Gait & Posture 38 (2013) 956–961

Contents lists available at SciVerse ScienceDirect

Gait & Posture

journal homepage: www.elsevier.com/locate/gaitpost

Gait initiation impairments in both Essential Tremor and Parkinson's disease

Kristina M. Fernandez [a,b], Ryan T. Roemmich [a,b], Elizabeth L. Stegemöller [a,b,c], Shinichi Amano [a,b], Amanda Thompson [b,c], Michael S. Okun [b,c], Chris J. Hass [a,b,*]

[a] Department of Applied Physiology and Kinesiology, University of Florida, Gainesville, FL, United States
[b] Center for Movement Disorders and Neurorestoration, University of Florida, Gainesville, FL, United States
[c] Department of Neurology, University of Florida, Gainesville, FL, United States

ARTICLE INFO

Article history:
Received 24 November 2012
Received in revised form 25 April 2013
Accepted 1 May 2013

Keywords:
Gait initiation
Parkinson's disease
Essential Tremor
Anticipatory postural adjustments
Motor control

ABSTRACT

Gait initiation is a transitional task involving a voluntary shift from a static, stable position to a relatively less-stable state of locomotion. During gait initiation, anticipatory postural adjustments precede stepping in order to generate forward momentum while balance is maintained. While deficits in gait initiation are frequently reported for persons with Parkinson's disease, there is a paucity of information regarding gait initiation performance in persons with Essential Tremor. We investigated anticipatory postural adjustments and spatiotemporal characteristics of gait initiation in persons with Essential Tremor and compared them to persons with Parkinson's disease as well as age-matched neurologically healthy adults. Twenty-four persons with Essential Tremor, 31 persons with Parkinson's disease, and 38 age-matched controls participated. We compared anterior–posterior and mediolateral center of pressure movements and spatiotemporal stepping characteristics during gait initiation among the three groups using Mann–Whitney *U*-tests with Bonferroni corrections for multiple comparisons and one-way ANOVAs. Persons with Parkinson's disease demonstrated significantly reduced displacement and velocity of the center of pressure during early phases of anticipatory postural adjustments relative to controls. Displacement of the center of pressure was also reduced in persons with Essential Tremor, although at a later stage of the gait initiation process. Persons with Parkinson's disease and Essential Tremor demonstrated similar reductions in step length during gait initiation when compared to controls. Persons with Parkinson's disease and Essential Tremor exhibit different deficits in gait initiation when compared to healthy older adults. Therefore, this study provides further evidence differentiating motor control features in these movement disorders.

1. Introduction

Gait initiation (GI) is a complex transitional locomotor task which requires a shift from a static, stable state to a relatively less stable, dynamic state of motion. GI is a challenging task that demands balance and postural control due to a decreasing base of support from a two leg stance to an alternating single leg stance. This is a very destabilizing period of the locomotor process, as the center of pressure (COP) separates from the center of mass (COM) and highly specific postural shifts occur to allow the body to begin forward motion [1]. Anticipatory postural adjustments (APAs) precede stepping during GI as a process necessary to generate momentum for efficient forward motion while the body is balanced between the two feet. Indeed, failure to generate sufficient forward momentum during GI has been shown to lead to overall poorer GI performance, as evidenced by decreased step length and decreased step velocity [2].

During quiet stance, movements of the COP and COM are relatively coupled. However, as gait is initiated, APAs function to shift the COP postero-laterally toward the stepping limb while the COM moves anteriorly and toward the stance limb. Because GI requires dynamic postural control to separate the COP from the COM, the outputs of the APAs (COP and COM movements) have been used as investigative tools to evaluate dynamic postural instability [3]. Indeed, previous research on APAs has suggested that COP excursions during GI are diminished in pathological populations at increased risk of falling [2,4–6].

Parkinson's disease (PD) is characterized by motor signs resulting from a degenerative loss of dopaminergic neurons in the Substantia nigra as well as multiple motor and non-motor regions of basal ganglia [2]. The basal ganglia are understood to be

* Corresponding author at: 122 Florida Gym, P.O. Box 118205, Gainesville, FL 32611-8205, United States. Tel.: +1 352 294 1716.
E-mail address: cjhass@hhp.ufl.edu (C.J. Hass).

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http://dx.doi.org/10.1016/j.gaitpost.2013.05.001

essential in planning and initiating movement, and consequently the APAs have been shown to be diminished in persons with PD when initiating gait [2,4,5]. Further, previous research has demonstrated that persons with PD exhibit deficits in postural control and momentum generation during GI as evidenced by reduction of APA magnitude and restriction of the COP/COM separation when compared to their neurologically healthy peers [3,4].

Essential Tremor (ET) is a neurodegenerative movement disorder which is characterized by an involuntary shaking predominantly in the hands, forearms, and head/neck. The typical tremor is postural/action, but can be present at rest. While static postural control appears to remain relatively intact [7], recent research has begun to describe a variety of locomotor deficits in ET. Earhart and colleagues recently reported decreases in cadence and walking speed in persons with severe ET, which were accompanied by impairment in dynamic stability as evidenced by reduction in double support time when compared to controls [8]. Moreover, persons with relatively advanced ET also demonstrated cerebellar-like deficits in dynamic stability during tandem walking tasks [9]. Locomotor deficits in this population have also been described more generally in a clinical setting, as a group of persons with ET composed of persons with varying degrees of severity demonstrated reduced performance on clinical measures of functional mobility whereas static balance control was unaffected [10]. Despite the breadth of information regarding the effects of ET on gait, mobility, and static postural control, little is known about the effects of ET on dynamic postural control during transitional locomotor periods such as GI. These periods are vitally important phases of locomotion, as it is during transitional periods when older adults are most susceptible to falling [11].

Therefore, the purpose of this study was to investigate APAs and spatiotemporal characteristics of GI in persons with ET. We compared these features of GI among persons with ET, PD, and neurologically healthy older adults. As dynamic stability and gait deficits seem to be evident in persons with relatively severe ET and somewhat similar to those seen in persons with mild-to-moderate PD, we hypothesize that persons with ET will demonstrate GI deficits which are similar to but potentially less severe than those seen in persons with PD, as severe GI deficits are not commonly reported in clinical settings in ET (as is often the case in PD). ET and PD are often quite similar phenotypically, as ET is one of the most common movement disorders in the adult population, and yet approximately 30–50% of persons with ET are misdiagnosed with PD or other tremor disorders [12]. Thus, in this study, we aimed to further the understanding of differential motor control deficits between PD and ET.

2. Methods

Twenty-four participants with Essential Tremor (mean age ± SD: 68 ± 6 years, mean height: 171 ± 9 cm, mean body mass ± SD: 93 ± 21 kg, mean Fahn-Tolosa-Marin Tremor Rating Scale (TRS) [13] Motor (subscores 1–14) score ± SD: 35 ± 12, mean TRS activities of daily living (subscores 15–21) score ± SD: 14 ± 5, mean TRS Total score ± SD: 49 ± 16) were referred from the Center for Movement Disorders and Neurorestoration at the University of Florida. ET participants were evaluated and diagnosed with ET by a movement disorder neurologist and were optimally treated prior to testing. Thirty-one participants with idiopathic PD (mean age ± SD: 68 ± 7 years, mean height ± SD: 170 ± 6 cm, mean body mass ± SD: 82 ± 15 kg, mean unified Parkinson's disease rating scale (UPDRS)[14] Motor score ± SD: 25 ± 7) were also recruited from the clinic and from the university community. PD participants were evaluated and diagnosis of PD was confirmed by a movement disorder neurologist using the UK Brain Bank criteria [15]. All PD and ET participants were tested at their self-reported best medicated state. Eight of the ET participants were taking either a beta-adrenergic antagonist, an anticonvulsant, or both while the remaining 16 ET participants were not taking any medication specifically intended to reduce tremor. All participants with PD were being treated with levodopa and, in some cases, a dopamine agonist. Thirty-eight healthy older adults (HOA; mean age ± SD: 68 ± 5 years, mean height ± SD: 167 ± 12 cm, mean body mass ± SD: 79 ± 14 kg) volunteered and were screened for neurological and musculoskeletal impairment. The HOA were enrolled from the university and the neighboring community. Before participation in the study, all subjects signed a written informed consent that was approved by the University's Institutional Review Board.

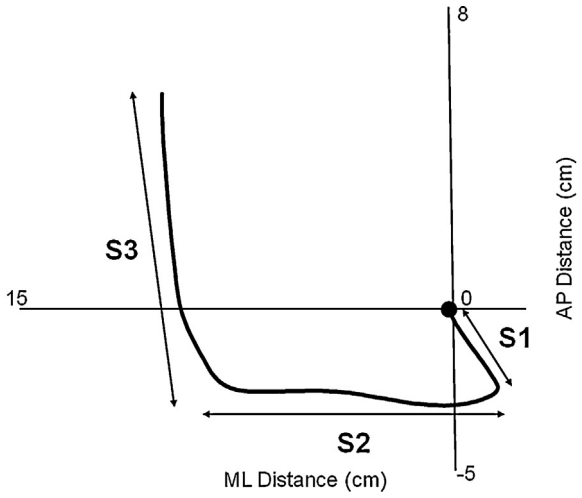

Fig. 1. A representative center of pressure movement pattern indicating the S1, S2, and S3 phases during gait initiation in a healthy older adult.

Participants began each experimental trial standing barefoot with both feet on one force-plate (Bertec Corporation, Columbus, OH). Upon a verbal instruction of "ready", the participants were asked to pause for several moments before volitionally initiating gait along a 12-m walkway at their own comfortable pace. Participants were given preference in the placement and position of their feet on the force-plate, which were then restricted to that particular position for the remainder of the trials. Participants performed three GI trials with the same self-selected leg. The walkway was surrounded by an 8-camera optical motion capture system (120 Hz; Vicon Nexus, Lake Forrest, CA). Passive, retroreflective markers were placed bilaterally over the second metatarsal head, lateral malleolus, and calcaneus. Gait events (heel-offs, heel-strikes, and toe-offs) were manually labeled in Vicon Nexus based on individual marker trajectories.

We measured COP displacements and velocities during GI by assessing the pattern of COP movement using custom-written MATLAB software (MathWorks, Natick, MA). We manually distinguished two events during GI that separate the COP trace into three distinct phases as described previously [16] (Fig. 1). Briefly, the S1 phase begins with the onset of the lateral, posterior shift of the COP and ends with the COP positioned at the most lateral and posterior position relative to the initial stepping limb (swing limb – SW limb). The beginning of the S2 phase is defined as the onset of a lateral shift of the COP in the opposite direction toward the contralateral limb (stance limb – ST limb), and ends when the COP is in its most lateral and posterior position under the ST limb. The S3 phase is defined as the forward translation of the COP trace under the ST limb until the instance prior to heel strike of SW limb. Displacement and velocity of the COP was calculated in the anterior–posterior (AP) and mediolateral (ML) directions during the S1, S2, and S3 phases of GI.

We also measured spatiotemporal gait parameters during GI including: (a) the length of the first SW limb step, which was defined as the displacement of the heel marker from static position to first heel-strike; (b) time of the first SW limb step, defined as the time between the first heel-off and first heel-strike of the SW limb; and (c) the velocity of the first SW limb, which was measured as the SW step length divided by SW step time. Step length, step time, and step velocity were also measured for the first ST limb step using similar methods. Thus, we ultimately calculated the following outcome measures: SW and ST step length, SW and ST step time, and SW and ST step velocity.

3. Analysis

As the distribution of all COP variables violated assumptions of normality, mean differences for all COP variables were compared among the HOA, ET, and PD groups using Mann–Whitney *U*-tests with Bonferroni corrections for multiple comparisons. One-way ANOVAs were conducted to compare age, mass, height, initial stance width, and mean spatiotemporal variables among groups. Level of significance was set at $\alpha = 0.05$ for both the Mann–Whitney *U*-tests (prior to Bonferroni correction) and the one-way ANOVAs.