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Effect of bilateral subthalamic nucleus stimulation on gait in Parkinson's disease

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Summary

The fundamental disturbance of the parkinsonian gait is the reduction in walking velocity. This is mainly due to reduction in stride length, while cadence (steps/min) is slightly enhanced. Treatment with L-dopa increases stride length while cadence is unchanged. Chronic stimulation of the thalamus has no effect on Parkinsonian gait. The efficacy of electrical stimulation of the subthalamic nucleus (STN) on gait in advanced Parkinson's disease has been clearly demonstrated clinically. The aim of the present study was to quantify the changes in gait measures induced by STN stimulation and L-dopa and to assess possible differential or additive effects. Eight Parkinson's disease patients (mean ± SD age 48.1 ± 7.3 years) with chronic bilateral STN stimulation (mean duration of disease 13.3 ± 2.4 years, mean stimulation time 15.4 ± 10.6 months) and 12 age-matched controls were investigated. Subjects walked on a special treadmill with a closed-loop ultrasound control system that used the subject's position to adjust treadmill speed continuously for the actual walking velocity. In an appropriate crossover design, spatiotemporal gait measures and leg joint angle movements were assessed for at least 120 stride cycles in four treatment conditions: with and without stimulation and with and without a supra-threshold dose of L-dopa. With STN stimulation, there were increases of almost threefold in mean walking velocity (from 0.35 to 0.96 m/s) and stride length (from 0.34 to 0.99 m). Cadence remained constant. The range of motion of the major leg joints also increased. L-Dopa alone had a slightly weaker effect, with an increase in walking velocity to 0.94 m/s and in stride length to 0.92 m at a similar cadence. These increased values were in the range of those for healthy age-matched subjects performing the same task. The combination of both treatments further increased the mean walking velocity to 1.19 m/s and stride length to 1.20 m at an unchanged cadence. However, not all patients receiving STN stimulation improved further when they also received L-dopa. These results demonstrate that chronic bilateral STN stimulation, like treatment with L-dopa, improves walking velocity by increasing stride length without changing cadence. STN stimulation almost exclusively affects mechanisms involved in the control of spatial gait measures rather than rhythmicity. The gait measures obtained with STN stimulation alone are in the range of control subjects.

Keywords: gait disorders; movement disorders; Parkinson's disease; gait analysis; STN stimulation

Abbreviations: GPi = internal globus pallidus; STN = subthalamic nucleus; UPDRS = Unified Parkinson's Disease Rating Scale

Introduction

A typical symptom of Parkinson's disease is a short-stepped, shuffling gait with slow walking velocity and decreased amplitude of the segmental movements. The problem in this gait disturbance is the regulation of stride length (Morris *et al.*, 1994*a*, *b*). Healthy subjects achieve an increase in walking velocity by increasing both stride length and cadence to a similar extent. Cadence regulation is not disturbed in Parkinson's disease. The slope of the stride length–cadence relationship is similar in healthy subjects and Parkinson's disease patients, whereas the intercept for stride length is reduced (Morris *et al.*, 1998). This means that Parkinson's disease patients are able to increase their stride length by amounts similar to those achieved by healthy subjects for any given increase in cadence, but that the stride length is preset at a lower level for the whole stride length–cadence relationship in Parkinson's disease. Furthermore, the usable