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RELATIONSHIPS BETWEEN MUSCLE CONTRIBUTIONS TO WALKING SUBTASKS AND FUNCTIONAL WALKING STATUS IN PERSONS WITH POST-STROKE HEMIPARESIS

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INTRODUCTION

Walking speed is commonly used to predict stroke severity and assess functional walking status (i.e., household, limited community and community walking status) post-stroke [1]. The underlying mechanisms that limit walking speed (and functional walking status by extension) need to be understood to improve post-stroke rehabilitation. Previous experimental studies have shown correlations between paretic plantarflexor output during the pre-swing phase and walking speed and suggest that the paretic hip flexors can compensate in some hemiparetic subjects [e.g., 2]. Modeling and simulation studies of healthy walking have shown that the ankle plantarflexors, soleus (SOL) and gastrocnemius (GAS), and uniarticular hip flexors (IL) are essential contributors to the walking subtasks of forward propulsion, swing initiation and/or power generation during pre-swing [3,4]. However, the relationships between functional walking status and individual muscle contributions to these walking subtasks in hemiparetic walking are unknown. The goal of this study was to use 3D forward dynamics simulations to investigate the relationships between functional walking status in post-stroke hemiparetic walking and muscle contributions to forward propulsion, swing initiation and power generation.

METHODS

A previously developed 2D modeling and simulation framework [3] was modified to generate 3D forward dynamics walking simulations (from midstance to toe-off) that emulated the experimental kinematics and ground reaction forces (GRFs) of two representative hemiparetic subjects walking at

their self-selected speed (limited community walker, 0.45 m/s; community walker, 0.90 m/s) and an age-matched healthy control subject walking at 0.6 and 1.0 m/s. Subjects walked on a split-belt instrumented treadmill (Tecmachine) for 30 seconds while kinematic, GRF and EMG data were collected. The EMG data were used to constrain the timing for each muscle excitation pattern in the optimization to ensure muscles were producing force in the appropriate phase of the gait cycle in the simulation. Excitation patterns for each muscle were defined using a bimodal Henning pattern and the patterns (timing and amplitude) and initial joint angular velocities were optimized using a simulated annealing algorithm [5] that minimized the differences between the simulated and experimental data. Muscle-induced acceleration and segment power analyses [3] were performed to quantify individual muscle contributions to forward propulsion (average horizontal pelvis acceleration), swing initiation (average power delivered to the leg) and power generation (average musculotendon power) during the pre-swing phase.

RESULTS AND DISCUSSION

Similar to the healthy control subject (Fig. 1A), the ankle plantar flexors (SOL, GAS) generated the majority of propulsion in the paretic leg in the community walker (Fig. 1B). However, in the limited community walker, the paretic leg muscles contributed little to forward propulsion and the non-paretic leg muscles (rectus femoris, RF and vastii, VAS) compensated for the reduced paretic leg output (Fig. 1B). This result is consistent with Bowden et al. [6] who showed that the non-paretic leg's contribution to the A/P GRF was increased in hemiparetic subjects who were more impaired. The

non-paretic leg muscles (primarily hamstrings, HAM) in the limited community walker increased their contributions to pelvis deceleration and the net effect from both legs (sum of Totals, Fig. 2B) was to decelerate the pelvis during pre-swing.

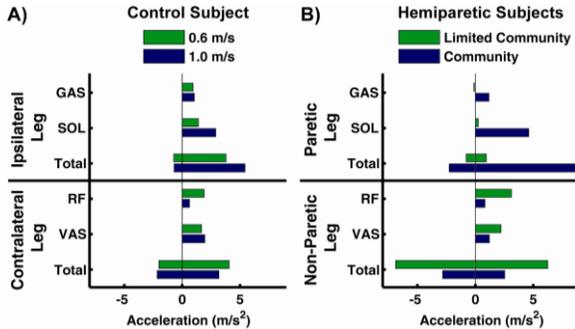


Figure 1: Primary muscle contributions to forward propulsion by A) the healthy control subject during ipsilateral pre-swing, and B) the hemiparetic subjects during paretic pre-swing, where *Total* is the positive and negative sums from all muscles for the respective leg.

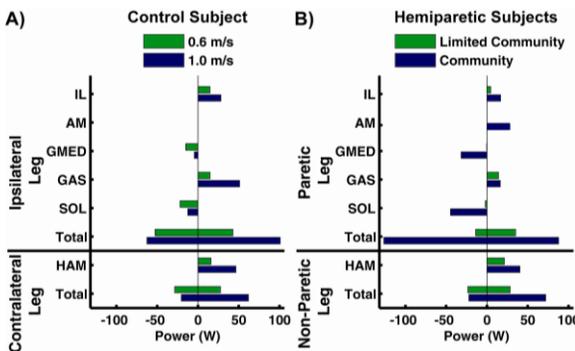


Figure 2: Primary muscle contributions to swing initiation by A) the healthy control subject during ipsilateral pre-swing, and B) the hemiparetic subjects during paretic pre-swing, where *Total* is the positive and negative sums from all muscles for the respective leg.

The community walker’s muscle contributions to swing initiation were similar to those seen in the healthy control subject (Fig. 2A) with paretic GAS, IL and non-paretic HAM contributing to paretic leg swing initiation (Fig. 2B). Paretic adductor magnus (AM) and gluteus medius (GMED) also contributed positively and negatively, respectively, to swing initiation in the community walker (Fig. 2B). Clear deficits existed in the paretic and non-paretic leg muscle contributions to swing initiation in the limited community walker (Fig. 2B). This is

consistent with previous studies showing reduced paretic leg kinetic energy at toe-off, which suggests impaired paretic leg swing initiation [e.g., 7]. The negative contributions from the paretic leg muscles (GMED and SOL) were also greatly reduced (Fig. 2B), allowing the leg to accelerate into swing.

Power generation by muscles in the community walker closely resembled those of the control subject. However, in the limited community walker, the paretic leg muscles, specifically GAS and IL, generated less power consistent with their reduced contributions to forward propulsion (GAS) and swing initiation (GAS and IL). Paretic SOL absorbed power in the limited community walker, reducing its contribution to forward propulsion.

CONCLUSIONS

The analyses showed that deficits in forward propulsion, swing initiation and power generation are related to functional walking status in hemiparetic walking. Increased contributions from the paretic leg muscles (i.e., plantarflexors and hip flexors) and reduced contributions from the non-paretic leg muscles (i.e., knee and hip extensors) to the walking subtasks were critical in achieving a higher functional walking status. Interventions targeting these muscle groups may improve rehabilitation outcomes and the functional walking status of persons with post-stroke hemiparesis.

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