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# Empirical evaluation of gastrocnemius and soleus function during walking

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#### ABSTRACT

Distinguishing gastrocnemius and soleus muscle function is relevant for treating gait disorders in which abnormal plantarflexor activity may contribute to pathological movement patterns. Our objective was to use experimental and computational analysis to determine the influence of gastrocnemius and soleus activity on lower limb movement, and determine if anatomical variability of the gastrocnemius affected its function. Our hypothesis was that these muscles exhibit distinct functions, with the gastrocnemius inducing limb flexion and the soleus inducing limb extension. To test this hypothesis, the gastrocnemius or soleus of 20 healthy participants was electrically stimulated for brief periods (90 ms) during mid- or terminal stance of a random gait cycle. Muscle function was characterized by the induced change in sagittal pelvis, hip, knee, and ankle angles occurring during the 200 ms after stimulation onset. Results were corroborated with computational forward dynamic gait models, by perturbing gastrocnemius or soleus activity during similar portions of the gait cycle. Mid- and terminal stance gastrocnemius stimulation induced posterior pelvic tilt, hip flexion and knee flexion. Mid-stance gastrocnemius stimulation also induced ankle dorsiflexion. In contrast mid-stance soleus stimulation induced anterior pelvic tilt, knee extension and plantarflexion, while late-stance soleus stimulation induced relatively little change in motion. Model predictions of induced hip, knee, and ankle motion were generally in the same direction as those of the experiments, though the gastrocnemius' results were shown to be quite sensitive to its knee-to-ankle moment arm ratio.

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#### 1. Introduction

Distinguishing the relative function of the gastrocnemius and soleus is relevant for treating gait disorders (e.g. equinus) in which abnormal plantarflexor activity may contribute to pathological movement patterns (Etnyre et al., 1993; Perry et al., 1974; Svehlik et al., 2010; Zwick et al., 2004). Due to their similar activation profiles and distal insertion onto the Achilles tendon, the gastrocnemius and soleus were traditionally assumed to have similar function during gait. However, this may not be true due to differences in architecture between the muscles. Most notably, the gastrocnemius is biarticular with the capacity to generate knee flexion and ankle plantarflexion moments. The soleus, on the other hand, is a uniarticular muscle, generating only a plantarflexion moment. This distinction is important to consider in whole body

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http://dx.doi.org/10.1016/j.jbiomech.2014.07.007 0021-9290/© 2014 Elsevier Ltd. All rights reserved. movement, where muscles have the ability to accelerate joints they do not span via dynamic coupling (Zajac, 1993).

Prior work has indeed suggested unique functions for the soleus and gastrocnemius during gait, though conclusions differ between studies. Models by Pandy et al. (2010) and Neptune et al. (2004) predicted that the muscles generate opposite accelerations at the hip (gastrocnemius flexes, soleus extends), but accelerations in the same direction at the knee (extension). In contrast, other models suggest the gastrocnemius may induce knee flexion (Kimmel and Schwartz, 2006; Neptune et al., 2001). The unique function was confirmed experimentally by a group who found that electrical stimulation of the gastrocnemius and soleus induced opposite motion at the knee and ankle during stance (Stewart et al., 2007). Surprisingly they found that the gastrocnemius induced ankle dorsiflexion, which may reflect the action of the knee flexion moment of the muscle in more extended postures (Zajac and Gordon, 1989). However a limited number of subjects were tested in the Stewart study and external muscle stimulation persisted through the majority of stance, making it challenging to delineate postural effects.

Another important consideration is the effect of anatomical variation on muscle function. Zajac and Gordon (1989) showed that, in standing, the motion generated at a joint by the gastrocnemius will vary based on posture and the ratio of knee to ankle moment arms. However this phenomenon has not been explored in the context of walking, where posture and support conditions continually change. Prior modeling studies of plantarflexor muscle contributions to gait (Kimmel and Schwartz, 2006; Liu et al., 2008; Neptune et al., 2001, 2004, 2008; Neptune and McGowan, 2011; Pandy et al., 2010) have used generic models without considering the influence that assumed geometry may have on results.

The first objective of this study was therefore to empirically measure lower limb movement induced by the soleus and gastrocnemius when activated at specific portions of the stance phase of gait. Based on prior work we hypothesized that gastrocnemius stimulation would induce limb flexion, whereas soleus stimulation would induce extension. The second objective was to compare empirical measures to the predictions of a computational gait model, and to assess the sensitivity of model predictions to variations in gastrocnemius geometry.

#### 2. Materials and methods

#### 2.1. Subjects and experimental overview

Twenty healthy young adults (13 females, mean  $\pm$  standard deviation: age 24.4  $\pm$  3.0 yr, mass 66.4  $\pm$  10.5 kg, height 1.71  $\pm$  0.10 m) with normal gait were recruited for participation. Subjects were excluded if they had a history of gastrocnemius/soleus muscle strain, bone fracture or knee injury within the past 24 months, prior surgery of the lower extremity, a latex allergy, or the inability to walk on a treadmill for 30 min. This protocol was approved by the University of Wisconsin-Madison Health Sciences Institutional Review Board. Each participant provided appropriate written informed consent prior to testing.

This study investigated the effect of electrically stimulating the gastrocnemius or soleus at different stages of the gait cycle. For each trial, muscle (gastrocnemius or soleus) and stimulation timings (20% or 30% of the gait cycle) were randomized. Trials were 90 s in duration, and included approximately 10 stimulations per trial. These results were then compared to computational modeling results of increased muscle activity at similar times in the gait cycle.

#### 2.2. Muscle stimulation protocol

Stimulating surface electrodes were placed on the mid-muscle belly of the medial gastrocnemius and the distal lateral soleus (Fig. 1a) of the right leg. Stimulating pulses were generated by a dual-channel, current-controlled stimulator (Grass S88, Astro-Med, Inc., West Warwick, RI). Subject-specific placement of the electrodes was determined by moving a surface electrode to the point where a maximum twitch response was observed in the muscle of interest, without visible contractions of other muscles. The stimulating current ( $\leq$  50 mA) was adjusted for each participant to a level that induced contractions and joint movement in a relaxed posture. During walking trials, stimulation was introduced to either the gastrocnemius or the soleus starting at 20% (referred to as mid-stance) or 30% (terminal stance) of the gait cycle (Perry, 1992). We note that we have previously shown these stimulation timings correspond well to the normal activation during walking (Francis et al., 2013).

Trials were randomized and stimulation occurred within a trial at random intervals, every 5–10 strides. The stimulation pulse train consisted of four pulses delivered over 90 ms. Timing of stimulation was controlled by a custom LabView (National Instruments, Austin, TX) program that identified heel strike events from the vertical ground reactions. Gait cycle duration was estimated by a moving average of 3 successive heel strikes of the same limb. Muscle stimulation was then introduced starting at either 20% or 30% of random gait cycles. At least five non-stimulated cycles occurred after each stimulated cycle to allow the transient effects to diminish.

#### 2.3. Motion analysis

Forty four reflective surface markers were used to track and record 3D wholebody kinematics using an 8 camera motion capture system (Motion Analysis, Santa Rosa, CA). Twenty five of these markers were placed on anatomical landmarks, and 14 were placed on rigid plates strapped to the shanks and thighs. Subjects were instructed to walk at a self-selected pace ( $1.14 \pm 0.10$  m/s) on a split-belt instrumented treadmill (Bertec Corp., Columbus, OH) (Fig. 1b). Kinematic data was



**Fig. 1.** Experimental setup. (a) Placement of the EMG and stimulation (Stim) electrodes. MGA=Medial Gastrocnemius, LGA=Lateral Gastrocnemius, SOL=Soleus (b) The instrumented treadmill provided ground reaction forces (GRF) that were recorded through data acquisition (DAQ). A LabView program continuously monitored heel strike events, and sent a signal to a muscle stimulator to initiate a pulse train (Stim) at the appropriate time in a random gait cycle. The stimulation occurred in either the gastrocnemius or the soleus.



**Fig. 2.** Muscle activity during the stimulation time period was determined by integrating the activity denoted by the shaded regions. Stimulation pulses (Stim), as well as muscle activity in the medial gastrocnemius (MGA), lateral gastrocnemius (LGA), soleus (SOL), tibialis anterior (TA), vastus medialis (VM), and medial hamstrings (MH), are all shown.

recorded at 100 Hz, and low-pass filtered at 6 Hz. The marker data were then used to compute the pelvis, hip, knee, and ankle joint angles throughout the trials. A whole body musculoskeletal model was scaled to align with anatomical marker positions of each subject in a standing posture. The base segment was the pelvis, with 6 degrees of freedom (dof). The trunk was attached to the pelvis with a ballin-socket joint with 3 dof. Each upper limb was modeled with 5 dof (shoulder adduction, flexion, rotation, elbow flexion, and supination/pronation). The hip was modeled as a ball-in-socket with 3 dof, and the ankle was allowed to plantar/ dorsiflex with 1 dof. The 1 dof knee had translations and nonsagittal rotations defined as functions of knee flexion (Arnold et al., 2010). Hip joint center was calculated based on a functional calibration (Leardini et al., 1999). Equations of motion were derived using SIMM/Dynamics Pipeline (Musculographics Inc. Santa Rosa, CA) and SD/FAST (Parametric Technology Corporation, Needham, MA). The inverse kinematics problem was solved using numerical optimization to minimize the sum of weighted squared errors between measured and model marker positions (Delp et al., 2007)

Pre-amplified, single differential electromyographic (EMG) electrodes (DE-2.1, Delsys Inc., Boston, MA) were placed over the medial and lateral gastrocnemius, soleus, tibialis anterior, vastus medialis, and medial hamstrings. The EMG activity, the stimulator's signal, and ground reaction forces were all sampled at 2000 Hz. During post-processing, EMG activities during cycles before and during stimulation were rectified. To evaluate spill-over, we quantified induced muscle activities by integrating rectified EMG between stimulus pulses, after a brief time period to allow for the direct stimulation pulse effects to dissipate on each electrode

(Hernandez et al., 2010) (Fig. 2). Potential reflex activity was then evaluated by comparing muscle activities in a post-stimulation window (150–300 ms after the stimulation onset) to baseline activity levels from non-stimulated strides.

#### 2.4. Statistical method used to detect induced perturbation of movement

Trials were binned according to actual time of stimulation, as 20% (between 15% and 25%) or 30% (25–35%) of the gait cycle. The absolute joint angles at the pelvis, hip, knee, and ankle were determined at points every 50 ms for a total of 200 ms after stimulation. We note that 50 ms is approximately equal to 5% of the gait cycle. The stimulated strides were compared to a control stride, comprising an average of the single non-stimulated strides immediately preceding stimulation. Repeated measures ANOVA was used to assess the effect of stimulation on the induced sagittal pelvis, hip, knee, and ankle angles at 50 ms intervals after stimulation onset. Post-hoc analyses (Tukey's Honest Significance Test) were performed to assess statistical significance, which was set at p < 0.05.

#### 2.5. Forward dynamic simulations of gait

A whole body, 3D musculoskeletal model was used to create a simulation of nominal gait. The model had the same degrees of freedom as those of the inverse dynamics model. To enable muscle-actuated simulations, we incorporated geometric descriptions of 92 Hill-type musculotendon units crossing the low back, hip, knee, and ankle (Arnold et al., 2010). The kinematics and kinetics during overground walking from a healthy young adult (height 1.7 m, mass 60 kg) were used to create the simulation. Residual elimination analysis first removed inconsistencies between ground reaction forces and kinematic measures (Remy and Thelen, 2009). Then, a computed muscle control algorithm determined muscle activations needed to track the joint angles over time (Thelen and Anderson, 2006). Minimization of the muscle volume-weighted sum of squared muscle activations resolved muscle redundancy.

After creation of a nominal gait simulation, perturbations were introduced to emulate the experimental stimulations. Specifically, the excitation of the medial gastrocnemius or soleus was increased for 100 ms starting at either 20% or 30% of the gait cycle and the simulation re-run. To allow for changes in foot-floor contact, dampers were placed between the perturbed and simulated foot positions. Both translational and rotational dampers were used to emulate a fixed foot during foot flat (10–35% of gait cycle), allowing forward progression of center of pressure for early stimulation as observed experimentally by Francis et al. (2013). A translational damper at the center of pressure was used to emulate a point constraint after heel off ( > 35% of gait cycle), which restricted progression of center of pressure (Francis et al. 2013). Kinematic changes were assessed by comparing joint angle trajectories to those seen in the nominal simulation, as was done experimentally. The excitation perturbation magnitudes (0.01 for soleus, 0.02 for gastrocnemius) were adjusted such that the change in knee motion approximately matched the experimental results.

Simulations were repeated after varying the insertion of the medial gastrocnemius by  $\pm 5$  mm and  $\pm 10$  mm in the anterior–posterior direction (Sheehan, 2008). With each variation, the perturbed gait simulations were re-run and the impact on induced motion determined as described above. Gastrocnemius geometry was characterized by its knee-to-ankle moment arm ratio ( $r_{\rm K}/r_{\rm A}$ ) in an upright, neutral position. The nominal configuration had  $r_{\rm K}/r_{\rm A}$ =0.34. Anterior translation led to increased  $r_{\rm K}/r_{\rm A}$  largely due to a decreased ankle moment arm.

#### 3. Results

#### 3.1. Experimental results

The gastrocnemius induced hip and knee flexion when stimulated during mid-stance, with the change in joint angles becoming statistically significant 150 ms after the stimulation onset (both p < 0.01) (Fig. 3a). Ankle dorsiflexion and posterior pelvic tilt were also induced at 200 ms after stimulation onset (both p < 0.01). The induced motion averaged across all subjects was 1.5° of hip flexion, 3.2° of knee flexion, 0.7° of dorsiflexion, and 0.4° of posterior pelvic tilt. Terminal stance gastrocnemius stimulation also induced hip and knee flexion (p < 0.01), with mean changes of 0.9° of hip flexion hip and 1.9° of knee flexion. The pelvis again tilted more posteriorly (p < 0.01).

Mid-stance soleus stimulation induced a significant shift toward ankle plantarflexion and knee extension (mean change of  $0.6^{\circ}$  (p < 0.01) and  $1.0^{\circ}$  degrees (p < 0.05) at 200 ms respectively) (Fig. 3b). Pelvic tilt was  $0.4^{\circ}$  more anterior after soleus stimulation

(p < 0.01). After mid-stance soleus stimulation the hip moved toward extension ( $0.3^{\circ}$ ), yet this change did not prove significant. Terminal stance soleus activity induced small initial shifts toward hip extension (average of  $0.3^{\circ}$ ), knee extension ( $0.6^{\circ}$ ), and ankle plantarflexion ( $0.3^{\circ}$ ), though none were significant. Pelvic tilt was again shifted anteriorly, averaging  $0.3^{\circ}$  after 200 ms following stimulation at 30% of the gait cycle (p < 0.01).

The EMG data indicate that a majority of the induced electrical activity occurred in the targeted muscle (Fig. 4). The induced gastrocnemius activity was > 6 times larger than that of any other muscle when it was stimulated, and the induced soleus activity was > 3 times larger. Gastrocnemius and soleus activities in the post-stimulation period (150–300 ms) were only 25% and 22% of their stimulated activities, respectively.

#### 3.2. Computational results

Forward dynamics model predictions of induced hip and knee motion were consistent with experimental results, while induced pelvis and ankle motion exhibited some differences (Fig. 5). Gastrocnemius perturbation at both time points led to a predicted increase in hip flexion and knee flexion, while soleus perturbation led to an increase in hip extension, knee extension, and plantarflexion. Model predictions of the relative magnitude of induced motion at the different joints were also similar to the experimental finding, with the model suggesting that both muscles induce greater motion at the knee than at the pelvis, hip, or ankle. Unlike our experimental results, the nominal model predicted slight plantarflexion with stimulation of the gastrocnemius. Additionally the models predicted that gastrocnemius and soleus stimulation at 20% of the gait cycle would have similar effects at the pelvis, with both inducing slight anterior pelvic tilt. Model predictions of gastrocnemius muscle function were sensitive to geometric changes. Anterior translation of the insertion  $(r_{\rm K}/r_{\rm A}$  greater than the nominal case, i.e. > 0.34) increased the magnitude of the muscle's induced motion at the knee and hip. Moreover anterior translation by 10 mm  $(r_{\rm K}/r_{\rm A}=0.44)$  led the gastrocnemius to induce dorsiflexion in the 20% condition, more closely aligning with experimental findings.

#### 4. Discussion

Our results show distinct roles of the gastrocnemius and soleus during the stance phase of gait (Fig. 6). Consistent with our hypothesis, mid-stance gastrocnemius activity did induce hip and knee flexion and ankle dorsiflexion in our experiments while mid-stance soleus activity induced ankle plantarflexion and knee extension. The empirical results suggest that the muscle function does evolve throughout stance, with later gastrocnemius stimulation having less effect on pelvic and ankle motion than earlier stimulation. Taken together these results support the concept of unique biomechanical function for the plantarflexors during gait, and that this function varies with posture.

Prior computational modeling studies have raised the possibility of the gastrocnemius and soleus inducing differing effects on joint and whole body motion (Francis et al., 2013; Liu et al., 2008; Neptune et al., 2004., 2008), but conclusions have differed. Pandy et al. (2010) used induced acceleration analysis of a 3D gait model and found that the gastrocnemius and soleus induced disparate actions at the hip (gastrocnemius flexion, soleus extension), but the same action at the knee (extension) and ankle (plantarflexion). In contrast, earlier gait models found that the gastrocnemius induced knee flexion (Kimmel and Schwartz, 2006; Neptune et al., 2001). While challenging to identify the exact cause of differences in conclusions, it is possible that



Fig. 3. Changes in hip, knee, and ankle angles induced by (a) gastrocnemius and (b) soleus stimulation (\*p < 0.05). Shaded region represents ± one standard deviation.



**Fig. 4.** Normalized average rectified EMG activity of several muscles during the experimental protocol. Data during trials with stimulation of the medial gastrocnemius and the soleus is shown during time of induced activity (0–150 ms), of potential reflex activity (150–300 ms of stimulated strides), and similar time periods during baseline (non-stimulated) strides. For each subject the data in each category was normalized by the total rectified EMG activity of all muscles, such that the sum of all muscles was one. To maintain the relative relationship between total EMG activity in each category, each category was again normalized by the ratio of total EMG activity in the induced period to the total activity in the period of interest. Results suggest induced muscle stimulation is primarily in the muscle of interest, with little effect in the reflex period.

underlying modeling assumptions may be a factor. It is recognized that induced acceleration analysis is sensitive to the degrees of freedom included in the model (Chen, 2006), footfloor modeling assumptions (Dorn et al., 2012; Hamner et al., 2013) and biarticular muscle geometry (Zajac and Gordon, 1989). Further, it is relevant to recognize potential differences between induced acceleration and induced position analysis. Induced accelerations characterize the instantaneous capacity of a muscle to generate accelerations, which is dependent on the current posture but independent of other muscles. In contrast our forward dynamics simulations measure changes in joint position, which evolve over time and therefore include the effect of biomechanical and neural interactions (Anderson et al., 2004; Hernandez et al., 2010).

Previous experimental work on plantarflexor function is consistent with our findings. Stewart et al. (2007) previously used a similar muscle stimulation protocol to elucidate roles of the gastrocnemius and soleus at the knee and ankle. Their observations, like ours, showed the muscles inducing directly opposite motions at these joints. However, their stimulation periods were maintained from foot-flat to toe-off such that they were not able to delineate evolving roles for these muscles over stance.

We note that our computational results do not match our experimental results regarding the gastrocnemius' impact on pelvis and ankle motion. Experimentally, we observed induced dorsiflexion in response to mid-stance gastrocnemius stimulation. The potential for the biarticular gastrocnemius to induce ankle dorsiflexion was first posited by Zajac and Gordon (1989). The non-intuitive action occurs when the gastrocnemius' knee flexor moment induces ankle dorsiflexion of greater magnitude than the motion induced by the muscle's plantarflexor moment. Our nominal model predicted initial effect of gastrocnemius stimulation toward plantarflexion. However of note is the strong dependence of gastrocnemius muscle function on the ratio of its moment arms at the knee and ankle. When in a relatively upright posture, Zajac and Gordon (1989) suggest that gastrocnemius would non-intuitively induce ankle dorsiflexion when the gastrocnemius' knee-to-ankle moment arm ratio exceeds approximately



**Fig. 5.** Computational modeling results for perturbation of the gastrocnemius (2% excitation) and soleus (SOL -1% excitation) at 20% and 30% of the gait cycle. Solid lines are the nominal perturbation, and dashed lines represent when the gastrocnemius insertion is translated anterior or posterior by 5 mm and 10 mm ( $r_{\rm K}/r_{\rm A}$ =knee-to-ankle moment arm ratio). Experimental results are presented with a circle, with error bars representing one standard deviation.

0.5. We altered the gastrocnemius knee-to-ankle moment arm ratio of the model by translating the muscle-tendon's distal insertion, and similarly predicted shifts toward dorsiflexion with larger moment arm ratios. In fact, when the insertion was translated 10 mm anteriorly resulting in a larger moment arm ratio, the induced motion was toward dorsiflexion (Fig. 5). The variabilities in our model predictions arising from manipulations of the gastrocnemius moment arm are of comparable magnitude to the experimental range, suggesting that anatomical differences may be a contributing factor to the variability observed across subjects. Model predictions of pelvic motion induced by the plantarflexors were not entirely consistent with the measurements. This difference may be attributable to the simplified representation of spinal motion (i.e. a single low-back joint) which could directly affect predictions of pelvic movement.

A few assumptions were made in the analysis of this data. We assumed that the stimulus induced motion representative of the muscle's function. To minimize the effect of reflex activity on our results, we considered only the first 200 ms after stimulation onset to assess muscle function. Monosynaptic reflexes could be induced as early as 50 ms after the stimulus (Burne and Lippold, 1996), but resulting motion at the joint level would not arise for some time due to neuromuscular and musculoskeletal delays. The observation that our significant findings are consistent with the initial direction of the perturbed motion suggests that our results likely reflect the direct muscle stimulation. Another potential concern is the use of surface stimulating electrodes creating spillover activity in other muscles. However, given that our quantification of muscle activity indicates primary induction in the muscle of interest and the perturbed motions of each muscle were opposite, we feel confident that our results represent differential stimulation.

It is important to note that the changes observed as a result of these stimulations occurred from 15% to 20% of the gait cycle later than delivery. Therefore, 20% and 30% stimulation led to observed changes at, respectively, 35–40% and 45–50% of the gait cycle. We have shown that stimulations at these timings match well with normal activation of these muscles during walking (Francis et al.,

2013), so we believe our results are reflective of the muscles' functions during gait.

While understanding normal muscle function in gait is important, our ultimate goal is to gain insights into pathological conditions. Equinus gait is a common abnormality in cerebral palsy, caused by contractures or spasticity of the plantarflexors. Our experimental results indicate that the soleus is primarily responsible for inducing plantarflexion during stance, while the gastrocnemius tends to induce dorsiflexion. This suggests that the excessive plantarflexion occurring in stance phase of dynamic equinus gait may be attributable to inappropriate soleus activity, as previously suggested (Svehlik et al., 2010). However, we note that our observations are limited to activations occurring at normal time periods in healthy adult gait. Future work will consider pathological variations in muscle activation timing, posture, and musculoskeletal geometry that can all affect function.

This work has been the first to describe gastrocnemius and soleus function in a well controlled paradigm with the ability to delineate variations in muscle function through the gait cycle. Further, we used computational work to show that our results are plausible and that variations in gastrocnemius geometry may explain some of the subject-specific variability in function. The experimental results of this study suggest that, during the stance phase of normal gait, the gastrocnemius can induce hip flexion, knee flexion and ankle dorsiflexion, while the soleus displays opposite functions as a knee extensor and an ankle plantarflexor. These findings highlight the need to understand more than just a muscle's anatomical classification when interpreting a muscle's function in dynamic whole body tasks such as walking.

#### **Conflict of interest statement**

The authors have no conflicts of interest to disclose.



**Fig. 6.** Progression of the difference in stance limb posture for stimulation of the gastrocnemius (red) and soleus (yellow) compared to the nominal stride (white, 20% stimulation, legs represent 20%, 30%, and 40% of the gait cycle). Magnitude of change is amplified by a factor of 5. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

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