Potential Muscle Function During the Swing Phase of Stroke Gait

Debra G. George-Reichley and Jill S. Higginson

The understanding of individual muscle impairments that affect swing phase in stroke gait will lead to better rehabilitation strategies for this population. We used induced acceleration analysis to evaluate the potential each muscle has to accelerate the hip and knee joints of the swing limb, using kinematics from three stroke subjects and five healthy subjects. To determine the influence of altered limb position on muscle function, we augmented hip extension by 10° in swing phase for all subjects. We found that in early swing, healthy subjects had greater potential to accelerate the knee into flexion than stroke subjects, whereas stroke subjects had greater potential to accelerate the hip into flexion. Perturbing the hip flexion angle into greater extension increased the potential of biarticular muscles to flex the knee in swing phase. The potential of muscles to improve swing phase dynamics depends on the initial posture of the limb and highlights the importance of subject-specific evaluations in the design of appropriate therapeutic interventions.

Keywords: kinematics, motion analysis, muscle, musculoskeletal, rehabilitation, stroke

Disability from stroke affects millions of individuals in the United States, with over 700,000 new cases annually (NINDS, 2007). Because regaining the ability to walk is integral to achieving independence after stroke, it is not surprising that improved walking function is the goal most frequently asserted by stroke survivors (Bohannon et al., 1988). A multitude of studies have attempted to establish new and better ways to restore as much gait function to stroke survivors as possible, employing surgery, treadmill training, functional electrical stimulation and strength training (Waters et al., 1979; Kwakkel & Wagenaar, 2002; Pohl et al., 2002; Sullivan et al., 2002; Burridge & McLellan, 2000). To design the most appropriate and effective interventions, a thorough understanding of individual muscle function during gait must be established.

Common obstacles to restoring gait function after stroke are spasticity and muscle weakness in the paretic leg. Spasticity, or velocity-dependent increase in muscle tone, often affects the quadriceps and plantar flexor muscles (Gelber, 2002). During swing phase, these muscles can generate excessive knee moments and thus prevent reaching adequate peak knee flexion, causing a condition identified as stiff-knee gait (Goldberg et al., 2004; Kerrigan et al., 1991; Piazza & Delp, 1996). Muscle weakness is often evident at more distal locations such as the ankle, where the inability to dorsiflex the foot during swing causes footdrop and impedes clearance of the swing limb (Adams et al., 1990; Jonkers et al., 2003).

Hemiparetic gait is characterized by slow gait speed, and asymmetric kinematics as well as inter- and intra-subject variability (Olney & Richards, 1996). Consistent with slow speed and decreased step length, hip excursions are reduced (Lelas et al., 2003). Treadmill training at fast speeds has been advocated as an effective therapy to improve function in a subset of stroke survivors (Lamontagne & Fung, 2004). However, it is unclear whether increasing hip extension would enhance the potential of paretic muscles to achieve more knee flexion in swing.

During gait, limb positions dictate muscle moment arms and the effect individual muscles can have on the body. Computer simulations of gait are useful tools in studying individual muscle functions. Induced acceleration analysis has been developed to identify and quantify the contribution of individual muscles to joint and segment accelerations (Fregly & Zajac, 1996; Neptune et al., 2001; Anderson & Pandy, 2001; Kimmel & Schwartz, 2006). Using this technique with subject-specific walking kinematics, Kimmel and Schwartz (2006) have identified a set of muscles that contribute to hip and knee flexion and extension and which largely agrees with clinical data (Perry, 1992). In the stroke population especially, where limb positions and muscle strength often differ significantly from that of their healthy counterparts, this tool can be customized for individual subjects to identify the unexpected roles sometimes played by muscles (Higginson et al., 2006). A few studies have examined the potential roles of muscles in normal gait to investigate abnormalities such as stiff-knee gait (Kimmel & Schwartz, 2006; Riley & Kerrigan, 1998; Arnold et al., 2005; Goldberg et al., 2004). In calculating muscle potential, the analysis ignores the actual strength of each

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muscle, thus allowing investigators insight into the possibilities that could likely become available if specific muscles could be strengthened and used.

The objective of this study was to determine which muscles should be targeted to improve knee flexion in patients with stroke. We compared the potential of lower extremity muscles to accelerate the hip and knee during swing phase using healthy and stroke gait kinematics. We first hypothesized that the potential of stroke subjects’ muscles to accelerate the hip and knee into flexion in early swing would be diminished compared with healthy controls. We further hypothesized that the potential of stroke subjects’ muscles to accelerate the hip and knee into flexion in early swing would increase when the hip was perturbed 10° into extension. In elucidating muscle function during swing, we hope to identify which subjects may benefit from strengthening or speed training.

Methods

Five healthy subjects (25 ± 7.4 years) and three subjects with a history of stroke (55 ± 9.8 years) signed informed consent forms approved by the Human Subjects Review Board (Table 1). For inclusion, stroke survivors were more than 6 months following cortical stroke and able to walk for 5 min at self-selected speed that was determined using the 6-m walk test. Subjects also received a clinical evaluation involving the Lower Extremity portion of the Fugl-Meyer scale (Fugl-Meyer et al., 1975). Twenty-three reflective markers (Helen Hayes set) were used to track kinematics, which were collected using a six-camera motion capture system (Motion Analysis, Santa Rosa, CA) sampled at 60 Hz, then low pass filtered at 6 Hz. Ground reaction forces were measured at 2400 Hz while walking on a split-belt treadmill with two force plates (Bertec Corporation, Columbus, OH) and then low pass filtered at 6 Hz. A vertical ground reaction force threshold of 25 N was used to identify swing phase while accounting for noise in the data due to treadmill belt movement. The kinematics for each subject during the swing phase of a single gait cycle at self-selected speed was imported into SIMM (MusculoGraphics, Inc., Santa Rosa, CA). The motion data were used to animate a generic musculoskeletal model made up of 23 segments, with 36 muscles on each leg (Delp et al., 1990). Broad muscles (e.g., gluteal muscles) were divided into three compartments (anterior, medial, and posterior) for greater accuracy in modeling their geometry. Segment length and mass values and muscle origin and insertion points used were those assigned to the generic model (Delp et al., 1990). The mass matrix and muscle moment arms, which varied with limb configuration, were calculated in SIMM.

To find the potential muscle function at each joint, custom algorithms were written in Matlab (The MathWorks, Inc, Natick, MA) to calculate the effect of each muscle in isolation. All muscle forces were set to zero except the muscle being tested, which was set equal to half its estimated physiological maximal isometric force designated in the generic model (Delp et al., 1990). Muscles were tested only on the right side for healthy subjects, given the assumption of symmetry, and on the involved side for stroke subjects. The potential torque generated about each joint caused by the muscle of interest was calculated by multiplying the muscle force by the appropriate moment arm(s). This torque value (or values, for biarticular muscles) was used to compute the potential of each muscle to affect hip and knee flexion according to the following equation,

$$[\alpha] = [M(\Theta)]^{-1}[T]$$

where $\alpha$ represents a vector of joint accelerations, $M$ is the mass matrix at the current time step and depends on joint configurations ($\Theta$), and $T$ is the vector of joint torques generated by the muscle of interest.

Potential muscle function, or the joint accelerations induced by each muscle, was computed at each time step throughout swing phase and reported for the period corresponding to the onset of swing phase. From this analysis, we can deduce whether a muscle will accelerate each joint into flexion or extension when force is generated. The relative magnitude of each muscle’s induced acceleration reflects which muscle has the greatest potential to flex or extend the joint of interest.

For each subject, a modified set of data were also created by augmenting hip extension by 10° during the entire swing phase corresponding to increased hip extension at toe-off consistent with faster walking speed. Muscle potentials were calculated in this new configuration.

### Table 1 Descriptive subject information

<table>
<thead>
<tr>
<th>Subject</th>
<th>Gender</th>
<th>Age</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>Self-Selected Speed (m/s)</th>
<th>AFO</th>
<th>Time Since Stroke (mo.)</th>
<th>Lower Extremity Fugl-Meyer Score</th>
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<tr>
<td>Stroke 1</td>
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<td>66</td>
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<td>82</td>
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<td>Y</td>
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<tr>
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<td>79</td>
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<td>N</td>
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<td>8</td>
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<tr>
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<td>127</td>
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<td>Y</td>
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<td>80</td>
<td>1.25</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<tr>
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<td>75</td>
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<td>—</td>
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<td>89</td>
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<td>77</td>
<td>1.27</td>
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</table>
Results

Original Kinematics

Kinematic patterns at the hip and knee were different between groups and individual stroke subjects (Figure 1). The five healthy subjects exhibited similar self-selected walking speed (Table 1) and peak swing phase hip and knee flexion angles. Each stroke subject had a unique walking pattern with self-selected speed between 0.27 and 0.67 m/s with swing phase characterized by less knee flexion and greater hip flexion than healthy adults.

Because kinematics were similar and a generic model was used, induced accelerations from the healthy group were averaged; data from individual stroke subjects is also presented (Figures 2 and 3). In initial swing, as the knee flexes, the data showed soleus (SOL), medial and lateral gastrocnemius (MG and LG), vastus lateralis (VL), vastus intermedius (VI), vastus medialis (VM) and rectus femoris (RF) to have the greatest potential to accelerate the knee into extension in both healthy and stroke subjects (Figure 2A). The strongest knee flexors in early swing in both subject groups were tibialis anterior (TA), semimembranosus (SM), biceps femoris long and short heads (BFSH and BFLH), and semitendinosus (ST). The muscles observed to have the greatest potential to extend the hip in early swing were SM, BFLH, gluteus maximus.

![Figure 1 — (A) Hip and (B) knee flexion angles during swing phase for five healthy subjects (solid line with error bars) and three stroke subjects (see legend).](image-url)
(GMAX), VL, VI, VM, and ST. The muscles ranking highly in hip flexion acceleration potential included RF, iliacus (IL), and psoas (PS) (Figure 2B).

Stroke subjects generally showed lower muscle potentials to flex the knee, compared with healthy, though the vasti of stroke subjects outpaced the healthy group in knee extension acceleration potential (Figure 2A). However, the stroke group showed greater muscle potential than the healthy group to accelerate the hip into flexion or extension (Figure 2B).

**Figure 2** — (A) Induced acceleration of the knee in flexion (+) and extension (−). (B) Induced acceleration of the hip in flexion (+) and extension (−). The muscles shown represent those having the greatest acceleration potential at initial swing. In all cases, the force generated by each muscle in the analysis was half its maximal isometric force. The healthy data represents an average over five healthy subjects and demonstrates greater potential for knee flexion than stroke subjects, though generally less potential for hip flexion.
Hip Flexion Angle Perturbation

Increased hip extension in early swing improved the potential of biarticular muscles to flex and extend the knee but decreased the hip flexion potential. Specifically, the potential for knee extension due to RF increased substantially in initial swing for all subjects aside from stroke subject 2 (Figure 3A). Of the noted knee flexors, all subjects except stroke subject 2 showed SM, BFLH, and ST having increased knee flexion potential in early swing, while BFSH showed negligible change in potential across all subjects. Several muscles with the greatest hip flexion potential, including RF, IL, and PS, decreased for all subjects in their potential to accelerate the hip in early swing (Figure 3B).

Discussion

The results of this study give new insight into the potential function of muscles during the swing phase of both stroke and healthy gait, governed by kinematic differences that affect the ability of muscles to produce movement. With only a few exceptions, potential muscle function at the

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**Figure 3** — (A) Changes to induced acceleration of the knee into flexion or extension after perturbing the hip flexion angle 10° toward extension. (B) Changes in induced acceleration of the hip in flexion or extension after perturbing the hip flexion angle 10° toward extension. Positive changes mean the muscle has more potential to flex the knee (hip) when extension is increased, whereas negative changes mean the muscle has more potential to extend the knee (hip).
hip and knee joints agreed qualitatively with other studies (Kimmel & Schwartz, 2006; Riley & Kerrigan, 1998; Jonkers et al., 2003).

Previous studies investigating muscle potential in healthy gait examined joint accelerations generated per newton of muscle force (Kimmel & Schwartz, 2006). We accounted for the maximum isometric force each muscle can produce because we believe it is informative to understand the potential acceleration a muscle can generate based not only on its configuration but also on its potential strength. This distinction is especially important in considering stroke subjects, because it is clinically useful to understand which muscles, if strengthened, would be most likely to produce the largest functional improvements.

Somewhat surprisingly, SOL and TA had the highest potentials to accelerate the knee into extension and flexion, respectively. However, other studies have found similar large effects from these muscles (Anderson et al., 2004; Arnold et al., 2007). Although SOL is not actively contracting during swing, Anderson et al. (2004) found that the passive effects of SOL and other plantar flexors are responsible for significant knee extension acceleration in swing. As TA brings the foot into dorsiflexion, the knee flexion acceleration it generates acts to balance the effect of SOL, culminating in a small net flexion effect (Anderson et al., 2004). In many stroke subjects, limited dorsiflexion action may not be sufficient to oppose passive plantar flexor forces; conversely, spasticity secondary to stroke may cause inappropriate SOL activity, further inducing extension acceleration at the knee. Without the balancing effect of the dorsiflexor muscles generating knee flexion acceleration, however, the strong, unchecked effects of spastic SOL could help explain problems with excessive knee extension in swing. Thus, muscle training should aim to balance the effects of SOL and TA on swing phase knee flexion in stroke survivors.

Gastrocnemius also has the potential to accelerate the knee into extension although the effect is not as pronounced as that of soleus or vastus. Previous studies have also shown that the induced acceleration of this muscle changed from flexion to slight extension in the early swing period (Kimmel & Schwartz, 2006; Neptune et al., 2001). Because biarticular muscles have the ability to act contrary to their anatomical alignment (Zajac, 1993), we can expect the function of gastrocnemius to be sensitive to ankle and knee postures.

Our results indicate that the potential knee flexion acceleration of most knee flexors in swing was greater for healthy subjects than stroke subjects, agreeing with our hypothesis that stroke subjects’ muscles would have lower knee flexion potential due to posture alone. Thus, the paretic muscles are not only weaker but also in a worse position to generate knee flexion. Although toe-off knee flexion velocity has been cited as a primary factor in generating adequate peak knee flexion in swing, our results suggest swing phase muscle activity may also contribute (Anderson et al., 2004; Goldberg et al., 2003). Across all subjects, in early swing the magnitude of knee flexion acceleration potential generated by BFLH is positively correlated with knee flexion angle at toe-off. This correlation suggests that stiff-knee gait is self-perpetuating, where a lack of knee flexion at toe-off makes it even harder to attain adequate peak knee flexion as swing progresses.

Hip flexion moment has also been shown to contribute significantly to knee flexion during swing (Kerrigan et al., 1998). In our study, we found that for the two muscles with the greatest hip flexion acceleration potential, RF and IL, every stroke subject showed slightly higher magnitudes compared with healthy subjects, perhaps due to increased moment arms associated with increased hip flexion in this group. The fact that the stroke subjects demonstrated greater hip flexion acceleration potential suggests that even with diminished hip extension at toe-off compared with healthy adults, their gait pattern allows them to more effectively use residual muscle strength to produce hip flexion. Kerrigan et al. (1998) described a case where hip flexor strengthening improved peak knee flexion, and hip flexor strength has also been correlated with faster gait speeds (Milot et al., 2007). Because our results suggest that stroke subjects’ kinematics can allow them to take greater advantage of their hip flexors, our study supports hip flexor strengthening to improve stiff-knee gait.

Our hypothesis that knee flexion potential would increase in stroke subjects when hip flexion angle was perturbed into extension was largely supported by our results, since SM, BFLH, and ST showed a marked increase in their potential to accelerate the knee into flexion in our healthy subjects and two out of three stroke subjects. Unlike the healthy subjects, the stroke subjects’ hip flexion angles did not actually go into extension even after being perturbed. In addition, stroke subject 2 showed extremely diminished effects, compared with all other subjects, in both hip and knee flexion potential when hip flexion angle was perturbed. This subject’s gait was extremely slow (0.27 m/s) and exhibited much more hip flexion at initial swing compared with the other subjects. The divergent effects shown between subjects in their sensitivity to altered hip flexion angle reinforce the importance of individualized gait analysis in understanding subject-specific abnormalities and deficiencies.

Increasing extension of the hip at toe-off may increase the effectiveness of RF to generate hip flexion acceleration by elongating the fiber length of RF, thus increasing force production and torque at the hip and improving forward movement of the swing limb while contributing to sufficient knee flexion (Piazza & Delp, 1996). One of the goals of our study was to examine the changes in muscle potential in early swing when the hip angle was perturbed 10° into extension, while neglecting the stretch response of RF (i.e., muscle forces were held constant in our analysis). The potential of RF to accelerate the hip into flexion actually decreased in early swing when hip flexion angle was perturbed toward extension, which does not support our hypothesis that hip flexion potential would increase. Instead these results suggest...
that the increased force due to the elongated fiber length reported by Piazza & Delp (1996) outweighs the diminished potential of the hip flexors caused by postural differences.

Limitations to this study include the absence of ground contact forces in our model; however, our results reflect the ability of muscles to influence limb motion once swing has begun (Goldberg et al., 2004). Furthermore, we cannot examine the role of contralateral muscles using this technique. Our small sample size is another limitation, yet the diversity of impairments among stroke patients encourages the type of individual analyses performed in our study that still make our results valuable. Lastly, we failed to use age-matched subjects, but Oberg et al. (1994) studied the differences in gait kinematics in healthy men aged 10–79 years, and found that hip and knee joint angles changed only about 0.5° per decade. Because the differences in kinematics between our stroke and healthy groups were on the order of 30–40°, we concluded that the abnormal kinematics caused by stroke dominated those due to age.

Future work would benefit from a ground interface that would provide appropriately responsive ground reaction forces. Furthermore, it would be enlightening to include the effects of stance limb muscles on swing limb dynamics, since stance muscles have been shown to affect the swing limb (Jonkers et al., 2003). Eventually, it would be valuable to corroborate the results of this study by strengthening or stimulating specific muscles of stroke subjects and observing if the predicted changes in musculoskeletal dynamics can be achieved in actuality.

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References


