

## SHORT REPORT

## Ability to modulate walking cadence remains intact in Parkinson's disease

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### Abstract

**Gait hypokinesia (slowness) is a characteristic feature of Parkinson's disease. It is not clear, however, whether the slowness is due to a problem in regulation of the timing of consecutive steps or the control of stride size. Examination of cadence control for slow to medium walking speeds has shown an increase in step frequency that was a compensation for reduced stride length. In this investigation the ability of Parkinsonian patients to modulate their cadence (steps per minute) at the fast walking speeds exhibited by age and height matched controls was examined. The findings indicated that cadence control remains unaffected throughout its entire range in Parkinson's disease and that gait hypokinesia is directly attributable to an inability to internally generate sufficiently large steps.**

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Gait hypokinesia (slowness) is one of the primary movement disorders in Parkinson's disease.<sup>1-3</sup> The progressive nature of the gait disturbance in Parkinson's disease can lead to an eventual loss of mobility, an increased incidence of falls, and loss of independence. Consequently both pharmacological and physical treatment have been directed towards the management of this condition so that patients can retain their ability to walk independently. Yet despite the common occurrence of gait hypokinesia and the therapeutic attempts to rehabilitate walking in Parkinson's disease, comparatively little is known of the underlying deficit in this complaint.

Our previous research on the determinants of gait hypokinesia in Parkinson's disease suggested that the fundamental problem is one of producing appropriate stride length.<sup>4</sup> Further, our findings indicated that compensation for the reduced stride size was achieved by increasing cadence to levels well above that of elderly control subjects. The ability to modulate cadence for a number of experimental conditions was retained in Parkinson's disease whereas normal stride length could only be achieved in the presence of external visual

cues.<sup>4</sup> In the previous studies we examined the relation of gait velocity to cadence and stride length for slow to medium walking speeds. Whether parkinsonian patients also retain the ability to appropriately regulate cadence at the fast walking speeds exhibited by elderly control subjects remains unresolved. The purpose of the present investigation was to clarify this issue by analysing the gait patterns of hypokinetic patients with Parkinson's disease walking at the fast speed of control subjects.

### Method

#### SUBJECTS

A total of 30 subjects were recruited for the investigation. There were 15 patients with Parkinson's disease and 15 age, sex, and height matched controls. In both groups there were six men and nine women. The mean age was 72.2 (SD 6.2) years in the Parkinson's disease group and 72.5 (6.46) in the control group. The mean height was 1.62 (0.09) m in the Parkinson's disease group and 1.64 (0.09) m in the control group. The mean body weight was 61.8 (12.9) kg in the Parkinson's disease group and 65.0 (12.0) kg in the control group.

To be included in the study subjects needed to be aged 60-85 years, be able to walk 10 m 10 times, and be able to provide informed consent according to the Helsinki declaration (1964). Parkinsonian patients were only included if they had idiopathic Parkinson's disease as diagnosed by a neurologist. Subjects were excluded if they had visual, musculoskeletal, or cardiovascular problems that affected walking ability. Subjects were also excluded if they had prior neurological disorders or if they were on major tranquillisers.

Parkinsonian patients were tested on average 1.0 (0.25) hour after their midmorning dose of medication, when they were judged to have an effective response and were in the "on phase". The type and dosage of medication were appropriate to the needs of each patient and included levodopa/benserazide hydrochloride; levodopa/carbidopa; bromocriptine, and benzhexol hydrochloride. To determine the level of functional disability patients were scored on the Webster (1968)<sup>5</sup> scale by a neurologist at the start of each

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testing session. They were also scored on the Hoehn and Yahr (1967)<sup>6</sup> scale. The mean Webster score was 13.5 (4) and the mean score on the Hoehn and Yahr scale was 2.7 (0.74).

#### APPARATUS

The walking patterns of patients with Parkinson's disease and control subjects were measured on a 12 m walkway in the Geriatric Research Unit at the Kingston Centre with a commercially available Clinical Stride Analyser (CSA) (B and L Engineering, Santa Fe Springs, CA, USA). The stride analyser enables measurement of the spatial (distance) and temporal (timing) variables of gait and consists of a set of footswitches, a microcomputer storage unit worn as a back pack, a hand held trigger, and a personal computer. The footswitches were worn as inner soles inside the subjects' shoes and included four pressure sensitive switches for detection of contact with the heel, first metatarsal, fifth metatarsal, and first toe. The status of footswitches was sampled every 2 ms with data stored only when a change occurred and remained active for at least 10 ms in one or more switches. An IBM compatible PC and 265 k RAM running DOS version 3.1 (C) was used for data transfer, analysis, and storage. The application software used was PCSA version 1.05 (C) (B and L Engineering, 1993).

Visual cues were used to match the stride length of patients with Parkinson's disease to that of control subjects. These cues were floor markers made of laminated strips of white cardboard that measured 50 mm × 500 mm × 1 mm. Markers were placed on the gait walkway, which was tiled in grey-green linoleum, at a distance equivalent to the mean step length for fast walking for the matched control of each patient with Parkinson's disease.

#### PROCEDURE

Data were first collected for control subjects so that patients with Parkinson's disease could be matched for age, height, and sex, which are known predictors of gait velocity and stride length.<sup>7-10</sup> The procedure for gait analysis was the same for all subjects. Footswitches were fitted inside the shoes and attached to the CSA storage unit, which was attached by a waist belt. A hand held trigger used to signal the start and finish of each gait trial was connected to the storage unit. Subjects were then instructed to perform two

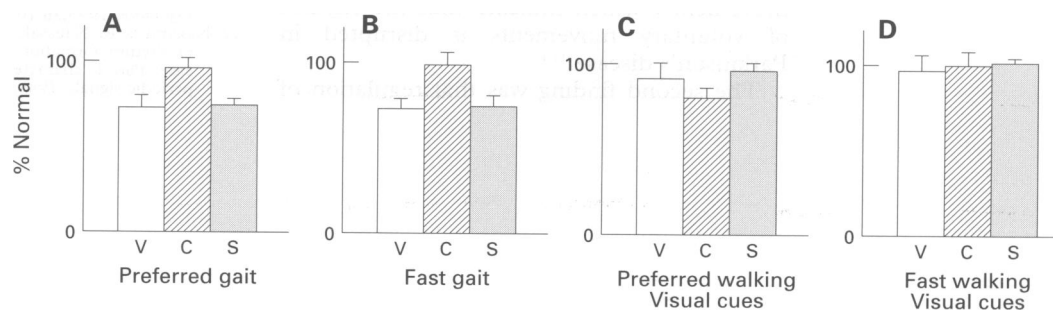
familiarisation trials on the 12 m walkway at their preferred walking pace. For the initial 2 m of each gait trial data were not collected.

One practice trial and two subsequent trials in which data were collected were then performed for each of four conditions. In the first condition subjects were instructed to walk at a comfortable pace to the end of the walkway. In the second condition fast walking was measured. The instruction to subjects was "everybody has a preferred slow, medium, and fast speed of walking. This time walk the full length of the walkway at your fast speed". In the third condition subjects were instructed to "walk over the white floor markers at your preferred speed". In the final condition subjects were instructed to "walk over the white floor markers at your fast speed". For the visual cue conditions the spacing of white floor markers was matched to the stride length of the control subjects walking at their fast speed.

#### Results

The figure shows the mean velocity, cadence, and stride length values for patients with Parkinson's disease expressed as a percentage of normal. Figure (A) presents values for preferred gait and figure (B) for fast walking. Figure (C) shows the results obtained for patients with Parkinson's disease walking at their preferred speed with visual cues placed at the stride length for fast walking found in control subjects. Figure (D) shows the results for patients with Parkinson's disease instructed to walk rapidly over the same visual cues.

The mean gait velocity of patients with Parkinson's disease for preferred walking (50.0 (SD 9.1) m/min.) was considerably slower than controls (71.2 (10.7) m/min,  $p < 0.001$ ; paired  $t$  test). This was not caused by a disturbance in cadence. Rather, the reduced velocity was due to a significantly shorter mean stride length in patients with Parkinson's disease (0.9 (0.1) m) than in controls (1.3 (0.2) m,  $p < 0.001$ ; paired  $t$  test). Moreover, when subjects were instructed to walk at their fast speed the velocity for patients with Parkinson's disease (65.2 (11.3) m/min) was significantly slower than for controls (92.8 (10.4) m/min,  $p < 0.001$ ; paired  $t$  test). Importantly, the slower velocity was due to a smaller mean stride length in parkinsonian patients (1.06 (0.2) m) than control subjects (1.43 (0.1) m,  $p < 0.001$ ; paired  $t$  test). By contrast there was no significant



Mean values and 95% confidence intervals for parkinsonian gait expressed as a percentage of the normal values for age-matched control subjects.

difference in cadence for fast walking (figure (B)).

In the visual cue condition patients with Parkinson's disease were required to walk over white floor markers spaced at the mean stride length for rapid walking in matched controls. Control subjects were required to walk over the markers set at their own mean stride length for fast walking. Figure (C) illustrates that for visual cue walking there was no statistically significant difference between groups for stride length. Gait velocity was slower, however, in patients with Parkinson's disease (74.8 (14.4) m/min) than controls (83.7 (12.5) m/min,  $p < 0.001$ ; paired  $t$  test) and cadence was also less in patients with Parkinson's disease (83.7 (12.5) steps/min) than in controls (108.1 (15.3),  $p < 0.001$ ; paired  $t$  test).

When patients were instructed to walk quickly over the same visual cues the velocity, cadence, and stride length all approximated to normal values with substantial overlap between corresponding confidence intervals (figure (D)). The mean velocity for patients with Parkinson's disease reached 90.7 (13.3) m/min, which was not significantly different from controls (94.5 (14.4) m/min). At this velocity the mean cadence in the Parkinson's disease group (129.9 (12.7) steps/min) was similar to the control group (132.7 (11.0) steps/min). Moreover, the mean stride length in the Parkinson's disease group (1.39 (0.13) m) was similar to that of the control group (1.42 (0.14) m). Figure (D) clearly illustrates that parkinsonian patients could achieve a fast walking speed with high cadence and very large steps when visual cues were coupled with instructions to walk fast.

### Discussion

The results of this study yielded two major findings. Firstly, parkinsonian patients were able to modulate their cadence for the full range of walking speeds. Our earlier experiments<sup>4</sup> showed that patients with Parkinson's disease could regulate their cadence for slow to moderate gait velocities (in the range 34–53 m/min). This study showed that they can also match their cadence to control values for fast speeds of walking (mean value 95 m/min). At this faster speed the mean cadence was 130 steps/min in the Parkinson's disease group and 133 steps/min in the control group. Therefore the control of foot step timing is not the central motor disturbance in gait hypokinesia. This is despite the findings of previous research on finger and hand movements, which indicate that the rhythm of voluntary movements is disrupted in Parkinson's disease.<sup>11 12</sup>

The second finding was that regulation of

stride length is the key deficit in gait hypokinesia. Without external cues the mean stride length was systematically shorter in patients with Parkinson's disease than in controls for both preferred walking and fast walking. The mean stride length for preferred walking was only 0.92 m in patients with Parkinson's disease compared with 1.3 m in controls. Although patients with Parkinson's disease could increase their mean stride length to 1.06 m for fast walking, this was still significantly less than the mean stride length for rapid gait in control subjects (1.43 m). Patients with Parkinson's disease could achieve the same stride as controls, however, when they were presented with visual cues. Furthermore, when instructed to walk quickly over the same cues the patients with Parkinson's disease were able to achieve an average stride length of 1.4 m while achieving cadence and velocity values that were not significantly different from normal fast walking. These results are in agreement with our earlier findings,<sup>4</sup> which suggested that the relative increase in cadence for any given velocity in Parkinson's disease is a compensatory mechanism to adjust for the smaller stride.

To summarise, the present investigation showed that patients with Parkinson's disease have the capacity to walk at fast speeds with very large steps and normal timing, provided that they are presented with appropriate stimuli. In determining optimal gait rehabilitation strategies for Parkinson's disease this functional reserve should be taken into consideration.

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