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The stroke-related effects of hip flexion fatigue on over ground walking

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ABSTRACT

Individuals post stroke often rely more on hip flexors for limb advancement during walking due to distal weakness but the effects of muscle fatigue in this group is not known. The purpose of this study was to quantify how stroke affects the influence of hip flexor fatigue on over ground walking kinematics and performance and muscle activation. Ten individuals with chronic stroke and 10 without stroke (controls) participated in the study. Maximal walking speed, walking distance, muscle electromyograms (EMG), and lower extremity joint kinematics were compared before and after dynamic, submaximal fatiguing contractions of the hip flexors (30% maximal load) performed until failure of the task. Task duration and decline in hip flexion maximal voluntary contraction (MVC) and power were used to assess fatigue. The stroke and control groups had similar task durations and percent reductions in MVC force following fatiguing contractions. Compared with controls, individuals with stroke had larger percent reductions in maximal walking speed, greater decrements in hip range of motion and peak velocity during swing, greater decrements in ankle velocity and lack of modulation of hip flexor EMG following fatiguing dynamic hip flexion contractions. For a given level of fatigue, the impact on walking function was more profound in individuals with stroke than neurologically intact individuals, and a decreased ability to up regulate hip flexor muscle activity may contribute. These data highlight the importance of monitoring the effect of hip flexor muscle activity during exercise or performance of activities of daily living on walking function post stroke.

1. Introduction

Many stroke survivors have leg weakness that limits walking capability [1]. Several studies show an association between baseline weakness of the paretic leg muscles, such as the hip flexors, with abnormalities in the kinematics and kinetics of walking [2–4]. However, less is known about how changes in paretic neuromuscular fatigability (the acute, exercise induced reductions in force [5]) impacts gait function. Accordingly, individuals with stroke demonstrate altered kinematics after short bouts of walking [6–8] and decreased distance walked over 6-min compared with healthy controls [9]. Furthermore, people post-stroke exhibit greater fatigability and lesser time to task failure (task duration) of leg muscle than healthy controls [10]. Thus, because individuals with stroke are weaker at baseline and this weakness limits walking function [3,11], stroke-related changes in fatigability could further impair walking deficits.

Neuromuscular fatigability post stroke and the effect on walking function is not well characterized [12–14]. Few studies have examined paretic fatigability during repetitive volitional activation of the musculature at sub-maximal force which is functionally relevant to many daily tasks. Recently, we showed that individuals with stroke had decreased task duration for a sustained, sub-maximal contraction of the paretic hip flexors compared with the non-paretic leg when target torques were matched [10]. Declines in torque were negatively associated with self-selected walking speed. Although this work established a potential relationship between hip flexor fatigability and walking function post stroke, there are no studies to date that quantify the effect of paretic hip flexor fatigue on the spatiotemporal and kinematics of over ground walking. This information would be clinically useful in order to optimize strength protocols to offset fatigue-related effects on walking.

Fatigue of the paretic hip flexors, in particular, is likely to significantly impact walking function given their biomechanical importance during walking [2,11,15–17]. In healthy individuals, propulsive forces at the ankle are primarily responsible for limb advancement. In contrast, after stroke, the ankle musculature frequently remains paralyzed, decreasing ankle propulsion. In non-fatiguing walking conditions, the hip musculature compensates for force deficits at the ankle [17] and hip power is a predictor of

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Table 1
Characteristics of participants with stroke.

Subject	Age, year	Sex	Hemisphere affected	Time since stroke, months	Fugl Meyer (lower extremity) max = 34	Self-selected walking speed, m/s
S1	64	M	Left	66	27	0.78
S2	54	M	Right	55	24	0.8
S3	48	M	Right	49	28	0.78
S4[a,b]	65	F	Left	79	25	0.22
S5[a,b]	60	F	Right	82	23	0.29
S6	64	F	Right	109	32	1.04
S7[a]	62	M	Right	90	21	0.42
S8[a]	79	F	Right	95	21	0.14
S9	59	M	Left	119	29	0.9
S10	61	F	Left	260	32	0.68
Mean	61.6			100.4	26.2	0.61
SD	8.0			60.3	4.1	0.31

[a] Used an AFO during ambulation.
[b] Used a straight cane.

maximal walking speed [3] an index of walking impairment [3,11]. Use of a hip flexion assist orthosis results in increased walking speed and distance in chronic stroke [18]. Increased dependence on the hip musculature is demonstrated in other disorders with distal weakness [19]. Given the task specificity of neuromuscular fatigability [5] and the increased fatigability of paretic musculature (REF), dynamic fatiguing contractions of the paretic hip flexors may impair power related aspects of walking.

The primary objective of this study was to quantify the effects of hip flexor fatigue on spatiotemporal and kinematics of over ground walking post stroke. We hypothesized that paretic hip flexor fatigue would result in larger decreases in (1) maximal walking speed and distance walked, and (2) joint velocities and range of motion compared with healthy controls. We also quantified muscle activation of several lower limb muscles with electromyogram (EMG) to provide insight into possible mechanisms.

2. Methods

2.1. Subjects

All activities in this study were approved by Marquette University's Institutional Review Board. All participants gave informed consent before participating in study activities. Ten neurologically intact subjects (5 men and 5 women; 62.4 ± 7.9 years) and 10 age-matched participants with chronic (>6 months) stroke (Table 1) participated in this study. Stroke subject inclusion criteria included: single, unilateral stroke (based on information obtained through verbal communication from the physician and consistent with neurological physical examination results), able to ambulate (with or without the use of an assistive device) at least 30 ft, and >6 months post stroke. Stroke subject exclusion criteria included: history of multiple strokes, brainstem stroke, any uncontrolled medical condition, contractures of any lower extremity joints, and the inability to follow 2–3 step commands.

2.2. Force and power measurements

Participants sat at the edge of a height-adjusted plinth with their test leg's hip and knee angles in 30° flexion (Fig. 1). The target range of motion to 80° of hip flexion was indicated by a laser pointer. A thigh strap was positioned above the knee at 20% femur length relative to the lateral epicondyle and greater trochanter (Fig. 1). A cable (9.5 mm diameter) ran between this strap and a custom-built weight stack, which enabled the testing of both dynamic and isometric contractions. Cable tension force was

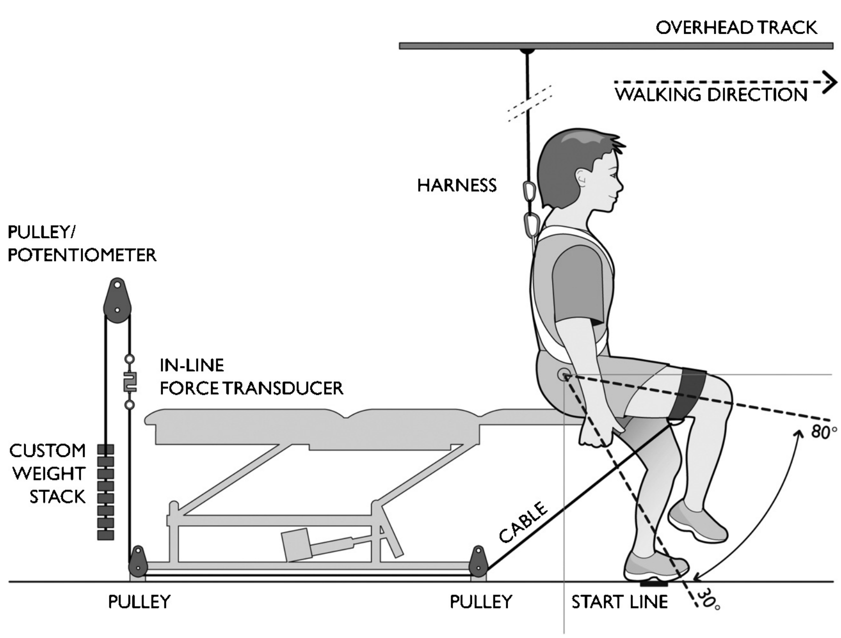

Fig. 1. Schematic of the experimental set-up.