson's disease subjects ceased the secondary tasks and performed retention trials, all of the gait parameters reverted to values that were not significantly different from criterion.

In the second experiment in Study Two we replicated the procedure except that the visual cues were replaced by training with the attentional strategy described in Study One (Table 7). As shown in Fig. 5, the results closely approximated those illustrated in Fig. 4. The main findings can be summarized as follows. For baseline walking there was a statistically significant difference between Parkinson's disease subjects and controls for stride length, cadence, velocity and DS. With the attentional strategy the means for the Parkinson's disease group for all dependent variables were not significantly different from normal. At T1 the differences between the Parkinson's disease group and controls for the four gait variables did not reach statistical significance. However, there was a significant difference between groups for mean stride length at T2 [$t(15) = 2.27$, $P = 0.04$], T3 [$t(14) = 5.07$, $P = 0.0002$] and T4 [$t(15) = 4.9$, $P = 0.0002$]. The mean Parkinson's disease velocity at T3 was also significantly different from controls [$t(14) = 3.35$, $P = 0.005$] as was the mean Parkinson's disease velocity at T4 [$t(15) = 4.72$, $P = 0.0003$]. However, the differences in cadence and DS between the Parkinson's disease group and the control group did not reach statistical significance for T1–T4.

For the eight control subjects who performed the secondary task conditions, there were no significant differences in any

Table 8 *Means and 95% confidence intervals (in brackets) for gait parameters for eight control subjects for preferred gait, secondary tasks (T1–T4) of progressive difficulty and covertly monitored gait*

Parameter	Preferred gait	T1	T2	T3	T4	Covert
Velocity (m min^{-1})	82.9 (73.8–91.9)	84.7 (72–97.4)	83.4 (70.5–96.3)	84.4 (71.5–97.2)	78.9 (68.7–89.2)	83.17 (70.5–95.8)
Cadence (steps min^{-1})	116.7 (110.9–122.4)	117.1 (107.8–126.5)	116.6 (106.6–126.5)	117.5 (109.1–125.8)	113.8 (104.4–123.2)	117.8 (109.7–125.9)
Stride length (m)	1.42 (1.3–1 53)	1.44 (1.30–1.58)	1.42 (1 29–1.55)	1.43 (1.27–1.59)	1.38 (1.28–1.49)	1.4 (1.27–1.54)
DS (% gait cycle)	31.9 (28.4–35.4)	31.3 (27.8–34.9)	31.7 (27.6–35.8)	32.3 (27 8–36.8)	32.3 (28.3–36.2)	31.13 (27.1–35.1)

of the gait parameters from preferred walking to T1, T2, T3 or T4 (*see* Table 8).

Discussion

The main finding of Study Two was that secondary task performance during the period following training led to a decrement in Parkinson's disease stride length and velocity that was proportional to the complexity of the task performed. However, the changes in Parkinson's disease cadence from the training phase to the secondary task phase were not statistically significant, except for the most difficult task, T4. This effect was seen for both visual cues and the attentional strategy, suggesting that both strategies may normalize gait by using similar mechanisms. During secondary task performance, one task is theoretically relegated to automatic control whilst attention is directed to the other. When Parkinson's disease subjects focused their attention on reciting sentences from the Benton and Hammer (1983) battery the walking pattern deteriorated, presumably because it was relegated to automatic control processes which involve the basal ganglia-thalamo-cortical circuitry and this circuitry is defective in Parkinson's disease. In two Parkinson's disease subjects (S9 and S10) the degree of interference for the most difficult cognitive task was so disabling that they experienced motor blocks midway through some of the gait trials and could not complete the test due to severe 'freezing'. The finding that the degree of performance deficit was proportional to the difficulty of the secondary task suggests that improvements with training were largely due to an increase in attention on the criterion stride length, which led to normalization of the other gait parameters. The findings are in agreement with previous studies on Parkinson's disease which have shown that performance declines during dual task conditions (e.g. Schwab *et al.*, 1954; Talland and Schwab, 1964; Brown and Marsden, 1991; Dalrymple-Alford *et al.*, 1994).

Control subjects also showed a slowing of the walking pattern accompanied by a small reduction in stride length and cadence in the most difficult secondary task conditions (Table 8). However, for the control group the changes in gait parameters from preferred walking to the secondary task conditions were not statistically significant.

Another finding of interest was that gait performance on the second day of testing had reverted to baseline conditions for all Parkinson's disease subjects following both rehabilitation methods. The reason why patients did not use their newly acquired attentional strategy in order to generate a normal gait pattern the next day remains unclear, although it is interesting that our subsequent clinical observations indicated that patients can generate the learned pattern the next day when prompted to do so. Hence, our clinical impression is that patients can learn an attentional strategy for walking more normally and can elect to use this strategy when there is an external demand to do so, yet when left to self monitor their walking they tend to revert to a typical hypokinetic gait pattern.

The results of Study Two also lend some support to our hypothesis that visual cues act to enhance the gait pattern by directing attention to the criterion stride length rather than by triggering sub-movements in a sequence. This conclusion is based on three main observations. First, during the retention phase of Experiment One a normal gait pattern could be generated even though visual cues were unavailable to 'trigger' stepping. Secondly, the results for the cues experiment and the attentional strategy experiment were very similar, suggesting that similar underlying motor control mechanisms might be responsible for the changes observed. Thirdly, when attention was apparently placed on a secondary speech task following visual cue training, gait performance deteriorated. A plausible explanation for all of these findings is that visual cue training enhanced stride length in Parkinson's disease by focusing attention on the criterion step size.

Alternatively, it could be argued that deterioration in gait performance during dual task conditions resulted from non-attentional mechanisms. Speech tasks, such as those used in the present investigation, might interfere with gait performance because they share a common timing mechanism, or because the timing of locomotion entrains to the natural speech rhythm. There is some evidence to suggest a predisposition for the rhythmicities evident in cyclical actions such as walking to become synchronized with the natural rhythm of speech, either because there is a coupling between neural oscillators or because there is a central timing mechanism that regulates both actions (Summers and Burns, 1990). Because the present investigation did not incorporate measures of speech performance for each secondary task condition, this possibility cannot be discounted. However, the finding that control subject's gait performance did not show significant change according to the complexity of the speech task condition reduces the strength of this line of argument. Furthermore, secondary task performance has been used in non-speech domains in Parkinson's disease and has been shown to produce a similar deterioration in motor

performance for the non-attentional task as shown in our study (Schwab *et al.*, 1954; Benecke *et al.*, 1986). Therefore, it is unlikely that the explanation for the phenomenon can be due to a linking of stepping to the frequency of syllable production of the speech task. In this regard, we used the secondary task technique as an already established tool to explore the role of attention in training with visual cues compared with the attentional strategy.

Study Three

Aim and procedure

Given that gait training with visual cues appears to work in a similar manner to cognitive strategies that utilize attentional mechanisms, effective gait performance in Parkinson's disease may be very dependent on the encouragement of a bystander or therapist to remind the patient to concentrate on stride regulation. Unfortunately, people with Parkinson's disease have to function without such continual reminders and therefore it would be of interest to determine if training effects persist if subjects believe that no recording is actually taking place. Study Three was designed to assess this influence of placebo behaviour on the maintenance of stride length in the retention phase, as demonstrated in Studies One and Two.

Eight Parkinson's disease subjects and eight age-, height- and sex-matched controls were recruited from the Kingston Centre Movement Disorders Clinic. Each subject was tested in a single session using the procedure for gait analysis outlined previously. The gait patterns of control subjects were first recorded, to be used as the criterion for Parkinson's disease gait. The mean of two trials of preferred gait in control subjects was used as a criterion reference against which Parkinson gait performance could be compared and is represented by dotted horizontal lines in Fig. 6.

Parkinson's disease subjects then performed two baseline 10 m walks, eight trials with visual cues and eight retention trials. In half of the visual cue trials, data were gathered as subjects walked over the cues and in the remaining trials data were covertly obtained as subjects walked back to the computer along an adjacent walkway without cues. Similarly, in half of the retention trials data were obtained when subjects believed that they were being tested and in the other half data were collected on the way back to the computer when subjects did not know that they were being measured. The order of overt measurement and covert measurement trials were randomized to control for series effects. The effects of the attentional strategy were not evaluated in this experiment.

Eight control subjects were also tested under overt and covert gait measurement conditions to determine whether expectancy effects related to the measurement process affected the walking pattern. The eight subjects who were used to control for the secondary task conditions in Study Two were recruited for this purpose.

Statistical analysis

The mean values for Parkinson's disease subjects and their 95% confidence intervals were expressed as a percentage of the values obtained for control subjects and a series of pair wise comparisons between the control group and Parkinson's disease groups were conducted using the *t* statistic for correlated samples.

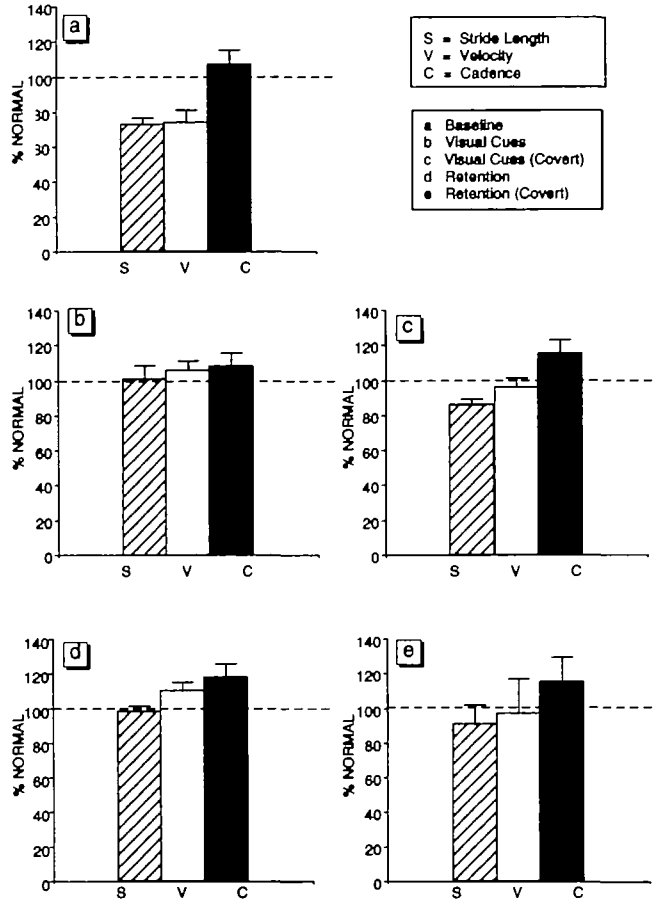

Fig. 6 Means and limits for the 95% confidence intervals for gait velocity, cadence and stride length for Parkinson's disease subjects expressed as a percentage of the values for matched controls.

Results

Figure 6 depicts the mean values and 95% confidence intervals for stride length, velocity and cadence in the Parkinson's disease subjects, expressed as a percentage of the values obtained for age-matched controls for each of the conditions.

For baseline walking, the mean Parkinson's disease stride length was significantly less than normal [$t(7) = -6.7$, $P = 0.0003$]. The mean velocity was also less than criterion [$t(7) = -4.12$, $P = 0.004$] (Fig. 6A). The mean baseline DS duration for the Parkinson's disease group was significantly higher than normal [$t(7) = 2.63$, $P = 0.034$]. However, the mean cadence at baseline for the Parkinson's disease group was not significantly different from the control group.

When Parkinson's disease subjects were measured during performance of the visual cue trials, the values for all four dependent variables were not significantly different from

normal (Fig. 6B). However, when Parkinson's disease subjects were covertly monitored on their walk back to the computer after each visual cue trial, the gait pattern deteriorated (Fig. 6C). There was a statistically significant decrease in mean stride length of Parkinson's disease subjects from the overtly monitored visual cue trials to the covertly monitored visual cue trials [$t(7) = 4.99$, $P = 0.002$]. The mean velocity was also significantly less than the covertly monitored visual cue trials [$t(7) = 3.02$, $P = 0.019$]. The mean cadence and DS did not show significant change.

For the standard retention test (Fig. 6D) none of the gait parameters were significantly different from criterion. However, the difference in stride length from the overtly monitored retention trials to the covertly monitored retention trials was statistically significant [$t(7) = 4.8$, $P = 0.002$]. The mean velocity was also significantly less than the velocity for the overtly monitored retention trials [$t(7) = 3.22$, $P = 0.015$]. The mean Parkinson's disease cadence and DS were not significantly different from the overtly monitored trials.

For the eight control group subjects there were no statistically significant differences in stride length, velocity, cadence or DS from the overt to the covert measurement trials (Table 8).

Discussion

The results for Study Three clearly show that when Parkinson's disease subjects attended to their walking, a normal gait could be achieved with visual cues and could be retained after the cues were removed. However, when there was not a perceived requirement for normal gait performance (e.g. a researcher instructing the patient to perform a measurement trial), the stride length decreased in six of the eight subjects. People with Parkinson's disease were able to generate the criterion gait at will with training. However, when they did not perceive that the walking pattern was being tested, it appears that they did not direct their full attention to their stepping pattern, with a resultant performance decrement, primarily in relation to stride length. These results reinforce our previous hypothesis that the positive effects of visual cues were due to increased attention on the criterion stride size. The results also reinforce the idea that Parkinson's disease subjects can learn an attentional strategy to elicit a normal gait pattern and can use this strategy at will to normalize walking, particularly when they are prompted by an external source to do so. However, over prolonged periods of time without continual encouragement the effect of training may wane to pre-training levels.

General discussion

The main finding to be highlighted by this series of studies was that Parkinson's disease subjects could generate normal stride length by use of visual cues or an attentional strategy. Study One revealed that maintenance of normal gait parameters persisted once training ceased for both strategies and that a normal stepping pattern could be generated for the 2 h of measurement that occurred after training. This finding suggested that both strategies may have utilized similar mechanisms to enhance the walking pattern. In Study Two we examined this point further by the use of simultaneous task performance during the retention phase. Again, very similar patterns of deterioration in gait parameters occurred for visual cues and the attentional strategy and the deterioration in gait parameters was graded according to the level of attention required for secondary task performance. Study Three demonstrated that factors which reminded Parkinson's disease subjects to use attentional mechanisms facilitated the maintenance of normal gait parameters, and waning of attention resulted in deterioration of performance. This latter study highlighted the need for vigilance in the effective use of attentional strategies in normalizing parkinsonian gait.

These findings suggest that people with Parkinson's disease rely heavily on attentional processes to modulate their walking patterns. Although there is prior literature on attentional factors in relation to motor control in Parkinson's disease, this has been obtained in studies of upper limb activities (e.g. Schwab *et al.*, 1954; Talland and Schwab, 1964; Benecke *et al.*, 1986; Bradshaw *et al.*, 1993). To our knowledge, these results represent the first example of research in which the role of attentional processes in the regulation of locomotion in Parkinson's disease has been examined. We do not believe that the upper limb results could have been automatically extended to cover lower limb function given the extensive literature on the differences in CNS mechanisms that apply to gait control. For example, the greater emphasis on spinal and brainstem structures in the generation of locomotion (Grillner *et al.*, 1986; Pearson and Rossignol, 1991) raises sufficient doubt to require original data rather than rely on extrapolation from upper limb movements.

The finding that Parkinson's disease subjects relied heavily on attentional resources to control stride length following training might suggest that the preferred gait pattern had not been properly learned, possibly because they had not undertaken sufficient practice to allow the new pattern to become automatic. One theory of motor learning put forward by Fitts (1964) is that people pass through three stages when acquiring a new skill. The first stage is known as the cognitive phase, during which the person gains an understanding of the global requirements of the task. During the second stage, known as the fixation stage, specific requirements such as the speed, amplitude and force are refined through large amounts of practice. During the fixation stage the person usually needs to closely attend to the movement during performance. By the third stage, known as the automatic phase, the motor skill is well established and can be performed automatically in a range of contexts with limited demands on attentional resources. The pattern of results obtained in this series of investigations might lead some to suggest that Parkinson's disease subjects were in the second stage of motor skill acquisition and the preferred gait pattern had not

yet become automatic. The counter argument is that learning the skill of walking takes place at a very young age and in adults this skill would have progressed to Fitts Stage Three long ago. Moreover, in Parkinson's disease automatic skilled movements cannot be executed normally due to the defective basal ganglia cue which, we have hypothesized, normally interacts with the SMA to string submovements together (Iansek *et al.*, 1995). Consequently, motor mechanisms in Parkinson's disease might revert to more primitive control strategies which place the level of skill acquisition at Stage Two of the Fitts model. We believe our results are consistent with this latter explanation.

This explanation raises the question of whether Parkinson's disease subjects can ever learn to walk permanently with normal stride length, velocity and cadence without consciously focusing on their stepping pattern. Even though we showed short-term performance effects, we also observed that walking had defaulted to baseline levels by the following morning, indicating that the gait changes had not become fully automatic. One possibility is that Parkinson's disease subjects might never be able to generate normal stride length automatically, even though they can learn strategies to elicit a normal stepping pattern consciously at will. Evidence is accumulating that the basal ganglia play a key role in maintaining movement automaticity (Iansek *et al.*, 1995) and when the basal ganglia are disrupted Parkinson's disease subjects cannot properly perform automatic sequential movements such as writing, speaking, swallowing and walking. In a well-known study by Seitz and Roland (1992) the results of PET studies demonstrated that during the early stages of learning a novel finger movement sequence, the cortex, PMA and SMA were metabolically active, whereas the basal ganglia activity was minimal. With repeated practice, the skill eventually became automatic and this was associated with a marked increase in the activity of the basal ganglia activity and a decline in the cortical regions. These results provide some indication that the basal ganglia play a primary role in allowing movement performance to shift from conscious control to automaticity. Whether other areas of the brain also participate in this control process remains open to question. It also remains to be established whether other alternative regions of the CNS can substitute for the basal ganglia in controlling automatic movements such as walking, given optimal practice conditions.

The lack of understanding regarding the neural correlates of movement automaticity is mirrored by an incomplete understanding of the role of the basal ganglia in the control of human locomotion, and in particular, stride length regulation. As outlined in the Introduction, we have hypothesized that the basal ganglia play two key roles in motor control. First, they provide a phasic cue to the SMA that is timed to terminate set related activity in the SMA and, therefore, they play a role in maintenance of adequate movement preparation for submovements performed in a sequence (Brotchie *et al.*, 1991*a*, *b*). Secondly, the basal ganglia contributes to motor set for whole movement sequences and the absent basal ganglia contribution may lead to inadequate gain in motor execution mechanisms which could result in an underscaling of successive steps during gait (*see* Hallett and Khoshbin, 1980). The fact that stride length was responsive to the attentional strategies provides some indication that stride length deficits in Parkinson's disease relate to problems in motor set rather than a deficit in the internal cueing of human locomotion.

This investigation provides the first data in the literature on the effects of attentional mechanisms in relation to training locomotor function in Parkinson's disease. As such, the results carry implications for clinical practice. In agreement with previous reports we showed that practice with visual cues has an immediate performance effect (Bagley *et al.*, 1991; Weissenborn, 1993). Moreover, a cognitive strategy, whereby Parkinson's disease subjects focused their attention on walking with a specified stride length, was equally effective as visual cues. The gains achieved with training were depleted during secondary task performance and locomotion was optimal under supervision. Thus, when patients do not focus their attention on walking with a more normal stride, or when a bystander is not present to remind them to concentrate on their walking pattern, there might be a tendency to revert to habitual movement patterns.

To summarize, these investigations confirm that the ability to generate a normal stepping pattern is not lost in Parkinson's disease, and that gait hypokinesia reflects a difficulty in activating the locomotor control system. Normal stride length can be elicited in Parkinson's disease using attentional strategies and visual cues, possibly because both of these methods focus attention on the criterion stride size.

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References

Aita JF. Why patients with Parkinson's disease fall. JAMA 1982; 247: 515–16.

Bagley S, Kelly B, Tunnicliffe N, Turnbull GI, Walker JM. The effect of visual cues on the gait of independently mobile Parkinson's disease patients. Physiotherapy 1991; 77: 415–20.

Banks MA, Caird FI. Physiotherapy benefits patients with Parkinson's disease. Clin Rehabil 1989; 3: 11–16.

Benecke R, Rothwell JC, Dick JPR, Day BL, Marsden CD. Performance of simultaneous movements in patients with Parkinson's disease. Brain 1986; 109: 739–57.

Benton AL, Hamsher K De S. Multilingual aphasia examination revised. Florida: Psychological Assessment Resources, 1983.

Berardelli A, Dick JPR, Rothwell JC, Day BL, Marsden CD. Scaling of the size of the first agonist EMG burst during rapid wrist movements in patients with Parkinson's disease. J Neurol Neurosurg Psychiatry 1986; 49: 1273–9.

Box GEP, Jenkins GM. Time series analysis: forecasting and control. Rev. ed. San Francisco: Holden Day, 1976.

Bradshaw JL, Waterfall ML, Phillips JG, Iansek R, Mattingley JB, Bradshaw JA. Re-orientation of attention in Parkinson's disease: an extension to the vibrotactile modality. Neuropsychologia 1993; 31: 51–66.

Brotchie P, Iansek R, Horne MK. Motor function of the monkey globus pallidus. 1. Neuronal discharge and parameters of movement. Brain 1991a; 114: 1667–83.

Brotchie P, Iansek R, Horne MK. Motor function of the monkey globus pallidus. 2. Cognitive aspects of movement and phasic neuronal activity. Brain 1991b; 114: 1685–702.

Brown RG, Marsden CD. Dual task performance and processing resources in normal subjects and patients with Parkinson's disease. Brain 1991; 114: 215–31.

Cunnington R, Iansek R, Bradshaw JL, Phillips J. Movement-related potentials in Parkinson's disease: presence and predictability of temporal and spatial cues. Brain 1995; 118: 935–50.

Dalrymple-Alford JC, Kalders AS, Jones RD, Watson RW. A central executive deficit in patients with Parkinson's disease. J Neurol Neurosurg Psychiatry 1994; 57: 360–7.

Fitts PM. Perceptual-motor skills learning. In: Melton AW, editor. Categories of human learning. New York: Academic Press, 1964: 243–85.

Formisano R, Pratesi L, Modarelli FT, Bonifati V, Meco G. Rehabilitation and Parkinson's disease. Scand J Rehabil Med 1992; 24: 157–60.

Forssberg H, Johnels B, Steg G. Is parkinsonian gait caused by a regression to an immature walking pattern? Adv Neurol 1984; 40: 375–9.

Georgiou N, Iansek R, Bradshaw JL, Phillips JG, Mattingley JB, Bradshaw JA. An evaluation of the role of internal cues in the pathogenesis of parkinsonian hypokinesia. Brain 1993; 116: 1575–87.

Georgiou N, Bradshaw JL, Iansek R, Phillips JG, Mattingley JB, Bradshaw JA. Reduction in external cues and movement sequencing in Parkinson's disease. J Neurol Neurosurg Psychiatry 1994; 57: 368–70.

Grillner S, Stein PSG, Stuart DG, Forssberg H, Herman RM, editors. Neurobiology of vertebrate locomotion. Wenner-Gren Centre International Symposium Series, Vol. 45. Basingstoke: Macmillan, 1986.

Hallett M, Khoshbin S. A physiological mechanism of bradykinesia. Brain 1980; 103: 301–14.

Iansek R, Bradshaw J, Phillips J, Cunnington R, Morris ME. Interaction of the basal ganglia and supplementary motor area in the elaboration of movement. In: Glencross D, Piek J, editors. Motor control and sensorimotor integration. Amsterdam: Elsevier, 1995: 49–60.

Kimura M, Aosaki T, Hu Y, Ishida A, Watanabe K. Activity of primate putamen neurons is selective to the mode of voluntary movement: visually guided, self-initiated or memory-guided. Exp Brain Res 1992; 89: 473–7.

Klawans HL. Individual manifestations of Parkinson's disease after ten or more years of levodopa. Mov Disord 1986; 1: 187–92.

Kokmen E, Naessens JM, Offord KP. A short test of mental status: description and preliminary results. Mayo Clin Proc 1987; 62: 281–9.

Marsden CD. Parkinson's disease [see comments]. [Review]. J Neurol Neurosurg Psychiatry 1994; 57: 672–81. Comment in: J Neurol Neurosurg Psychiatry 1995; 58: 392. Comment in: J Neurol Neurosurg Psychiatry 1995; 58: 521.

Marsden CD, Parkes JD, Quinn N. Fluctuations of disability in Parkinson's disease: clinical aspects. In: Marsden CD, Fahn S, editors. Movement disorders. London: Butterworth, 1982: 96–122.

Martin JP. The basal ganglia and posture. London: Pitman Medical, 1967.

Martin KE, Phillips JG, Bradshaw J, Iansek R. Could bradykensia in Parkinson's simply be compensation? J. Neurol 1994; 241: 439–47.

Morris ME, Iansek R. Characteristics of motor disturbance in Parkinson's disease and strategies for movement rehabilitation. J Hum Mov Sci 1996. In press.

Morris ME, Iansek R, Matyas TA, Summers JJ. The pathogenesis of gait hypokinesia in Parkinson's disease. Brain 1994a; 117: 1169–81.

Morris ME, Iansek R, Matyas TA, Summers JJ. Ability to modulate walking cadence remains intact in Parkinson's disease. J Neurol Neurosurg Psychiatry 1994b; 57: 1532–4.

Morris ME, Iansek R, Summers JJ, Matyas. Motor control considerations for the rehabilitation of gait in Parkinson's disease. In: Glencross D, Piek J, editors. Motor control and sensorimotor integration. Amsterdam: Elsevier, 1996. In press.

Mushiake H, Inase M, Tanji J. Selective coding of motor sequence in the supplementary motor area of the monkey cerebral cortex. Exp Brain Res 1990; 82: 208–10.

Pearson KG, Rossignol S. Fictive motor patterns in chronic spinal cats. J Neurophysiol 1991; 66: 1874–87.

Phillips JG, Martin KE, Bradshaw JL, Iansek R. Could bradykinesia in Parkinson's disease simply be compensation? J Neurol 1994; 241: 439–47.

Quintyn M, Cross E. Factors affecting the ability to initiate movement in Parkinson's disease. Phys Occ Ther Geriatrics 1986; 6: 51–60.

Schenkman M. Physical therapy intervention for the ambulatory patient. In: Turnbull GI, editor. Physical therapy management of Parkinson's disease. New York: Churchill Livingstone, 1992: 137–92.

Schmidt RA. Motor control and learning. Champaign (IL): Human Kinetics, 1983.

Schwab RS, Chafetz ME, Walker S. Control of two simultaneous

voluntary motor acts in normals and in parkinsonism. Arch Neurol Psychiatry 1954; 72: 591–8.

Seitz RJ, Roland PE. Learning of sequential finger movements in man: a combined kinematic and positron emission tomography (PET) study. Eur J Neurosci 1992; 4: 154–65.

Sheridan MR, Flowers KA, Hurrell J. Programming and execution of movement in Parkinson's disease. Brain 1987; 110: 1247–71.

Summers JJ, Burns BD. Timing in human movement sequences. In: Block RA, editor. Cognitive models of psychological time. New York: Erlbaum. 1990: 181–205.

Talland GA, Schwab RS. Performance with multiple sets in Parkinson's disease. Neuropsychologia 1964; 2: 45–53.

Wall JC, Turnbull GI. The kinematics of gait. In: Turnbull GI, editor. Physical therapy management of Parkinson's disease. New York: Churchill Livingstone, 1992: 49–67.

Webster DD. Critical analysis of the disability in Parkinson's disease. Mod Treat 1968; 5: 257–82.

Weissenborn S. The effect of using a two-step verbal cue to a visual target above eye level on the parkinsonian gait: a case study. Physiotherapy 1993; 79: 26–31.

Winter DA. Biomechanics and motor control of human movement. 2nd ed. New York: Wiley, 1990.

Yekutiel MP, Pinhasov A, Shahar G, Sroka H. A clinical trial of the re-education of movement in patients with Parkinson's disease. Clin Rehabil 1991; 5: 207–14.

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