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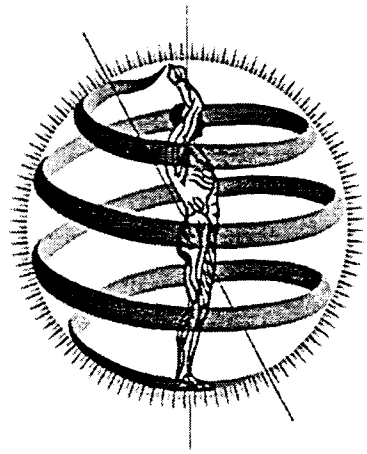

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THE EFFECTS OF PHYSIOTHERAPY STRATEGIES ON THE KINEMATICS AND KINETICS OF GAIT IN PARKINSON'S DISEASE

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The Effects of Physiotherapy Strategies on the Kinematics and Kinetics of Gait in Parkinson's Disease

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PURPOSE

The purpose of this investigation was to measure the effects of visual cues on the kinematics and kinetics of gait in patients with idiopathic Parkinson's disease (PD). Parkinson's disease is a debilitating and progressive condition of the basal ganglia, deep within the brain. Due to a relentless death of neurones in the substantia nigra pars compacta of the brainstem, movements become very slow and scaled down in size.

Our research team has conducted a series of experiments on the effects of visual cues on walking in people with PD (Morris et al., 1994;1996;1999). These studies hypothesized that the ability to generate a normal footstep pattern is not lost in people with PD, rather there appears to be an activation problem. When provided with visual cues spaced on the floor at the appropriate distance for the person's age, normal temporal and spatial parameters of gait could be generated.

What these earlier studies did not explore was whether the normalization of the footstep pattern was accompanied by normalization in the lower limb kinematics (in this case angular displacement of the joints over time) or kinetics (powers and moments).

METHOD

All PD subjects were recruited from the Movement Disorders Program and Volunteer Services Division at Kingston Centre. There were 10 subjects with idiopathic PD and 10 age, height and sex matched control subjects. To be included participants needed to be able to walk 10 metres up to 20 times independently. Subjects were excluded if they had dyskinesia, a past history of neurological conditions or impaired gait.

Three dimensional gait analysis was conducted using a Vicon 140 Motion Analysis system (Oxford Metrics Ltd, Oxford, UK) using four cameras to capture and reconstruct movement trajectories from 13 retroreflective markers. A Kistler Force Plate (Kistler Instruments, Sweden) was used along with the Vicon to collect data on moments and powers at the hip knee and ankle.

All PD subjects were measured in three conditions:

at the "end-of-dose" after 12 hours withdrawal of their levodopa medication;
(2) approximately one hour after their first morning dose of levodopa in the "on" phase of the medication cycle; and
(3) in the "on" phase in the presence of 10 metres of visual cues spaced at the appropriate distance for their age, sex and height.

RESULTS

At the end-of-dose subjects with PD were very hypokinetic. Their mean stride length was only 0.97m and their mean gait speed only 0.9 m/s, even though their cadence was within normal range (118). In addition kinematics at the hip, knee and ankle were markedly abnormal, with reduced hip extension at late stance phase (4.8 degrees flexion rather than 7 degrees extension for controls). There was excessive knee flexion throughout swing and stance, despite a normal basic pattern for the knee trajectory. The total range of ankle movement was only 19 degrees in the PD group compared to 24 degrees in the control group. Ankle kinetics were markedly abnormal, with power generation at A2 limited to approximately 1 Watt, rather than around 2 Watts for control subjects.

With the introduction of levodopa medication, the total range of movement and the spatial and temporal parameters of gait moved towards the normal range. However only with the introduction of visual cues in the "on" phase were normal values achieved for some of the gait variables. In the "on" phase with cues PD stride length reached a mean of 1.44m, cadence was 117 steps/minute and gait speed reached 1.4 m/s. Despite the normalization of the footstep pattern, the kinematics and kinetics of gait did not reach normal values. Maximum hip extension at late stance was only 3.6 degrees flexion, compared to 7 degrees extension in controls. Maximum knee flexion was excessive, at 65 degrees rather than a mean of 61 degrees for

controls. The maximum plantarflexion was only half that of control subjects. Moreover, the A2 power burst at the ankle at push off was underscaled.

DISCUSSION AND CONCLUSION

These results confirmed our earlier studies which showed normalization of the footstep pattern with visual cues provided in the "on" phase of the medication cycle. Despite achieving a normal footstep pattern, gait kinematics and kinetics remained abnormal. Total range of movement across all joints remained reduced, particularly for hip and knee extension in stance. Ankle power was also significantly reduced in PD subjects across all conditions. The results suggest a scaling disorder in PD which is only partially responsive to medication and external cues. Thus visual cues do not simply "activate" a normal gait pattern, rather patients utilize novel kinetic strategies to negotiate cues, which are not necessarily energy efficient. This might be one reason why PD patients revert to a hypokinetic gait pattern a number of hours after visual cue training.

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