INTRODUCTION

Gait initiation (GI) involves a shift from a static, stable state to a destabilizing, dynamic state of locomotion. It 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. GI is a complex transition phase consisting of anticipatory postural adjustments (APAs) that provide the momentum needed for efficient forward motion. 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. Because GI requires a separation of the center of mass (COM) from the center of pressure (COP), it has been used as an investigative tool to observe balance dysfunction and postural instability [1].

Parkinson’s disease (PD) is characterized by a degenerative loss of dopaminergic neurons in the substantia nigra of the basal ganglia. The main symptoms of PD are tremor, rigidity, and bradykinesia, and it is common for PD patients to exhibit stooped posture, shuffling, and freezing of gait. The basal ganglia are commonly understood as essential in planning and initiating movement, and thus APAs have been shown to be diminished in persons with PD leading to shorter, slower steps during GI. Furthermore, it has been shown that persons with PD have shorter COP-COM distances compared to healthy older adults, indicating a decline in postural control and momentum generation.

Essential Tremor (ET) is a neurodegenerative movement disorder that is characterized by an involuntary shaking predominantly in the hands, arms, neck, and head that worsens with movement. Recent research indicates that ET is caused by brainstem and cerebellar dysfunction. In addition to the basal ganglia, both the brainstem and the cerebellum also play an important role in GI. Although ET is the most prevalent movement disorder, little is known about how ET deficits affect dynamic control and postural stability, particularly during GI.

Because there are no quantifiable diagnostic measures for PD or ET, PD and ET are discernible only by clinical criteria. Since these two neurological disorders share common characteristics of tremor and gait disturbances, there is a greater possibility for misdiagnoses, as approximately 30-50 % of persons with ET are misdiagnosed with PD or other tremor disorders [2]. Due to differences in neuropathology between PD and ET, we expect that GI in persons with ET will resemble healthy older adults more closely than persons with PD. This study aims to utilize GI as a quantifiable measure to aid in the clinical diagnostic process of PD and ET.

METHODS

Ninety-three participants, 23 persons with ET (67.5 ± 6.0 y, 172.4 ± 8.4 cm, 99.0 ± 19.8 kg), 32 with PD (67.5 ± 7.4 y, 170.3 ± 6.1 cm, 82.6 ± 14.4 kg), and 38 healthy older adults (68.1 ± 5.8 y, 169.1 ± 5.8 cm, 79.3 ± 16.1 kg) participated. All PD participants were tested at their self-reported best medicated state. None of the PD subjects displayed signs of dyskinesia or non-purposeful movements. Similarly, persons with ET were optimally treated during testing.

GI trials began with the participant standing quietly on force platform (Bertec, Columbus Ohio)
mounted flush with the laboratory floor. Initial positioning of the feet was self-selected. In response to an auditory signal, the participants initiated walking and continued to walk for several steps. Each participant performed three to five trials.

Kinematic data were collected using an 3D Optical Capture system (Vicon Peak, Oxford, UK) collecting at 120Hz. Thirty-five passive reflective markers were attached to the body in accordance with the Vicon Plug-in-Gait marker system. We manually distinguished two events during GI that separated the COP trace into three distinct phases as defined by previous research on APAs [3].

The following outcome measures were collected for the three phases of gait initiation: (1) S1 anteroposterior (AP) displacement, (2) S1 mediolateral (ML) displacement, (3) S2 ML displacement, (4) S3 AP displacement, (5) S1 AP velocity, (6) S1 ML velocity, (7) S3 AP velocity, and (8) S2 ML velocity.

We analyzed mean differences among the HOA, ET, and PD groups using Mann-Whitney U-tests with Bonferroni corrections for multiple comparisons for all COP variables. Level of significance was set at .05 for the Mann-Whitney U-tests (prior to Bonferroni correction).

**RESULTS AND DISCUSSION**

Persons with PD had a significantly diminished S1 AP displacement (17.8 ± 9.5 vs. 27.2 ± 19.3 mm, p=.003), S1 ML displacement (17.9 ± 11.1 vs. 24.4 ± 15.6 mm, p=.017), S1 AP velocity (52.0 ± 26.9 vs. 101.6 ± 62.4 mm/s, p<.001) and S1 ML velocity (52.8 ± 26.3 vs. 88.3 ± 55.4 mm/s, p=.002) in comparison to healthy older controls. Persons with ET had a significantly diminished S3 AP displacement (70.9 ± 27.6 vs. 87.7 ± 42.4 mm, p=.013) in comparison to healthy older controls. When comparing persons with ET to those with PD, persons with PD had significantly decreased S1 AP (52.0 ± 26.9 vs. 94.9 ± 71.7 mm/s, p=.004) and S1 ML (52.8 ± 26.3 vs. 86.1 ± 54.6 mm/s. p=.005) velocities than persons with ET.

Persons with PD exhibit disturbances during GI, and advanced cases display freezing of gait. This disturbance in GI is due to dopaminergic dysfunction of the basal ganglia, and potentially dysfunction of the pedunculopontine nucleus. However, our results suggest that persons with ET also exhibit GI disturbances despite experiencing lesser dopaminergic deficits. Furthermore, previous research has shown that the brainstem and cerebellum are affected in ET. As the cerebellum and basal ganglia both send descending signals to the brainstem to regulate locomotion, we postulate that dysfunction of brainstem structures may be the cause of GI deficits in persons with ET. Thus, GI impairment may be more severe in PD due to the compounding nature of basal ganglia and brainstem deficits whereas GI impairments in ET likely are localized to brainstem control.

**REFERENCES**


