Published in final edited form as:

Gait Posture. 2013 June ; 38(2): 270–275. doi:10.1016/j.gaitpost.2012.11.027.

Understanding compensatory strategies for muscle weakness during gait by simulating activation deficits seen post-stroke

Brian A. Knarr, PhD^{1,3}, Darcy S. Reisman, PT, PhD^{1,2}, Stuart A. Binder-Macleod, PT, PhD^{1,2}, and Jill S. Higginson, PhD^{1,3}

¹Biomechanics and Movement Science

²Dept. of Physical Therapy

³Dept. of Mechanical Engineering; University of Delaware Newark, DE

Abstract

Musculoskeletal simulations have been used to explore compensatory strategies, but have focused on responses to simulated atrophy in a single muscle or muscle group. In a population such as stroke, however, impairments are seen in muscle activation across multiple muscle groups. The objective of this study was to identify available compensatory strategies for muscle weakness during gait by simulating activation deficits in multiple muscle groups. Three dimensional dynamics simulations were created from 10 healthy subjects (48.8 ± 13.3 yrs, self-selected speed 1.28 ± 0.17 m/s) and constraints were set on the activation capacity of the plantar flexor, dorsiflexor, and hamstrings muscle groups to simulate activation impairments seen post stroke. When the muscle groups are impaired individually, the model requires that the plantar flexor, dorsiflexor, and hamstrings muscle groups are activated to at least 55%, 64%, and 18%, respectively, to recreate the subjects' normal gait pattern. The models were unable to recreate the normal gait pattern with simultaneous impairment of all three muscle groups. Other muscle groups are unable to assist the dorsiflexor muscles during early swing, which suggests that rehabilitation or assistive devices may be required to correct foot drop. By identifying how muscles can interact, clinicians may be able to develop specific strategies for using gait retraining and orthotic assistance to best address an individual's needs.

Keywords

Gait; Stroke; Musculoskeletal Simulation; Muscle Function

INTRODUCTION

Stroke is a leading cause of disability in the US. A common goal of stroke survivors is to regain community ambulation¹ and therefore, gait retraining is an important part of rehabilitation after stroke. Gait retraining often attempts to address abnormal gait

© 2012 Elsevier B.V. All rights reserved.

Corresponding Author: Brian A. Knarr Phone: 908-380-4413 bknarr@udel.edu University of Delaware 126 Spencer Lab Newark, DE 19716.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Conflict of interest statement The authors have no financial or professional conflicts of interest relating to this manuscript.

kinematics² that are related to functional deficits such as decreased walking speed after stroke. Such abnormal gait patterns occur in part due to compensation strategies used by the patient in an attempt to make up for inadequate muscle function.² By identifying how muscles may adopt different activation patterns to achieve ambulation in response to muscle impairment, the specific gait deficits observed in individual stroke survivors may be more readily understood.

Musculoskeletal simulations have been used to explore possible compensatory strategies in response to reduced force capability of a single muscle.³ Goldberg and Neptune (2007) used a forward dynamic simulation of normal walking to observe muscle compensations in response to plantar flexor, quadriceps, and hamstrings weakness over a gait cycle. This study highlighted the ability of the plantar flexor muscle group to compensate for weak hip and knee flexors and extensors. However, the model was unable to reproduce a normal walking pattern when the plantar flexor strength was reduced as a group. A study by Jonkers et al. (2003) examined the contributions of individual muscles during stance, concluding that a combination of multiple muscles is likely required to compensate for weakness in a single muscle.⁴ A study by Steele et al (2012) determined the minimum isometric force requirements for varying muscle groups in children with cerebral palsy during crouch gait by systematically reducing maximum isometric force of muscle groups⁵. Similarly, a recent study by van der Krogt et al (2012) studied the ability of models to reproduce healthy gait in response to muscle weakness induced by a reduction in maximum isometric force in various muscle groups⁶. This study concluded that weakness of individual muscles results in increased activation of the weak muscle and an overall increase in total muscle activation and cost of walking.

Because these studies manipulate the model parameters corresponding to maximum isometric force, they indirectly explore the effects of atrophy in a single muscle or muscle group on gait, and on the body's ability to compensate with other muscles to maintain normal gait. In a stroke population, however, there is often significant impairment of muscle activation. This is an important distinction, as atrophy and activation failure have different roles in the force production capacity of muscle. With activation impairment, the passive forces of the muscle are not weakened, and force production can exceed that of a fully activated atrophied muscle. This is an important distinction when considering the level of activation required for a muscle to produce a given force, as passive forces require no volitional effort. No modeling studies have looked at the effects of reduced muscle force due to simulated muscle activation impairment.

For many musculoskeletal simulations, the problem of muscle redundancy is solved using a cost function that minimizes a function of muscle activation⁷. Thus, predicted muscle patterns may differ when weakness is emulated by reduced maximum isometric force compared to activation levels. Consider the case when muscle is weakened by reducing maximum isometric force by 50%, the optimizer would require 100% activation of the muscle to reach its maximum force potential. In contrast, a muscle which has a reduction in maximum activation, but preservation of its maximum isometric force, the optimizer would require only 50% activation force (even less, if the preservation of passive forces is also considered) to achieve the same maximum force. In this scenario, an optimizer that minimizes muscle activation would calculate a greater cost at the same force production for a muscle with reduced maximum isometric force compared to reduced maximum activation. Similarly, a cost function which minimizes muscle stress⁶ (the ratio of force produced to maximum force) would also calculate a greater cost at the same force for a muscle with reduced maximum isometric force compared to reduced maximum activation.

The compensation strategies employed by patients with activation impairment may be different than those with muscle atrophy, particularly for neurologically impaired populations such as stroke, who often demonstrate preservation or enhancement of passive muscle forces⁸ concurrent with activation impairments. Recently, Barber et al (2012) demonstrated that young adults with spastic CP may have a greater ability to produce passive force through altered muscle-tendon properties. Consideration and preservation of passive properties in musculoskeletal simulations may be of increased importance when studying pathologic gait⁹.

The objective of this study was to identify available compensatory strategies for muscle weakness during gait by simulating activation deficits in multiple muscle groups. We created three dimensional dynamic simulations from healthy walking data. The effects of activation impairment were explored by unilaterally constraining the activation of the plantar flexor, dorsiflexor, and hamstrings muscle groups in the simulations to emulate activation deficits seen post-stroke. Muscle groups were impaired individually in a first group of simulations, then simultaneously in a second set of simulations. Muscle coordination and function were compared between the healthy and impaired simulations to identify plausible compensation strategies. We hypothesized that with a combination of plantar flexor, dorsiflexor, and hamstrings impairment, the model would no longer be able to reproduce healthy gait.

METHODS

A total of 10 healthy subjects (48.8 ± 13.3 yrs, self-selected speed 1.28 ± 0.17 m/s) participated in this study. All subjects read and signed an informed consent form approved by the Human Subjects Review Board at the University of Delaware. Data were collected on an instrumented split belt treadmill (Bertec Corp., 2000Hz) using an 8 camera motion capture system (Motion Analysis Corp., 200Hz) with the subjects walking at their self-selected speed.

A 23 degree of freedom model with 54 muscle actuators was used to generate a 3D, forward dynamic simulation based on motion capture walking trials from each subject.¹⁰ Inverse kinematics and residual reduction analysis (RRA) were run on all models with the results of RRA within acceptable limits for simulations as outlined in the OpenSim User's Guide. Using Computed Muscle Control (CMC),¹¹ the muscle forces and activations required to reproduce normal gait kinematics and joint torques were computed. Constraints were set on the activation capacity of the plantar flexor (medial gastrocnemius, soleus, tibialis posterior), dorsiflexor (tibialis anterior), and the primary hamstrings (biceps femoris long and short head) muscle groups to simulate activation impairment seen post-stroke^{12–16}. Each muscle group was progressively impaired individually and CMC was repeated at each level of impairment until the model could no longer generate the joint torque (within 5%) required to produce the subject's normal gait pattern, as quantified by the reserve actuator torque (Figure 1). From this, the minimum level of activation required for each muscle group was determined through trial and error refinement with a minimum step of 0.5% activation. CMC was run a final time to reoptimize muscle controls with the imposed muscle activation limitations. The final calculated set of muscle activations, forces and resulting kinematics were analyzed to determine compensation strategies.

To quantify the changes in muscle activation, we calculated percent activation as the average activation of a muscle over a period of time. This value was computed during the second double support phase of gait while the limb of interest is the trailing limb for the plantar flexors, first half of swing for the dorsiflexors, and first half of stance for the

hamstrings. Paired t-tests were used to compare the average percent activation between the normal and impaired simulations for each muscle in the model.

A muscle force perturbation was performed to quantify individual muscle contribution to joint and center of mass accelerations.¹⁷ This was done for the plantar flexors during the second double support, in light of recent studies that have highlighted the importance of plantar flexor function in post-stroke gait,^{16,18} and the dorsiflexors during the first half of the swing phase of gait, to examine the effect of impaired function on foot drop. The accelerations induced by each muscle were averaged over the gait phase of interest. Paired t-tests were used to assess significant ($p < 0.05$) differences for each muscle between the normal and impaired simulations.

Using the results from the simulations of individual muscle group impairment, a final set of simulations was run with plantar flexor, dorsiflexor and hamstrings impaired simultaneously. The simulations were run with a combination of the maximum plantar flexor, dorsiflexor and hamstrings impairment that could reproduce the experimental kinematics and kinetics when the muscle groups were impaired alone. From the results of these simulations, we identified the stages of gait during which the model could not reproduce the required joint torques and determined the percentage of torque that could not be generated (or percent weakness).

RESULTS

Plantar flexor Impairment

Muscle Activation—Overall, the simulations required that the plantar flexors must have the capacity to be activated to at least $55.6 \pm 8\%$ to allow the model to reproduce the subjects' normal gait pattern (Figure 2). Nine muscles showed a significant ($p < .05$) change of greater than 5% in percent activation between the normal and plantar flexor-impaired model during double support (Figure 2, Figure 3a). As expected, the medial gastrocnemius had a significant decrease in activation in the impaired model, decreasing $21.1 \pm 6\%$. Concurrently, the tibialis anterior decreased $4.3 \pm 4\%$ after plantar flexor impairment. In contrast, the tibialis posterior exhibited an increase in activation of $8.3 \pm 6.1\%$. The remaining six muscles exhibited a significant increase in activation in the impaired model, with the largest increase of $22 \pm 6\%$ occurring for biceps femoris short head.

Muscle Function

Knee Joint Acceleration: All muscles that changed activation except the tibialis anterior showed a change in knee joint acceleration during double support in the plantar flexor-impaired model. The medial gastrocnemius showed a change towards knee extension, decreasing its contribution to knee flexion acceleration (Figure 3b) while the psoas, iliacus, and biceps femoris short head showed the largest increases in knee flexion acceleration over the period of double support in the impaired model. In contrast to the short head, the biceps femoris long head caused $38.3 \pm 30.9\%$ more knee extension acceleration for the impaired model during double support.

Forward center of mass acceleration: In the plantar flexor-impaired model, the medial gastrocnemius showed a significant decrease in its contribution to forward center of mass acceleration, reducing $0.45 \pm 0.15 \text{ m/s}^2$ from the normal to impaired case (Figure 3c) during double support. The iliacus, psoas, and biceps femoris short head showed significant increases in forward center of mass acceleration. Additionally, the tibialis anterior decelerated the center of mass less during double support.

Dorsiflexor Impairment

Muscle Activation—Overall, the simulations required that the dorsiflexors must have the capacity to be activated to at least $64 \pm 15.4\%$ to reproduce subjects' normal gait patterns (Figure 2). Four muscles showed a change of greater than 5% in percent activation between the normal and dorsiflexor impaired model during the first half of swing (Figure 4a). In addition, tibialis anterior showed a decrease of $4.6 \pm 5.5\%$. Three subjects did not have a decrease in total dorsiflexor activation percentage with impairment during early swing. The biceps femoris short head showed the largest increase in activation at $19.5 \pm 4.3\%$, followed by the tensor fasciae latae at $13.3 \pm 8.8\%$.

Muscle Function

Ankle Joint Acceleration: Of the four muscles with increases in activation, only the biceps femoris short head showed a significant change in ankle function, increasing its contribution towards ankle dorsiflexion acceleration during early swing in the dorsiflexor-impaired model. The increased dorsiflexion acceleration by the biceps femoris short head was only a small fraction of the acceleration induced by the tibialis anterior.

Hamstrings Impairment

Muscle Activation—Overall, the simulations required that the hamstrings must have the capacity to be activated to $18.9 \pm 13.2\%$ to reproduce the subjects' normal gait pattern (Figure 2). Thirteen muscles showed a significant change in muscle activation during early stance with hamstrings impairment (Figure 4b). Five muscles exhibited decreased activation, with the biceps femoris long head showing the largest decrease. Conversely, the tibialis anterior and medial gastrocnemius had the largest increases in activation with hamstrings impairment.

Combined Impairment

For the combined impairment model, the maximum possible plantar flexor, dorsiflexor, and hamstrings activations were constrained to the lowest level of activation that could reproduce subject's normal gait pattern, as determined from the individually impaired models. With simultaneous plantar flexor, dorsiflexor, and hamstrings activation impairment, none of the models from any subject were able to reproduce the experimental joint torques required for healthy gait. Nine models demonstrated inadequate knee flexion moment during early stance ($24.7 \pm 12.7\%$ weakness) and seven of the models demonstrated inadequate knee flexion moment during double support ($33.1 \pm 17.2\%$ weakness). Three of the ten models were unable to produce adequate plantar flexion moment during double support ($9.8 \pm 3.9\%$ weakness) and three models were unable to produce adequate dorsiflexion moment during early swing ($7.2 \pm 0.4\%$ weakness).

DISCUSSION

The three dimensional dynamic simulations of gait created in this study identified compensatory strategies available to cope with muscle weakness of the plantar flexor, dorsiflexor and hamstring muscles. With weakness isolated to the hamstrings or plantar flexor groups, other muscles were able to compensate to successfully reproduce the subjects normal gait pattern. With concurrent weakness in these muscle groups, however, the model is unable to reproduce the necessary torques required to enable a healthy gait pattern, suggesting the need for orthotic or rehabilitative intervention.

Effects of Plantar flexion Impairment

The plantar flexor-impaired model demonstrated that changes in muscle activation are greatest during the double support phase of gait. This period of gait is important for both forward acceleration and for generating knee flexion during swing.¹⁹ To compensate for plantar flexor deficiency, our model demonstrated that significant increases in the activation of the hamstrings and hip flexors are needed. The biceps femoris short head exhibited the largest increases in muscle activation with plantar flexor activation impairment, a compensation that has also been reported previously.⁴ The simulation results are also consistent with what has been observed during post-stroke gait. Specifically, Nadeau et al (1999) has shown that while plantar flexor weakness is a major contributor to slow gait speed after stroke, patients who were able to generate large hip flexor torques were able to overcome the effect of plantar flexor weakness and walk at a relatively rapid pace by pulling the limb forward rather than pushing off the ground with the plantar flexors. Recent rehabilitation protocols have begun focusing on improving limb position and plantar flexor activation during double support to improve post-stroke gait²⁰ and our results indicate that this may be particularly important for those stroke survivors who have concomitant plantar flexor and hip flexor weakness.

It is likely that the increased knee flexion acceleration generated by these muscles reflects an attempt to compensate for reduced knee flexion contribution from the medial gastrocnemius and tibialis anterior, as well as increased knee extension from the biceps femoris long head. Contribution of the hamstrings to knee extension due to bi-articulation has been previously reported in a healthy population.²¹

Effects of Dorsiflexor Impairment

Dorsiflexion moment is important during the swing phase of gait to lift the foot and provide floor clearance. However, the model was only able to compensate for a small loss in dorsiflexion activation during early swing (~4.5%). Foot drop during the swing phase of gait is a common issue associated with dorsiflexor weakness, and is often seen in post-stroke gait.² It is likely that the inability of proximal muscles to compensate for dorsiflexor weakness during early swing is a contributing factor to the selection of abnormal gait patterns such as hip hiking and circumduction, which are commonly used to accommodate foot drop by increasing swing phase foot clearance.²

Effects of Hamstrings Impairment

For the impaired hamstrings model, knee flexion moment during early stance was a limiting factor in reproducing the subjects' healthy gait pattern. It is possible that weakness due to hamstring impairment during early stance may cause insufficient knee flexion torque, leading to hyperextension of the knee. This provides one possible explanation for the knee hyperextension seen in persons post-stroke, where a rapid extension of the knee occurs shortly after initial contact.²

Effects of Multiple Muscle Impairment

Generally when one muscle is weakened, other muscles produce more force to compensate. However, when multiple muscle groups are impaired, our simulations demonstrate that the ability to compensate is greatly diminished. It was shown that the hamstrings and plantar flexor muscle groups are able to compensate for each other when only one group is impaired. However, when both muscle groups are impaired simultaneously, the model is no longer able to achieve the necessary joint torques required for normal healthy gait, particularly at the knee during early stance and double support. Seven of the ten combined impairment models demonstrated insufficient knee flexion moment during double support.

This provides a potential explanation for the decreased knee flexion observed during the swing phase of gait in some stroke survivors.² That is, our results suggest that stroke survivors who have combined impairment of the plantar flexor, dorsiflexor and hamstring muscles do not have the compensatory muscle activations necessary to produce a pre-swing knee flexion moment that can lead to appropriate knee flexion during swing. In contrast, data from the plantar flexor-impaired model suggests that stroke survivors who do not have substantial impairments of the short head of the biceps may be able to compensate for effects of plantar flexor impairment on knee acceleration through greater activation of uniarticular hamstrings.

Limitations

This set of simulations explores a simplistic case of isolated activation impairment and the effect on gait. In a pathological population, a combination of both atrophy and activation impairment is likely to occur to varying degrees across many muscles. The cost function used in our simulations minimizes the sum of the squares of the muscle stress. This could have an impact on the compensation strategy selected by the simulation and may not be appropriate for post-stroke gait. Future studies should explore muscle compensation when realistic deviations in gait are permitted. Inclusion of subject-specific parameters in the model would allow for better prediction of compensation strategies. Quantification of subject-specific impairments may enable personalized analysis of muscle compensation strategies and will be addressed in future studies.

Potential application to rehabilitation

Manipulation of activation capacity in a set of musculoskeletal simulations revealed changes in muscle function in response to muscle deficits. When plantar flexors are impaired, the biceps femoris long head served to extend the knee during double support as a result of its extension at the hip. In fact, hip flexors, which do not cross the knee joint, showed more net contribution to knee flexion than the hamstrings. With additional impairment of the hamstrings muscle group, the hip flexors were unable to further compensate to maintain healthy kinematics. This suggests that training of the hip flexors and hamstrings may help impaired post-stroke patients achieve more normal knee kinematics. Moreover, the results suggest that in a population with combined hamstrings and plantar flexor weakness, achieving normal kinematics may not be possible without either improving muscles' force generating ability or use of an assistive device depending on the individual's overall level of impairment.

In this study, muscle weakness was simulated through reduced maximum activation of the muscle as opposed to reduced maximum isometric force. This is a novel approach that allowed exploration of model results in response to impairment while preserving passive forces within impaired muscle groups, which can occur in neurologically impaired populations such as stroke⁸. The results of this study suggest that compensation for impaired dorsiflexor activity through the activation of other muscles is virtually impossible. Therefore, in the absence of an assistive device, rehabilitation to improve dorsiflexor activation is necessary to achieve normal gait kinematics when this muscle is impaired.

By identifying how muscles can interact and adapt in response to activation impairment, as well as which muscle groups can have the greatest net effect, clinicians may be able to develop specific strategies focusing on gait retraining and orthotic assistance to optimize the gait pattern tailored to an individual's needs.

Acknowledgments

We would like to acknowledge Tyler Richardson and Andy Kubinski for their assistance with data collection. Funding was provided by NIH NS 055383, NIH NR 010786, and NIH P20-RR16458.

REFERENCES

1. Bohannon RW, Horton M, Wikholm J. Importance of four variables of walking to patients with stroke. *Int J Rehabil Res.* 1991; 14(3):246–250. [PubMed: 1938039]
2. Olney SJ, Richards C. Hemiparetic gait following stroke. Part I: Characteristics. *Gait and Posture.* 1996; 4(2):136–148.
3. Goldberg EJ, Neptune RR. Compensatory strategies during normal walking in response to muscle weakness and increased hip joint stiffness. *Gait and Posture.* 2007; 25(3):360–367. [PubMed: 16720095]
4. Jonkers I, Stewart C, Spaepen A. The complementary role of the plantarflexors, hamstrings and gluteus maximus in the control of stance limb stability during gait. *Gait and Posture.* 2003; 17(3): 264–272. [PubMed: 12770640]
5. Steele KM, van der Krogt MM, Schwartz MH, Delp SL. How much muscle strength is required to walk in a crouch gait? *Journal of Biomechanics.* 2012; 45(15):2564–2569. [PubMed: 22959837]
6. van der Krogt MM, Delp SL, Schwartz MH. How robust is human gait to muscle weakness? *Gait & Posture.* 2012; 36(1):113–119. [PubMed: 22386624]
7. Erdemir A, McLean S, Herzog W, van den Bogert AJ. Model-based estimation of muscle forces exerted during movements. *Clinical Biomechanics.* 2007; 22(2):131–154. [PubMed: 17070969]
8. Lorentzen J, Grey MJ, Crone C, Mazevet D, Biering-Sørensen F, Nielsen JB. Distinguishing active from passive components of ankle plantar flexor stiffness in stroke, spinal cord injury and multiple sclerosis. *Clinical Neurophysiology : Official Journal of the International Federation of Clinical Neurophysiology.* 2010; 121(11):1939–1951. [PubMed: 20457538]
9. Barber L, Barrett R, Lichtwark G. Medial gastrocnemius muscle fascicle active torque-length and Achilles tendon properties in young adults with spastic cerebral palsy. *Journal of Biomechanics.* 2012; 45(15):2526–2530. [PubMed: 22867763]
10. Delp SL, Anderson FC, Arnold AS, Loan P, Habib A, John CT, et al. OpenSim: open-source software to create and analyze dynamic simulations of movement. *IEEE Transactions on Biomedical Engineering.* 2007; 54(11):1940–1950. [PubMed: 18018689]
11. Thelen DG. Generating dynamic simulations of movement using computed muscle control. *Journal of Biomechanics.* 2003; 36(3):321–328. [PubMed: 12594980]
12. Klein CS, Brooks D, Richardson D, McIlroy WE, Bayley MT. Voluntary activation failure contributes more to plantar flexor weakness than antagonist coactivation and muscle atrophy in chronic stroke survivors. *Journal of Applied Physiology.* Aug.2010 :1337–1346. 2010. [PubMed: 20724561]
13. Horstman AM, Beltman MJ, Gerrits KH, Koppe P, Janssen TW, Elich P, et al. Intrinsic muscle strength and voluntary activation of both lower limbs and functional performance after stroke. *Clinical Physiology and Functional Imaging.* 2008; 28(4):251–261. [PubMed: 18355344]
14. Prado-Medeiros CL, Silva MP, Lessi GC, Alves MZ, Tannus A, Lindquist AR, et al. Muscle atrophy and functional deficits of knee extensors and flexors in people with chronic stroke. *Physical Therapy.* 2012; 92(3):429–439. [PubMed: 22135704]
15. Kesar TM, Reisman DS, Roos M, Perumal R, B.Farquhar W, Binder-Macleod S. Novel Locomotor Training Improves Gait Performance, Activity, and Participation in Individuals with Chronic Stroke. *Physical Therapy Combined Sections Meeting.* 2010; 34
16. Kesar TM, Perumal R, Reisman DS, Jancosko AM, Rudolph KS, Higginson JS, et al. Functional electrical stimulation of ankle plantarflexor and dorsiflexor muscles: effects on poststroke gait. *Stroke.* 2009; 40(12):3821–3827. [PubMed: 19834018]
17. Liu MQ, Anderson FC, Pandy MG, Delp SL. Muscles that support the body also modulate forward progression during walking. *Journal of Biomechanics.* 2006; 39(14):2623–2630. [PubMed: 16216251]

18. Peterson CL, Hall AL, Kautz SA, Neptune RR. Pre-swing deficits in forward propulsion, swing initiation and power generation by individual muscles during hemiparetic walking. *Journal of Biomechanics*. 2010; 43(12):2348–2355. [PubMed: 20466377]
19. Neptune RR, Kautz SA, Zajac FE. Contributions of the individual ankle plantar flexors to support, forward progression and swing initiation during walking. *Journal of Biomechanics*. 2001; 34(11): 1387–1398. [PubMed: 11672713]
20. Kesar TM, Reisman DS, Perumal R, Jancosko AM, Higginson JS, Rudolph KS, et al. Combined effects of fast treadmill walking and functional electrical stimulation on post-stroke gait. *Gait and Posture*. 2010
21. Arnold AS, Anderson FC, Pandy MG, Delp SL. Muscular contributions to hip and knee extension during the single limb stance phase of normal gait: a framework for investigating the causes of crouch gait. *Journal of Biomechanics*. 2005; 38(11):2181–2189. [PubMed: 16154404]

Highlights

- Three dimensional dynamics simulations were created from 10 healthy subjects.
- Constraints were set on the activation capacity of muscle groups to simulate activation impairments seen post stroke.
- Muscle groups are impaired individually using simulation techniques.
- To compensate for plantar flexor deficiency increases in the hamstrings and hip flexors were needed.
- Muscle groups are unable to assist the dorsiflexors during early swing.

Figure 1. Plantar flexor, dorsiflexor and ankle reserve moments for a representative subject at the greatest level of plantar flexor impairment allowed by study criteria. Ankle reserve moment reaches ~5% of plantar flexor moment just prior to 60% of the gait cycle.

Figure 2.

Average muscle activation for 10 subjects showing the targeted muscles with and without muscle activation impairment for the impaired hamstrings simulations (BF LH/SH – Biceps Femoris Long Head/Short Head), impaired plantar flexors simulations (MG – Medial Gastrocnemius, Sol – Soleus, TibPost – Tibialis Posterior) and impaired dorsiflexor simulations (TibAnt – Tibialis Anterior).

Figure 3.

(a) Change in percent activation of the nine muscles exhibiting changes greater than 5% during the double support phase of gait with plantar flexor impairment. (b) Change in knee joint acceleration during the double support phase of gait with plantar flexor impairment. Positive values represent extension. All changes except those in the tibialis anterior were significant ($p < 0.05$) (c) Change in forward center of mass acceleration induced by each muscle during the double support phase of gait with plantar flexor impairment. All except biceps femoris long head and tibialis posterior are significant ($p < 0.05$) (ant: anterior portion; LH: long head; SH: short head).

Figure 4.

(a) Change in percent activation of the four muscles exhibiting changes greater than 5% during the early swing phase of gait with dorsiflexor impairment. Tibialis anterior is also included as it the muscle of focus in the simulation. (b) Change in percent activation of the thirteen muscles exhibiting significant change (greater than 5%) during the first half of stance with hamstrings activation impairment. 'Ant,' 'int,' and 'post' represent the anterior, intermediate, and posterior portions of the muscle.