Preswing Knee Flexion Assistance Is Coupled With Hip Abduction in People With Stiff-Knee Gait After Stroke

James S. Sulzer, PhD; Keith E. Gordon, PhD; Yasin Y. Dhaher, PhD;
Michael A. Peshkin, PhD; James L. Patton, PhD

Background and Purpose—Stiff-knee gait is defined as reduced knee flexion during the swing phase. It is accompanied by frontal plane compensatory movements (eg, circumduction and hip hiking) typically thought to result from reduced toe clearance. As such, we examined if knee flexion assistance before foot-off would reduce exaggerated frontal plane movements in people with stiff-knee gait after stroke.

Methods—We used a robotic knee orthosis to assist knee flexion torque during the preswing phase in 9 chronic stroke subjects with stiff-knee gait on a treadmill and compared peak knee flexion, hip abduction, and pelvic obliquity angles with 5 nondisabled control subjects.

Results—Maximum knee flexion angle significantly increased in both groups, but instead of reducing gait compensations, hip abduction significantly increased during assistance in stroke subjects by 2.5°, whereas no change was observed in nondisabled control subjects. No change in pelvic obliquity was observed in either group.

Conclusions—Hip abduction increased when stroke subjects received assistive knee flexion torque at foot-off. These findings are in direct contrast to the traditional belief that pelvic obliquity combined with hip abduction is a compensatory mechanism to facilitate foot clearance during swing. Because no evidence suggested a voluntary mechanism for this behavior, we argue that these results were most likely a reflection of an altered motor template occurring after stroke. **(*Stroke*. 2010;41:1709-1714.)**

Key Words: gait ■ muscle synergies ■ robotics ■ stroke

Stiff-knee gait (SKG), defined as reduced and delayed knee flexion during the swing phase,[1] is a common disability in people with stroke. Individuals with SKG often exhibit exaggerated frontal plane movements such as hip circumduction and hip hiking.[2] These frontal plane movements are energetically demanding[3] and may lead to chronic joint pain.[4] There is believed to be a cause-and-effect relationship between these two sets of movements.[1] Reduced knee flexion decreases an individual's ability to clear the foot during the swing phase. In turn, subjects make compensatory frontal plane movements that facilitate toe clearance. Although this compensatory theory is commonly accepted, there are no quantitative in vivo studies examining the interaction between the swing phase knee flexion angle and proximal frontal plane kinematic behaviors in individuals with poststroke hemiparesis. If a kinematic connectivity indeed exists, it would indicate that the observed frontal plane behaviors may in part be a response to mechanical constraints imposed at the knee.

Alternatively, exaggerated frontal plane motions may be due to abnormal motor control. Recent studies in patients with stroke have found abnormal across-joint torque activation coupling, or synergies, between hip and knee activation. For example, several studies have found evidence of abnormal coupling of knee flexion/extension and hip abduction/adduction activity in both reflex[5] and voluntary[6,7] muscle activity. These findings suggest that improving swing phase knee flexion angle may not resolve exaggerated frontal plane movements.

Accordingly, we sought to examine the effect of assistive knee flexion torque during preswing on frontal plane gait compensations. To achieve this goal, we developed a lightweight, backdrivable actuator capable of selectively applying knee flexion torque during the preswing phase of gait without imposing any significant mechanical constraints on the subject.[8] To assess if the observed frontal plane behaviors during the assisted steps were a result of a mechanically coupled response to the "perturbation" or due to motor control compensation to the assistance, catch trials (no assist) were introduced at random.[9] If kinematic changes persist during the no assist trials, it would suggest the presence of an acute motor adaptation induced by knee flexion assistance.[10] Thus, to examine the potential effect of anticipatory motor commands to the repeated assistance, kinematic comparisons between the assisted and unanticipated steps with no assistance (catch trials) were conducted. Quantitative metrics of

Received April 8, 2010; accepted April 27, 2010.

From the Rehabilitation Institute of Chicago (J.S.S., K.E.G., Y.Y.D., M.A.P., J.L.P.), Chicago, Ill; the Departments of Mechanical Engineering (J.S.S., M.A.P.) and Biomedical Engineering (Y.Y.D.), Northwestern University, Evanston, Ill; and the Department of Bioengineering (J.L.P.), University of Illinois–Chicago, Chicago, Ill.

Correspondence to Yasin Dhaher, 345 E Superior Street, Suite 1407, Chicago, IL 60611. E-mail y-dhaher@northwestern.edu

© 2010 American Heart Association, Inc.

Stroke **is available at http://stroke.ahajournals.org** **DOI: 10.1161/STROKEAHA.110.586917**

Table. Data for 9 Hemiparetic Stroke Subjects*

Subject No.	Age, Years	G	W, kg	Post yrs	Meds	AFO	Speed, m/s	Modified Ashworth KE	Modified Ashworth KF	Knee (U/l), Degrees/ Degrees	Hip, Degrees	Pelvis, Degrees
1	49	M	59	1	N	N	0.80	0	0	71/16	1	13
2	18	M	52	2	Y	Y	0.76	0	0	54/32	9	7
3	59	F	58	16	N	N	0.53	3	1	53/19	−16	18
4	38	M	91	1	Y	Y	0.72	1	0	60/26	1	6
5	51	F	75	3	Y	Y	0.92	1	0	52/15	6	12
6	40	F	82	3	Y	N	1.1	3	0	54/34	−3	11
7	31	F	59	3	N	Y	0.74			54/32	6	6
8	67	M	54	7	N	N	0.38	2	3	64/30	−1	5
9	43	M	85	29	N	N	1.0	0	1	54/38	4	9
Mean	44		68	7			0.78			57/27	1	10
SD	15		15	9			0.23			6/8	7	4

*All subjects who wore an AFO used a hinged version. "Speed" refers to each subject's self-selected gait speed during overground walking. Modified Ashworth scores are for ankle knee extensors (KE) and knee flexors (KF) based on a 0 to 5 scale. "Knee" refers to the range of motion (unimpaired and impaired sides) of the knee during gait without the orthosis. "Hip" and "Pelvis" refer to the difference of maximum hip abduction and pelvic obliquity of the impaired limb and unimpaired limbs, respectively. Subject 7 did not have her Ashworth scores measured.

G indicates gender; W, weight; Post, years poststroke; Meds, taking Baclofen; AFO, use of an ankle–foot orthosis; M, male; F, female; N, no; Y, yes.

gait stability[11] were used to evaluate the potential effect that the knee flexion assistance may have had on locomotor stability in both the stroke and healthy participants. Knowledge gleaned from this study will improve our understanding of the relationship between knee impairments and the proximal 3-dimensional movements in people with SKG poststroke. Specifically, this study will help identify whether abnormal frontal plane behaviors are a response to local knee impairment or a manifestation of an abnormal multisegmental motor program. We argue that identifying these aberrant biomechanical interactions will help guide future clinical investigations and the development of assistive technology for individuals with stroke.

Methods

Nine hemiparetic persons with stroke (Table) gave written informed consent according to the guidelines approved by Northwestern University and Department of Veterans Affairs Institutional Review Boards.

Although SKG after stroke has clinically been recognized as reduced and delayed knee flexion angle during the swing phase,[1] a clear quantitative metric for the severity in SKG is lacking. Hence, we included subjects whose knee range of motion was at least 16° less on the affected limb than the unimpaired side. Subjects also had to be able to walk continuously for 20 minutes at 0.55 m/s. All participants were left-sided hemiparetics. Exclusion criteria were (1) knee hyperextension during the stance phase (genu recurvatum); and (2) inability to provide informed consent. Five nondisabled control subjects also completed the protocol. Some of the control data have been previously published.[10]

A powered knee orthosis known as the SERKA (Series Elastic Remote Knee Actuator) was used in the study.[8] The SERKA consists of a modified off-the-shelf anterior cruciate ligament knee brace that can be adjusted to fit comfortably about the paretic knee for each individual subject. A servomotor located away from the user controls the excursion of a sheathed (Bowden) cable transmission. The cable pulls on a capstan coupled to the brace by a compliant torsional spring controlling for spring deflection and, thus, torque (Figure 1). The device is capable of exerting large torques (28 N · m) in 40 ms with a maximum torque of 41 N · m. Because the motor is remotely located, the weight of the device on the leg is 1.2 kg (approximately 10% of limb mass). The unpowered device exerted no appreciable resistance (torque <1 N · m) to the subject's walking ability. During treadmill walking, no visual cues were given to the subjects. In addition, the actuators did not provide auditory cues detectable over the background noise of the treadmill.

Lower limb kinematics were collected at 100 Hz using an 8-camera video system (Motion Analysis, Santa Rosa, Calif). Thirty markers were placed bilaterally on the lower limbs and pelvis.[12]

Both the control and stroke subjects were instructed to walk on an instrumented split-belt ADAL treadmill (Tecmachine, Andrez Boutheon, France) at 0.55 m/s, approximately the average speed for people with SKG.[13] Subjects were instructed to walk as naturally as possible and allowed to hold onto the treadmill handrails. Subjects were also instructed to try to walk with 1 foot on either tread but to prioritize walking naturally. Each subject donned a harness to protect from falls but not support weight. An emergency stop switch was available to both the subject and experimenter (Figure 1). Subjects were permitted to wear their ankle–foot orthoses.

The treadmill vertical ground reaction forces were used to calculate the maxima and minima of center of pressure of both the paretic

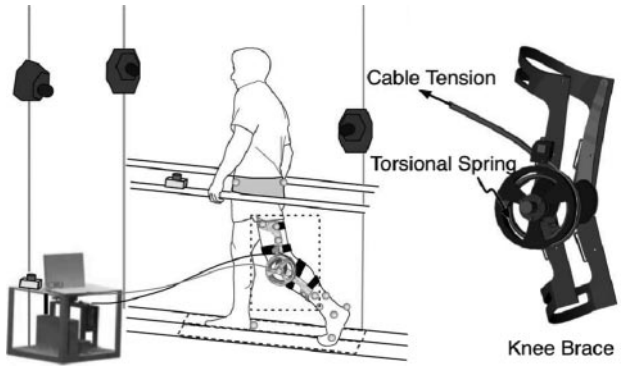

Figure 1. The experimental setup consisted of a subject walking on a split-belt force treadmill used to measure individual ground reaction forces. Kinematics was measured using motion capture cameras and reflective markers attached to both legs. Each subject wore a powered knee brace to provide knee flexion torque.

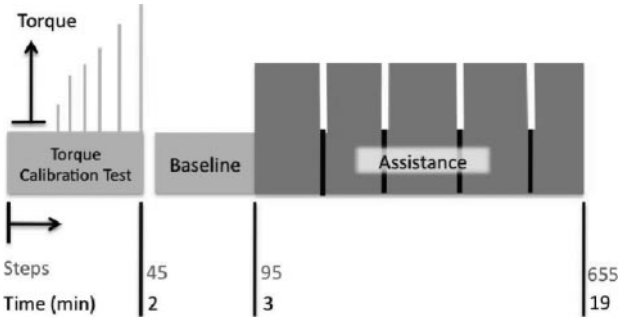

Figure 2. The experimental protocol consisted of 3 treadmill-walking stages. The torque calibration test determined the magnitude of the torque used during assistance. During this stage, a range of knee flexion torques was applied to determine an appropriate level of assistance for each subject. After the baseline stage (no assistance), the assistance stage provided constant, open-loop flexion torque assistance during the preswing phase of each step. There were 4 catch trials without assistance in this stage.

and nonparetic feet to identify gait events.[14] For control subjects, the powered knee orthosis exerted a flexion torque that began at contralateral heel strike and ended at swing phase initiation. However, stroke subjects had a longer double-support period; therefore, torque onset began 250 ms before paretic swing phase initiation (predicted from the timing of the previous step). This timing more closely matches the double-support time of healthy gait. The ramp time of the torque pulse was set at 170 ms. Preliminary data showed that the changes in ramp time did not affect outcome measures; hence, the ramp time was chosen to maintain an acceptable level of comfort for the stroke participants. Analog data, including force plate data and applied torque, was acquired at 1 kHz.

An initial torque calibration test was conducted during treadmill walking (45 steps) to determine the appropriate amplitude of knee flexion assistance for each of the participants. We applied knee flexion torque during preswing to random individual steps during the torque calibration test. Knee flexion torque began at 10 N · m and increased by approximately 3 N · m during each application to a maximum of 35 N · m. We selected the torque assistance amplitude to be used for the data acquisition test from post hoc torque calibration test data that corresponded to the greatest peak knee flexion during swing or 60° of knee flexion, whichever was lower.

The data acquisition test consisted of 610 steps, lasting approximately 16 minutes (Figure 2). During the first 50 steps (baseline), no forces were exerted on the subject. The last 10 steps of this phase were used as baseline data. During the next 560 steps (assistance), subjects received a knee flexion torque assistance every step as described previously. We collected 15 trials in 10-step increments interspersed throughout this phase. Also interspersed were 4 nonconsecutive trials with no torque assistance during a single step cycle (catch trials). Subjects were instructed to report any perceived changes in their gait patterns during the experimental paradigm to obtain a qualitative assessment of performance.

All data were imported into Matlab (Natick, Mass) with marker data and torque data time-synchronized. Kinematic data were filtered with a fifth-order low-pass Butterworth filter at 8 Hz. Each stride was separated and normalized at left foot strike determined using the maximum forward displacement of the toe marker. Pelvic obliquity was determined by calculating the rotation matrix between the coordinate system of the pelvis[15] and world coordinates followed by Euler angle decomposition. Because markers on the affected thigh were placed on the brace, therefore not directly on the thigh, thigh segment position was determined by a coordinate system, including the projected hip center from the pelvis and medial and lateral knee center from the shank. Hip abduction was calculated based on the Euler angle decomposition between this projected thigh coordinate system and the pelvis. Knee flexion angle was determined from the rotation matrix between the projected thigh and shank coordinates. Absolute angles were calculated from standing data. Center of mass movement was estimated from the centroid of the sacrum and left and right anterior–superior iliac spine markers.[16] Kinematics were linearly interpolated to match the difference in sampling frequency with torque data.

The main outcomes consist of the peak kinematics of the paretic side during the swing phase; specifically, maximum knee flexion and simultaneously with peak knee flexion, hip abduction, and pelvic obliquity. The kinematics during assistance was compared with baseline and catch trials. Any potential anticipation or adaptation would be found in catch trials. Thus, we used repeated-measures analysis of variance (ANOVA) with 3 levels: baseline, assistance (subject mean), and catch trials (subject mean) followed by Tukey-Kramer post hoc testing. A significance level of $\alpha=0.05$ was selected. We also used a 2-sample t test to compare these parameters between stroke and control data.

To assess the potential effect of loss of balance on the observed kinematic adaptations, the extrapolated center of mass was calculated. Comparing the extremes of this outcome, which comprised of lateral center of mass position and velocity, with lateral center of pressure resulted in a margin that, when negative, showed an instability or lean.[11] Another indirect measure of lean can be calculated through the sum of the lateral forces on the nonparetic foot. Both of these outcomes were compared using the same 3-level repeated-measures ANOVA mentioned previously.

Results

Peak kinematics without the orthosis shows a higher incidence of exaggerated pelvic obliquity but no correlation with peak knee flexion angle. When stroke subjects walked without the orthosis, peak knee flexion in the affected limb was 30.4±12.0° (mean±SD) less than the healthy limb (t test; $P<0.0001$). In addition, asymmetry was observed in pelvic obliquity (10±4.0°, t test; $P<0.001$) between the healthy and affected sides but not in peak hip abduction (1.0±7.0°, t test; $P=0.75$). Pelvic obliquity and hip abduction asymmetry were not significantly correlated with peak knee flexion angle asymmetry (Pearson, $P=0.34$ and 0.59, respectively).

Both groups increased knee and hip flexion angle in response to the assistance. Selected kinematic parameters are shown for representative subjects of the control and stroke groups in Figure 3 and Figure 4, respectively. Stroke subjects significantly increased peak knee flexion angle by 13.8±11.4° when receiving the supplemental knee flexion torque from the powered knee orthosis (repeated-measures ANOVA; $F_{[2,8]}=9.21$, $P=0.002$), and control subjects' knee flexion angle increased significantly by 12.4±3.3° above baseline (ANOVA; $F_{[2,4]}=26.0$, $P<0.001$). In both groups, knee flexion angle during knee flexion torque assistance was significantly greater than during catch trials (Tukey honestly significant difference [THSD]; $P<0.05$). There was no significant difference in knee flexion between the first and last steps of assistance (t test, $P>0.05$) for either group (Figure 5). There was no significant difference between the change in peak knee flexion of the control and stroke groups (t test, $P>0.05$).

Control subjects significantly increased hip flexion angle during assistance by 9.1±4.4° (ANOVA; $F_{[2,4]}=15.7$, $P=0.002$) and significantly more than during catch trials (THSD; $P<0.05$). Stroke subjects also increased hip flexion 9.9±8.8° (ANOVA; $F_{[2,8]}=7.42$, $P=0.005$), but not significantly higher than during catch trials (THSD; $P>0.05$).

Results from the repeated-measures ANOVA revealed small but significant shifts in hip abduction in response to assistance in the stroke group, 2.31±1.15° above baseline (ANOVA; $F_{[2,8]}=21.0$, $P<0.001$). No significant difference was detected

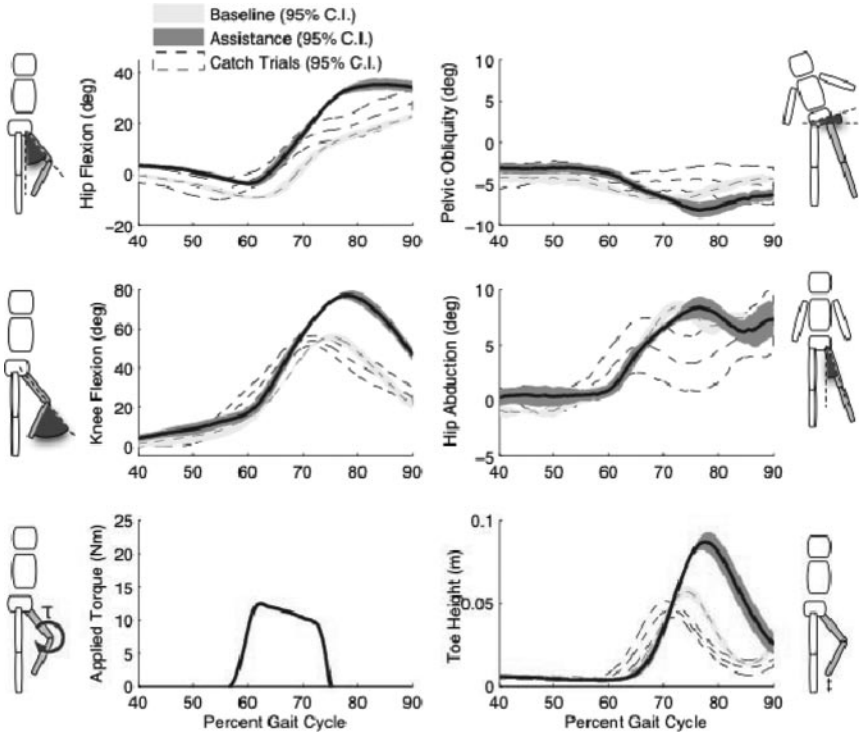

Figure 3. Kinematics from a representative control subject. Each trace is an average and 95% CI for 4 steps. Knee flexion torque increases peak knee flexion, hip flexion angle, and toe clearance, but has no apparent effect on frontal plane characteristics.

for control subjects (ANOVA; $F_{[2,4]}=3.54$, $P=0.079$). In stroke subjects, hip abduction increased significantly compared with baseline and catch trials (THSD; $P<0.05$). Neither group exhibited a significant difference from initial to final steps of assistance (t test, $P>0.05$; Figure 5). There were no significant differences between catch trials and baseline for stroke subjects or for control subjects (THSD; $P>0.05$). Stroke subjects' change in hip abduction was significantly higher than control subjects' (t test, $P<0.05$).

Peak pelvic obliquity significantly changed during this experiment for stroke (ANOVA; $F_{[2,8]}=5.00$, $P=0.021$), but not in control subjects (ANOVA; $F_{[2,4]}=1.96$, $P=0.203$). In the stroke group, catch trials were significantly greater than both baseline ($1.24\pm1.32°$) and assistance ($1.22\pm1.68°$; THSD; $P<0.05$), but no difference was found between assistance and baseline (THSD; $P>0.05$). Neither group exhibited a significant difference between first and last steps of assistance (t test, $P>0.05$; Figure 5).

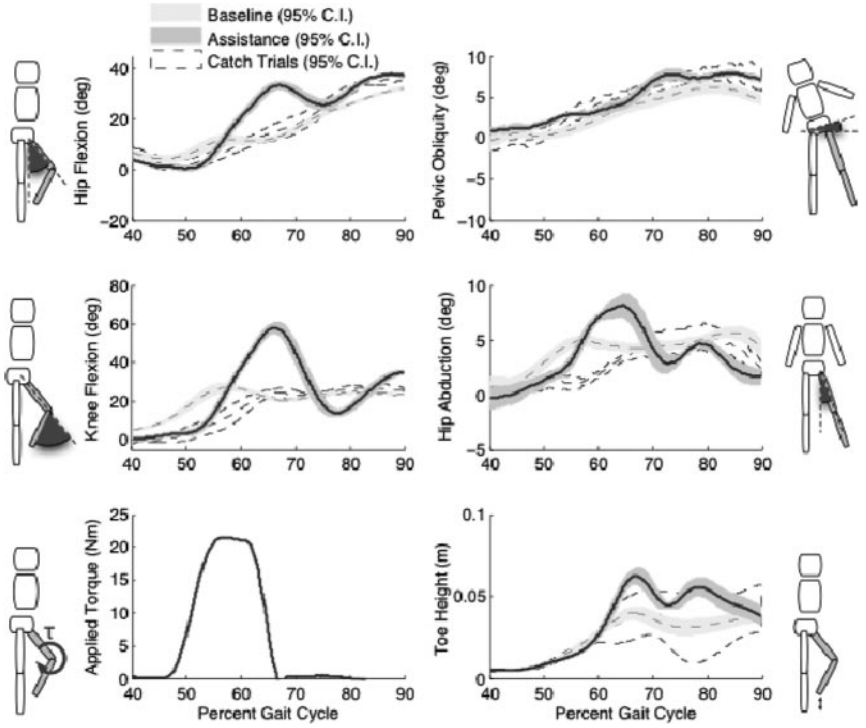

Figure 4. Kinematics from a representative stroke subject. Each trace is an average and 95% CI for 10 steps. At peak toe height, knee flexion, and hip flexion angle, when compensations are needed the least, more hip abduction occurs.

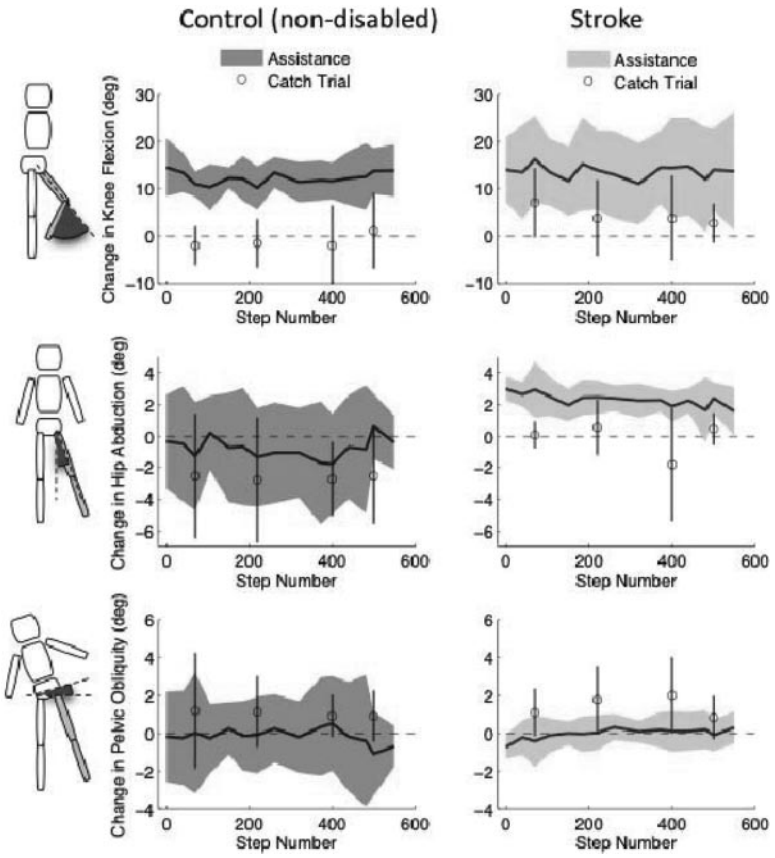

Figure 5. Key kinematic outcome measures show increase knee flexion of both control (left) and stroke (right) subjects, but a significant increase in hip abduction only for stroke subjects. Horizontal dashed line at zero represents baseline and is used for reference.

Finally, our statistical examination of the stability margins showed no significant change compared with baseline in either the stroke subjects (-7 ± 19 cm, ANOVA; $F_{[2,8]}=1.04$, $P=0.37$) or the control subjects (10 ± 16 cm, ANOVA; $F_{[2,4]}=0.95$, $P=0.42$) across conditions (baseline, catch, and assistance). The lateral force impulse on the right foot also showed no change from baseline in either the stroke (-25 ± 67 N·s, ANOVA; $F_{[2,8]}=0.66$, $P=0.53$) or control (-58 ± 43 N·s, ANOVA; $F_{[2,4]}=3.82$, $P=0.069$) groups. After the initial adaptation trials, subjects reported no perception of lack of balance during the knee assistance trials.

Discussion

In this study, we sought to examine the role of preswing knee flexion on frontal plane movement in SKG after stroke. As expected, we found an increase in knee flexion angle during swing when subjects received knee flexion torque assistance during preswing. Surprisingly, our findings also indicated that hip abduction increased when stroke subjects received assistive knee flexion torque at foot-off despite improved ability to clear the foot, whereas control subjects had no such behavior. Our data further indicated that these changes were neither a reflection of assistance-induced instability in the frontal plane nor leaning. These findings are in direct contrast to the traditional belief that hip abduction is solely a compensation to clear the foot during swing. Because no evidence suggested a voluntary mechanism for exaggerated abduction, we argue that these results were most likely a reflection of an altered motor template occurring after stroke.

One may argue that the increased hip abduction during assistance observed in stroke subjects is a strategy to enhance locomotor stability or as a reaction to the imposed knee flexion assistance. No significant changes in stability margins or leaning forces for either group were observed as a result of the assistance. Additionally, no subjects reported any perception of imbalance by midtraining. One may also argue that exaggerated abduction is an anticipatory gesture, but no such effect was observed in catch trials. These findings suggest that the observed kinematic changes are not due to imbalance, but may be a manifestation of a more intrinsic motor behavior mediated by the stroke.

The peak hip flexion angle of the control group was greater during assistance than during the catch trials. It is likely that these differences may be due in part to an increase in the shank-to-thigh intersegmental coupling mediated by the applied sagittal plane torque at the knee.[17] This mechanical effect was muted in the stroke group. Stroke subjects' peak hip flexion angle calculated during the assist trials was similar to those calculated during the unanticipated catch trials. This hip angle invariance provides indirect evidence of an abnormal neuromechanical coupling between the hip and knee sagittal plane mechanics in the stroke group.[18]

Our 3-dimensional analysis of the proximal kinematics indicated that hip abduction increased significantly compared with baseline and catch trials in the stroke group, whereas no frontal plane changes were observed in healthy control subjects. The presence of abnormal muscle synergies provides one possible explanation for the observed knee flexion-mediated increase in

hip abduction. A recent examination of lower limb volitional synergies found a coupling between voluntary hip abduction and knee flexion in patients with stroke in a static position imitating paretic foot-off.[7] Hence, it is possible that the greater hip abduction observed in this study may have been a reflection of such across-joint synergies observed under static conditions, a knee flexion coupled with hip abduction. It may also be possible that the exaggerated frontal plane movement was due to abnormal knee–hip reflex couplings, similar to those found previously in a seated posture in the same population.[5] To explore this, a thorough examination of lower limb muscle activity associated with the assistance is warranted. Delineating the differential effects of these multisegmental mechanisms to the observed behaviors is likely to improve clinical treatment of gait dysfunction after stroke and will provide guidance for future clinical and basic science investigations.

Musculoskeletal models are increasingly used to investigate underlying biomechanical factors in gait abnormalities after stroke[19]; such abnormalities are characterized by excessive frontal plane movements. However, most of the existing model-based analyses of such pathologies have thus far been limited to the sagittal plane mechanics.[18–20] Given the significant 3-dimensional interactions presented in this study, we argue that special attention should be given to the use of generic musculoskeletal models in the study of pathological gait. Specifically, our data indicated that an increase in knee flexion velocity preswing resulted in an increase in the peak knee flexion angle during swing, a finding that is consistent with musculoskeletal modeling predictions.[21,22] However, although sagittal plane behaviors were consistent across groups, 3-dimensional kinematic differences between the control and stroke groups persisted in response to the externally applied knee flexion torque. One could argue that these differences are potentially a manifestation of poststroke neural constraints.[23] Taken together, inclusion of such neural constraints in future computational studies may increase the efficacy of musculoskeletal modeling for the study of pathological gait such as in stroke.

Our goal in this study was to examine the kinematic connectivity between knee sagittal plane kinematic impairments and frontal plane movements proximal to the knee. In this context, subjects were instructed to "walk as naturally as possible." Our data suggest that abnormal frontal plane gait patterns persisted regardless of the imposed knee flexion assistance. It remains to be seen if, with additional kinematic feedback or instruction,[24] subjects would be able to volitionally reduce these aberrant frontal plane movements during gait. Thus, combining knee flexion assistance with supplemental strategies that reduce frontal plane gait compensations may improve walking efficiency in those with SKG after stroke.

Finally, our findings indicate that facilitation of knee flexion did not lead to the restoration of symmetrical gait behavior mediated by reduced hip hiking and normal hip abduction/adduction gait patterns. To this end, the current study provides a basic examination of recent developments in the assistive technology targeting the design of adaptive knee flexion tools to restore asymmetrical gait.

Acknowledgments

We thank T. George Hornby, PT, PhD, and Ross Bogey, DO, for their advice and help in subject recruitment.

Sources of Funding

This work has been supported by a predoctoral fellowship from the Department of Veterans Affairs and the Davee Foundation.

Disclosures

None.

References

1. Perry J. *Gait Analysis: Normal and Pathological Function.* Thorofare, NJ: Slack, Inc; 1992.
2. Kerrigan D, Frates E, Rogan S, Riley P. Hip hiking and circumduction: quantitative definitions. *Am J Phys Med Rehabil*. 2000;79:247.
3. Abdulhadi HM, Kerrigan DC, LaRaia PJ. Contralateral shoe-lift: effect on oxygen cost of walking with an immobilized knee. *Arch Phys Med Rehabil*. 1996;77:670–672.
4. Kerrigan D, Abdulhadi H, Ribaudo T, Croce U. Biomechanic effects of a contralateral shoe-lift on walking with an immobilized knee. *Arch Phys Med Rehabil*. 1997;78:1085–1091.
5. Finley J, Perreault E, Dhaher Y. Stretch reflex coupling between the hip and knee: implications for impaired gait following stroke. *Exp Brain Res*. 2008;188:529–540.
6. Rogers LM, Brown DA, Gruben KG. Foot force direction control during leg pushes against fixed and moving pedals in persons post-stroke. *Gait Posture*. 2004;19:58–68.
7. Cruz T, Dhaher Y. Evidence of abnormal lower-limb torque coupling after stroke: an isometric study. *Stroke*. 2008;39:139.
8. Sulzer J, Roiz R, Peshkin M, Patton J. A highly backdrivable, lightweight knee actuator for investigating gait in stroke. *IEEE Transactions on Robotics*. 2009;25:539–548.
9. Thoroughman K, Shadmehr R. Learning of action through adaptive combination of motor primitives. *Nature*. 2000;407:742.
10. Sulzer J, Gordon K, Hornby T, Peshkin M, Patton J. Adaptation to knee flexion torque during gait. International Conference for Rehabilitation Robotics. Kyoto, Japan; June 2009.
11. Hof A. The extrapolated center of mass concept suggests a simple control of balance in walking. *Hum Mov Sci*. 2008;27:112–125.
12. Cruz T, Lewek M, Dhaher Y. Biomechanical impairments and gait adaptations post-stroke: multi-factorial associations. *J Biomech*. 2009;42:1673–1677.
13. Kerrigan D, Karvosky M, Riley P. Spastic paretic stiff-legged gait: joint kinetics. *Am J Phys Med Rehabil*. 2001;80:244.
14. Roerdink M, Coolen B, Clairbois B, Lamoth C, Beek P. Online gait event detection using a large force platform embedded in a treadmill. *J Biomech*. 2008;41:2628–2632.
15. Wu G, Siegler S, Allard P, Kirtley C, Leardini A, Rosenbaum D, Whittle M, DíLima D, Cristofolini L, Witte H. ISB recommendation on definitions of joint coordinate system of various joints for the reporting of human joint motion. Part I: ankle, hip, and spine. *J Biomech*. 2002;35:543–548.
16. Gard S, Miff S, Kuo A. Comparison of kinematic and kinetic methods for computing the vertical motion of the body center of mass during walking. *Hum Mov Sci*. 2004;22:597–610.
17. Hunter B, Thelen D, Dhaher Y. A three-dimensional biomechanical evaluation of quadriceps and hamstrings function using electrical stimulation. *IEEE Trans Neural Syst Rehabil Eng*. 2009;17:167–175.
18. Riley P, Kerrigan D. Torque action of two-joint muscles in the swing period of stiff-legged gait a forward dynamic model analysis. *J Biomech*. 1998;31:835–840.
19. Higginson J, Zajac F, Neptune R, Kautz S, Delp S. Muscle contributions to support during gait in an individual with post-stroke hemiparesis. *J Biomech*. 2006;39:1769–1777.
20. Parvataneni K, Olney S, Brouwer B. Changes in muscle group work associated with changes in gait speed of persons with stroke. *Clin Biomech*. 2007;22:813–820.
21. Piazza S, Delp S. The influence of muscles on knee flexion during the swing phase of gait. *J Biomech*. 1996;29:723–733.
22. Goldberg S, Ounpuu S, Delp S. The importance of swing-phase initial conditions in stiff-knee gait. *J Biomech*. 2003;36:1111–1116.
23. Cruz T, Dhaher Y. The 31st Annual International IEEE EMBS Conference. Minneapolis, MN; September 2009.
24. Crippa A, Jonsdottir J, Parelli R, Rabuffetti M, Casiraghi A, Cattaneo D. Positional biofeedback to improve gait in subjects with stroke. *Gait Posture*. 2009;30:23.