



# Empirical assessment of dynamic hamstring function during human walking



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## ABSTRACT

The hamstrings are often associated with the development of crouch gait, a fatiguing form of walking characterized by excessive hip flexion, knee flexion and ankle dorsiflexion during stance. However, recent studies have called into question whether abnormally active hamstrings induce the limb to move into a crouch posture. The purpose of this study was to directly measure the influence of the hamstrings on limb posture during stance. Nineteen healthy young adults walked on an instrumented treadmill at their preferred speed. A 90 ms pulse train was used to stimulate the medial hamstrings during either terminal swing or loading response of random gait cycles. Induced motion was defined as the difference in joint angle trajectories between stimulated and non-stimulated strides. A dynamic musculoskeletal simulation of normal gait was generated and similarly perturbed by increasing hamstring excitation. The experiments show that hamstring stimulation induced a significant increase in posterior pelvic tilt, knee flexion and ankle dorsiflexion during stance, while having relatively less influence on the hip angular trajectory. The induced motion patterns were similar whether the hamstrings were stimulated during late swing or early stance, and were generally consistent with the direction of induced motion predicted by gait simulation models. Hence, we conclude that overactive hamstrings have the potential to induce the limb to move toward a crouch gait posture.

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## 1. Introduction

The hamstrings have long been associated with the development of crouch gait, a fatiguing form of walking characterized by excessive hip flexion, knee flexion and ankle dorsiflexion during stance (Rodda and Graham, 2001; Sutherland and Davids, 1993). However, clinical treatment of the hamstrings (e.g. surgical lengthening) does not consistently correct crouch gait in children, with some individuals demonstrating much improved hip and knee extension while others exhibiting little change (DeLuca et al., 1998; Novacheck et al., 2002). In addition, excessive anterior pelvic tilt can be an unintended result of hamstring lengthening (Chang et al., 2004; Hoffinger et al., 1993). These observations have led to development of dynamic gait models to rigorously investigate muscular contributions to limb motion during gait (Arnold et al., 2005, 2007; Jonkers et al., 2003; Kimmel and Schwartz, 2006; Neptune et al., 2004; Piazza and Delp, 1996). Interestingly, these models suggest that the hamstrings have greater capacity to induce

hip extension than knee flexion during stance, and may even induce knee extension (Arnold et al., 2005; Jonkers et al., 2003; Kimmel and Schwartz, 2006). This potential non-intuitive function at the knee is a result of multi-body dynamic effects, which allow for biarticular muscles to induce motion opposite to that assumed based on traditional anatomical classifications (Hernandez et al., 2010; Zajac and Gordon, 1989). However, there is a lack of empirical data to substantiate model predictions of muscle function (Piazza, 2006), which make it challenging to translate modeling results to clinical diagnosis and treatment of crouch.

Electrical stimulation experiments can be used to selectively activate individual muscles and thereby assess the influence that muscle has on skeletal movement (Hernandez et al., 2008, 2010; Hunter et al., 2009; Stewart et al., 2007, 2008). Stewart et al. (2008) used such a paradigm to show that the biarticular hamstrings induce knee flexion in upright postures, but tend to induce knee extension in crouch postures. Hunter et al. (2009) measured induced limb accelerations when subjects were held in swing limb postures by a robotic exoskeleton, and found that the hamstrings may have greater potential to induce knee flexion than hip motion. However, these assessments were done in static postures, which does not account for the time-varying limb configurations and foot–floor interactions that occur in gait. Our

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group has established an electrical stimulation protocol for the direct measurement of dynamic muscle function during walking and recently used it to investigate the motion induced by the rectus femoris (Hernandez et al., 2010). The purpose of this study was to empirically assess the influence of hamstring activity on stance limb motion during the terminal swing-to-early stance transition in normal gait. Based on prior models (Arnold et al., 2007; Kimmel and Schwartz, 2006), we hypothesized that the hamstrings would induce hip extension when activated in terminal swing or early stance, while having less influence on knee motion. We additionally explored the effects of hamstring stimulation on pelvic and ankle motion, to better understand how surgical treatment of the hamstrings could alter motion at joints not crossed by the muscle. Finally, we compared our experimental measures of muscle function to dynamic gait simulations, thereby providing a direct assessment of model predictions.

## 2. Methods

### 2.1. Experimental methodology overview

Nineteen healthy young adults (age =  $26.8 \pm 5.4$  yr, mass =  $69.5 \pm 11.3$  kg, height =  $1.73 \pm 0.10$  m) participated in this study, which was approved by the University of Wisconsin's Health Science's Internal Review Board. Subjects performed 90 s walking trials on a split-belt instrumented treadmill (Bertec Corp., Columbus, OH) at their preferred gait speed ( $1.15 \pm 0.12$  m/s).

### 2.2. Electrical stimulation synchronized to the gait cycle

A current-controlled stimulator (Grass S88, Astro-Med Inc., West Warwick, RI) was used to stimulate the hamstrings via surface electrodes (Fig. 1). For each subject, we first located the motor point of the MH by moving surface stimulating electrodes over the skin until a maximum twitch response was observed. The desired electrode positions on the skin were prepared by shaving and cleaning the skin with alcohol pads prior to the placement of self-adhering surface electrodes ( $1.25 \times 1.5$  inches). Ninety ms current pulse trains (four  $300 \mu\text{s}$  pulses at 33 Hz) were delivered to the MH at selected times on random gait cycles (Fig. 1). The stimulating current ( $\leq 50$  mA) was set for each subject to a level that was tolerable but could also elicit hip and/or knee motion in a relaxed limb. Stimulation timing was controlled using a custom LabView (National Instruments, Austin, TX) program that identified heel strikes by monitoring vertical ground reactions under each foot. Successive heel strikes were used to maintain a moving average estimate (over 3 strides) of the stride duration. The stimulator was triggered at either 90% (terminal swing) or 0% (loading response) of random gait cycles (Fig. 1). A minimum of five non-stimulated strides followed every stimulated stride.

### 2.3. Kinematics analysis

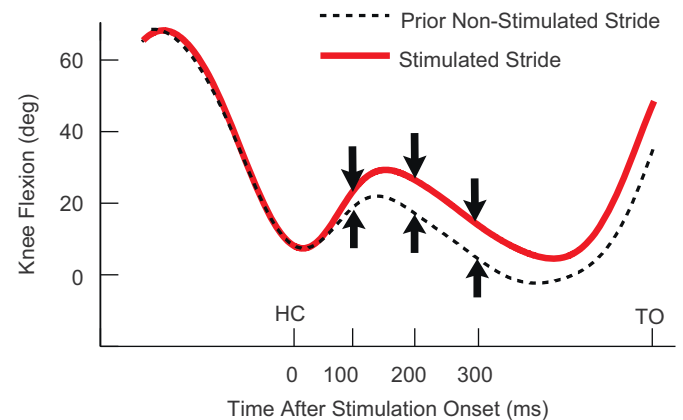
Whole body kinematics were recorded at 100 Hz using an 8-camera motion capture system (Motion Analysis, Santa Rosa, CA) to track 44 reflective markers (Fig. 1a). Twenty-five markers were placed over anatomical bony landmarks and additional tracking markers were attached to plates that were strapped tightly to

lower limb segments. All kinematic data were low-pass filtered at 6 Hz. Joint angles were computed using a whole body model that included 23 segments and 21 lower extremity degrees of freedom (dof) to represent the low back, hip, knee and ankle joints (Thelen and Anderson, 2006). The pelvis was the base segment with 6 dof. Each lower limb included a 3 dof ball-and-socket representation of the hip, a 2 dof ankle with non-intersecting talocrural and subtalar joints, and a 1 dof knee where translations and non-sagittal rotations were functions of knee flexion (Arnold et al., 2010). Segment lengths in the model were first scaled to each subject using anatomical marker positions measured in a standing upright trial. The hip joint center in the pelvic reference frame was then calibrated using a functional joint center identification routine (Piazza et al., 2004). We then used a global optimization inverse kinematics routine to compute pelvic position and joint angles that minimize the discrepancy between measured marker positions and corresponding markers fixed to the body segments (Lu and O'Connor, 1999).

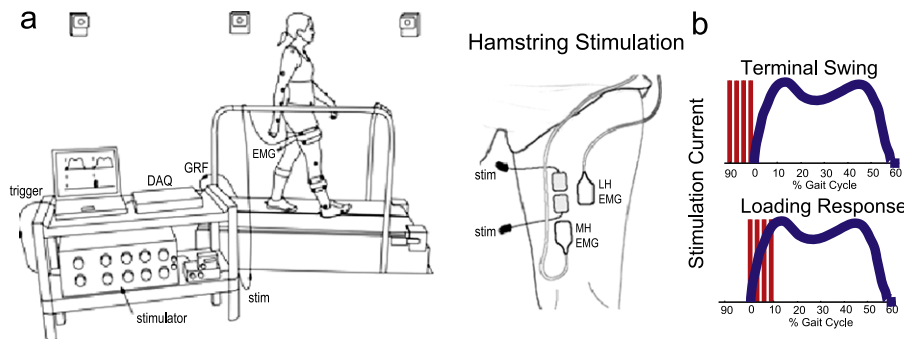
The stimulation-induced motion was determined by comparing joint kinematics in a stimulated stride to the preceding non-stimulated stride. We quantified the effect by determining the difference in joint angles between a stimulated stride and the preceding non-stimulated stride at 100 ms intervals following stimulation onset (Fig. 2). We also measured the difference in joint angles between the same time points in the two strides preceding the stimulation, so as to have a baseline reference of normal stride-to-stride variability. Joint angle differences were computed for each stimulated stride and the average difference was taken across all stimulated strides in a trial. Differences were then averaged across the two repeat trials for each condition. A two-way repeated measure analysis of variance was then used to determine the effect of stimulation onset time (90%, 0% gait cycle) and the presence of stimulation (baseline, stimulated) on the change in joint angles at 100, 200 and 300 ms following stimulation onset. Tukey's post-hoc tests were used to perform follow-up pair wise comparison. Significance level was set at  $p=0.05$ .

### 2.4. Muscle activity

Pre-amplified, single differential EMG electrodes (DE-2.1, DeSys Inc., Boston, MA) were placed on the rectus femoris, vastus lateralis, vastus medialis, medial



**Fig. 2.** Representative knee flexion curves with stimulation introduced at heel contact (HC). An increase in knee flexion persisted through stance and the subsequent toe off (TO). Induced motion was defined as the difference in joint kinematics between the stimulated stride and prior non-stimulated stride at discrete time points.



**Fig. 1.** (a) Subjects performed constant speed walking trials on a split-belt instrumented treadmill. Ground reaction forces (GRF) were monitored in real-time via a data acquisition unit (DAQ) and used to track heel strike events. In random strides, the stimulator was triggered at either 90% (terminal swing) or 0% (loading response) of the gait cycle. (b) A 90 ms long stimulation pulse train (four pulses delivered at 33 Hz) was delivered to the medial hamstrings via surface electrodes. Surface EMG was recorded from the medial and lateral hamstrings (MH, LH).

hamstrings, lateral hamstrings and adductor muscle group of the right limb (Fig. 1b). These EMG activities, the ground reaction forces from the treadmill and the stimulator's trigger signal were sampled synchronously at 2000 Hz. The EMG recordings were full wave rectified and used to evaluate stimulus spill-over and potential reflex activity as a consequence of the electrical stimulation. To evaluate spill-over, we quantified induced EMG activity between stimulus pulses (Fig. 6a) and determined the percentage of total activity seen in individual muscles. Reflex activity was 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.

### 2.5. Forward dynamic simulations of gait

A whole body musculoskeletal model was used to simulate normal walking mechanics. To perform simulations, we added 92 musculotendon actuators to the linked segment model, representing the major muscles acting about the low back, hip, knee and ankle joints (Arnold et al., 2010). The input to each muscle was an excitation that could vary between 0 and 1. Excitation-to-activation dynamics were represented via a bi-linear differential equation with activation and deactivation time constants of 10 and 40 ms, respectively (Thelen, 2003). A Hill-type musculotendon model was used to describe contraction dynamics (Thelen, 2003). We generated simulations that closely emulated kinematic and kinetic measures of a young adult (height = 1.7 m, mass = 60 kg) walking overground at 1.2 m/s. To do this, we first used a least squares forward dynamic algorithm to resolve dynamic inconsistencies between measured kinematics and ground reactions over a gait cycle (Remy and Thelen, 2009). A computed muscle control algorithm was then used to compute muscle excitations (Fig. 3) that drive the model to track these joint angle trajectories, with measured ground reactions applied directly on the feet (Thelen and Anderson, 2006). Muscle redundancy was resolved by using a static optimization routine to minimize the muscle volume weighted sum of squared muscle activations at each time step (Happee, 1994). Upper extremity kinematics were prescribed to track measured values. The simulation emulated pelvis tilt and ankle dorsiflexion angles that were within  $1^\circ$  of kinematic measures, and hip and knee flexion angles that were within  $2^\circ$  of kinematic measures.

After generating a nominal simulation, we then perturbed the medial hamstrings excitation patterns to emulate the experimental study. This involved increasing the semitendinosus or semimembranosus excitation pattern starting at either 90% or 0% of the gait cycle for a 100 ms period, and then re-running the simulation. Our initial simulations demonstrated that the direction of linear induced motion increase was independent of the magnitude of the change in excitation. Hence, the change in excitation was set to a value (0.1 units) that induced joint motions of comparable magnitude to that observed experimentally. Changes in the interactions between the stance-limb foot and the ground were accounted for by applying a translational damper between the foot and ground at the current center of pressure of the foot. Hence, the ground reaction forces could change in response to the perturbation. As in the experimental case, the change in sagittal pelvis, hip, knee and ankle angles were determined by comparing the kinematic trajectories between the nominal and perturbed simulations.

To better understand the influence of musculoskeletal geometry on muscle function, we varied the anterior–posterior position of the distal hamstring insertion by  $\pm 5$  mm. For each variation in geometry, we generated a gait

simulation using computed muscle control (Thelen and Anderson, 2006), and then altered the hamstring excitation pattern to predict the induced motion at the pelvis, hip, knee and ankle. Changing the hamstring insertion altered the knee flexor moment arm of the muscle while leaving the hip extensor moment arm relatively constant. To characterize this change in geometry, we computed the average hip extension and knee flexion moment arm over a 300 ms period after stimulation onset, and used this data to compute the hip extensor-to-knee flexor moment arm ratio of both the semimembranosus and semitendinosus muscles. The influence of this moment arm ratio on induced motion at the pelvis, hip, knee and ankle was determined.

## 3. Results

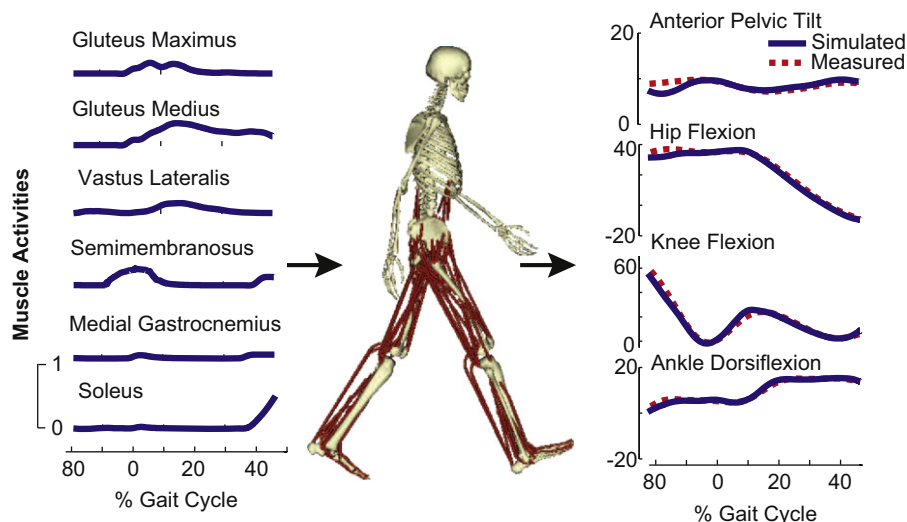
### 3.1. Measurements of hamstring function

Hamstring stimulation during either terminal swing or loading response induced significant ( $p < 0.05$ ) shifts toward posterior pelvic tilt and knee flexion at 200 ms after stimulation onset (Fig. 4). There was also a shift toward increased dorsiflexion, though the change did not reach significance until 300 ms after stimulation onset. There were no significant differences in hip flexion between baseline and stimulated strides at any of the time points considered. There were also no significant differences in induced motion measures at any of the joints between the terminal swing and loading response stimulations.

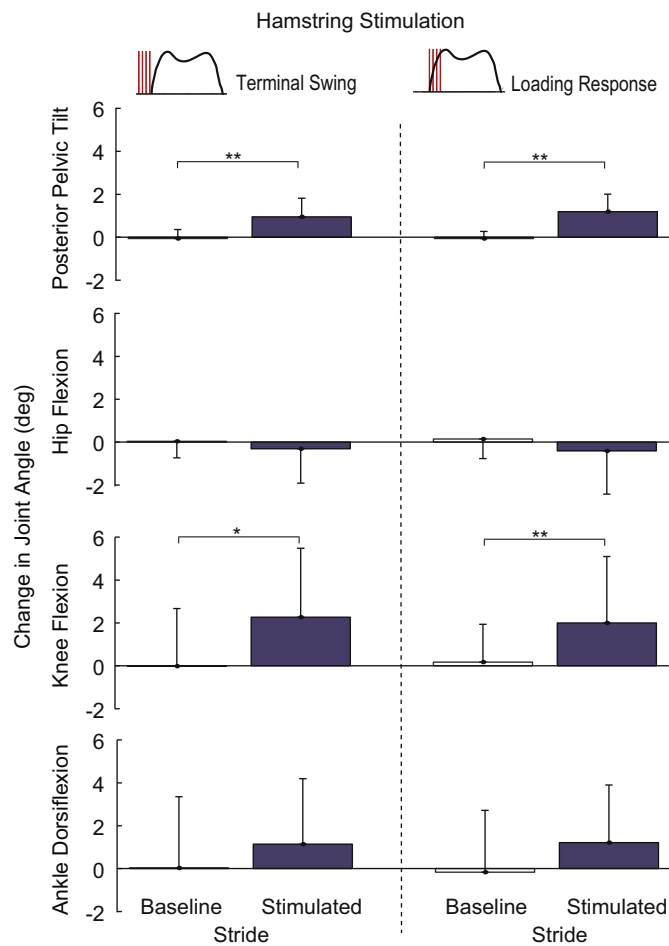
### 3.2. Model predictions of hamstring function

The model predicted that increased hamstring excitation in terminal swing would increase posterior pelvic tilt, hip extension and knee flexion at heel contact (Fig. 5). After heel contact, the model predicted that hip extension would cease to increase, while posterior tilt would continue progress. The same magnitude of hamstring excitation had less effect on the pelvis, hip and knee angle trajectories when stimulation onset occurred at heel strike. Model predictions of induced motion were generally consistent with empirical measures, and did not differ substantially between the semimembranosus and semitendinosus muscles.

Altering the anterior–posterior position of the distal hamstring insertion varied the hip extensor-to-knee flexor moment arm ratio from 1.1 in the anterior insertion case to 1.9 in the posterior insertion case. The moment arm ratio had a large influence on the induced motion patterns at the hip and knee. At the hip, all moment arm ratios resulted in a prediction of induced hip



**Fig. 3.** Computed muscle control was used to determine muscle excitations that drive a dynamic musculoskeletal model to emulate experimental gait kinematics. The gait simulation was subsequently perturbed with the medial hamstring excitations (semitendinosus, semimembranosus) increased at either 90% or 0% of the gait cycle, and the simulation re-run to predict induced changes in motion.



**Fig. 4.** Average ( $\pm 1$ SD.) change in pelvic, hip, knee and ankle angles induced 200 ms after hamstring stimulation was introduced. Baseline measures represent deviations in joint angles between two successive non-stimulated strides. Hamstring stimulation induced a significant ( $*p < 0.05$ ,  $**p < 0.005$ ) increase in posterior pelvic tilt and knee flexion when introduced during either terminal swing or loading response.

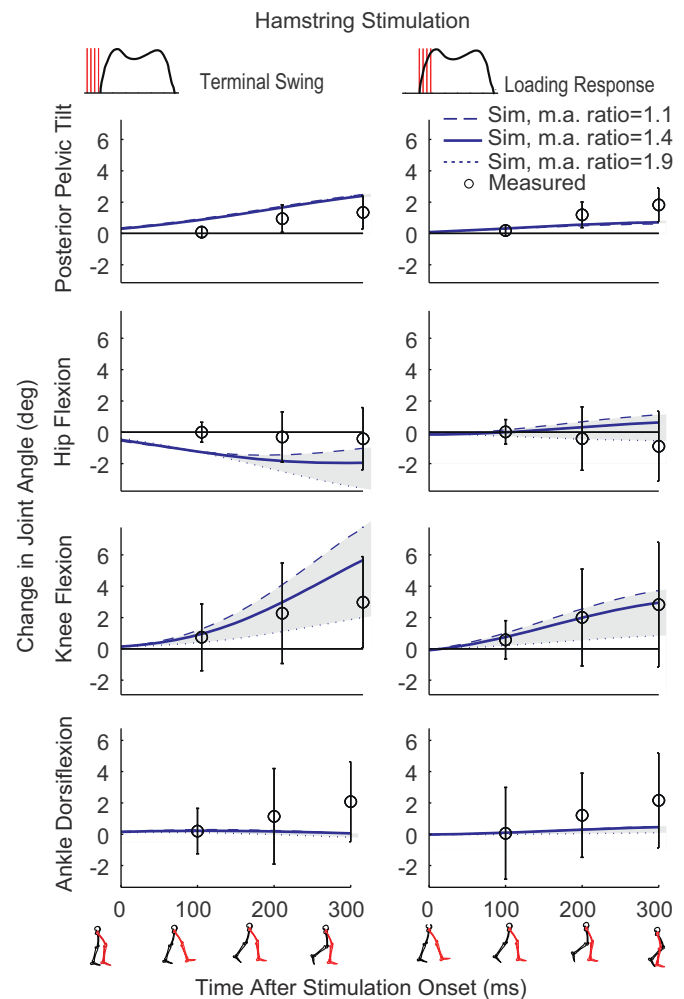
extension when stimulation was introduced in late swing. However when stimulation was introduced at heel contact, a large moment arm ratio induced hip extension while a small ratio resulted in hip flexion being induced. At the knee, the model predicted that the hamstrings induced flexion for all moment arm ratios, though the magnitude of knee flexion was greatly reduced for large moment arm ratios.

### 3.3. Muscle activities

The vast majority ( $\sim 65\%$  on average) of the induced EMG activity was measured in the medial hamstrings during the brief periods following stimulation pulses (Fig. 6). The average activity of any muscle in the post-stimulation (150–300 ms) period, during which reflexes might be expected, was less than 10% of the MH EMG average during a stimulated stride.

## 4. Discussion

This study represents the first use of muscle stimulation to directly assess dynamic hamstring function during gait. The empirical results show that hamstring stimulation in terminal swing or early stance induces a significant increase in posterior pelvic tilt and knee flexion during stance, while also tending to increase ankle

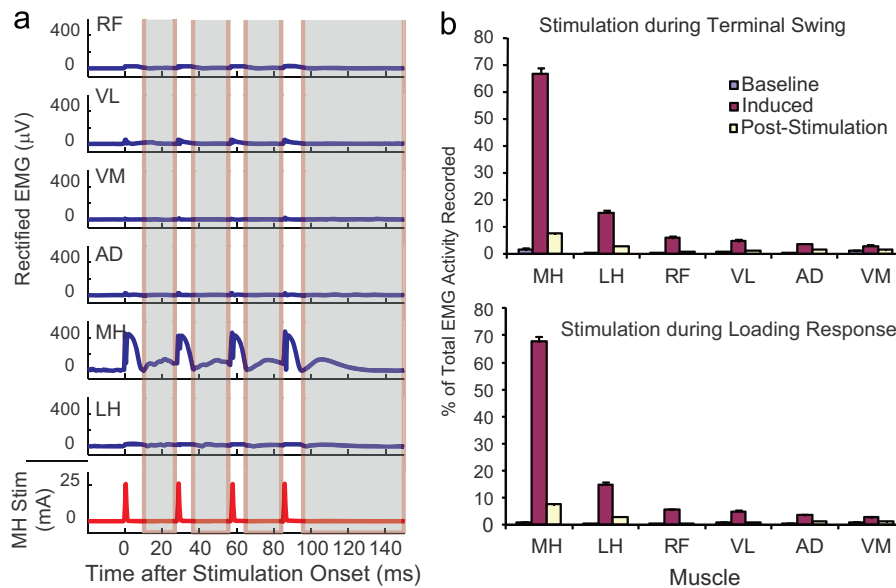


**Fig. 5.** Simulated (Sim) predictions of the change in pelvic, hip, knee and ankle angles induced by increasing semimembranosus excitation during terminal swing and loading response. Nominal model predictions (solid line) are generally within 1 standard deviation of experimental measures, and correctly predict the induced increases in posterior pelvic tilt and knee flexion. Shaded areas (bounded by dashed curves) represent the range of predictions obtained when simulations were performed with different hamstring insertion points, which varied the average hip extension-to-knee flexion moment arm (m.a.) ratio from 1.1 to 1.9. Induced hip and knee motion were quite sensitive to hamstring geometry, with a smaller m.a. ratio driving the limb more toward hip and knee flexion.

dorsiflexion. These induced motion measures were generally consistent with the direction of induced motion predicted by perturbations to a normative gait simulation. Interestingly, the simulations suggest that the same magnitude of hamstring stimulation introduced in terminal swing has greater influence on pelvis, hip and knee angle trajectories, than that of stimulation introduced in double support (Fig. 5).

Prior gait models suggest that substantial changes in muscle function can occur after heel contact, due to the distinct change in linked segment dynamics that occurs with double support (Frigo et al., 2010). However, our experimental data does not show evidence of sudden changes in muscle function at heel contact, suggesting that any changes in muscle function that arise with double support are more gradual. It is also important to note that we empirically investigated the relationship between muscle excitation and joint angles, which necessarily occurs sometime after stimulation and can reflect the influence of other muscles generating forces on the skeleton. In contrast, muscle function is often characterized by induced accelerations, which would represent the instantaneous relationship between an individual muscle





**Fig. 6.** (a) EMG measures demonstrate that the stimulation pulses induced the largest muscle activity (see shaded areas after stimulation pulse) in the medial hamstrings (MH). (b) Comparison of average ( $\pm$  1SD) EMG activity in non-stimulated baseline strides with the induced EMG activity seen between stimulation pulses (Induced, shaded areas in 6a) and in a 150–300 ms window (Post-Stimulation) following each pulse train. Over 60% of the induced EMG activity was present in the MH, with the lateral hamstrings (LH), rectus femoris (RF), vastus lateralis (VL), adductors (AD) and vastus medialis (VM) exhibiting much smaller induced activities. Post-stimulation (> 150 ms) EMG activities were considerably smaller than that induced directly by the stimulation pulses.

force and joint accelerations (Fregly and Zajac, 1996; Zajac and Gordon, 1989; Zajac et al., 2002). We note that observations at the position level are likely to be more consistent with qualitative assessments of muscle function based on clinical gait analysis.

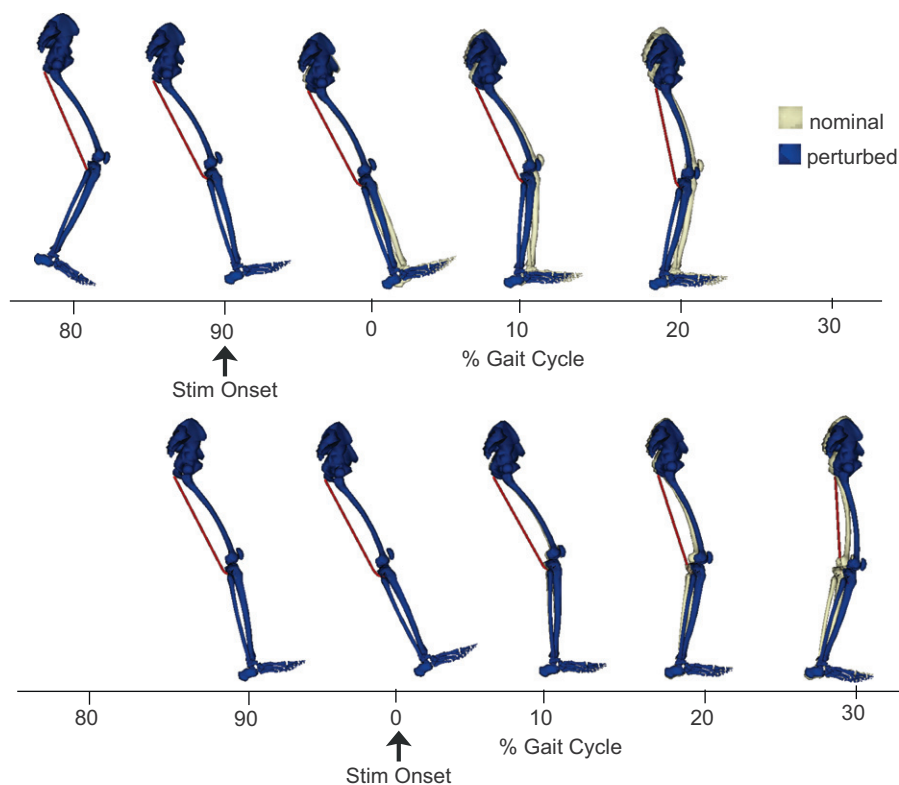
The empirical measures of induced motions were not completely consistent with our hypotheses, which were based on prior gait models. In particular, gait models have suggested that the hamstrings have greater influence on hip than knee motion in stance (Arnold et al., 2005; Jonkers et al., 2003; Kimmel and Schwartz, 2006). In contrast, our data show that both terminal swing and early stance hamstring stimulation induced knee flexion in stance, and that the magnitude of induced knee flexion exceeded the change in the hip angular trajectory (Fig. 4). Though the exact cause of discrepancies with models in the literature is uncertain, it is known that model-based predictions of muscle function are sensitive to variations in musculoskeletal geometry (Hernandez et al., 2008; Zajac and Gordon, 1989), foot–floor model assumptions (Dorn et al., 2012) and the number of degrees of freedom included in the linked segment model (Chen, 2006). Our gait simulations incorporated a recent description of lower limb musculoskeletal architecture (Arnold et al., 2010), and incorporated both three-dimensional effects and upper body dynamics.

We performed a sensitivity analysis to better understand how variations in hamstring geometry could affect our model predictions. In the nominal model, the average hip extension moment arms (5 cm for semimembranosus, 6 cm for the semitendinosus) and knee flexion moment arms (3.6 cm and 4.3 cm) during terminal swing/early stance were comparable to cadaveric measures at similar postures (Arnold et al., 2000; Buford et al., 1997). These nominal values give rise to a hip extension-to-knee flexion moment arm ratio of 1.4, which was then varied from 1.1 to 1.9 by altering the hamstring insertion. Our results show that induced hip and knee motion was strongly dependent on the moment arm ratio. A small ratio resulted in the non-intuitive prediction that the hamstrings can induce hip flexion when stimulated in stance. Further, the model suggests that moment arm ratios approaching 2 can result in the hamstrings inducing

knee extension in stance, which would reflect the influence of the hip extension moment on knee motion (Fig. 5). Thus it seems feasible that differences in muscle geometry could well underlie some of the variable predictions of muscle function that exist in the literature. For example, the discrepancy of this study with Kimmel and Schwartz (2006) may arise from the fact that they presented the induced acceleration capacity of the lateral hamstrings (biceps femoris long head), while we measured induced motion of the medial hamstrings (semitendinosus, semimembranosus). The lateral hamstrings have a considerably smaller knee flexion moment arm than that of the medial hamstrings (Arnold et al., 2000), such that it would have a greater hip extension-to-knee flexion moment arm ratio and thus greater potential to induce knee extension. These sensitivity results highlight the importance of documenting muscle geometry assumptions and performing parametric studies when using models to investigate muscle function.

The results of this study are potentially relevant for assessing the causes and treatment of crouch gait. Short and/or spastic hamstrings are considered potential contributors to crouch gait (Rodda and Graham, 2001; Sutherland and Davids, 1993). Either situation could give rise to abnormally high muscle forces when the hamstrings lengthen in terminal swing. Our data suggest that such excessive hamstring force could induce the lower limb to move toward a crouch gait posture in stance (Fig. 7). Hence, considerations of hamstring length and velocity seem warranted when trying to assess the causes of an individual's crouch pattern in gait analysis labs (Arnold et al., 2006). Our study also illustrates the strong influence of hamstring activity on posterior pelvic tilt. Hence, the clinical observation that hamstring lengthening can give rise to excessive anterior pelvic tilt (Chang et al., 2004; Hoffinger et al., 1993) likely reflects diminished capacity of the hamstrings to maintain posterior tilt.

There are limitations of the study that are important to consider. First, for simplicity and subject comfort, we used surface electrodes rather than fine wire electrodes to stimulate the hamstrings. While surface electrodes increase the potential for stimulus spillover to neighboring muscles, we did not see much



**Fig. 7.** Gait model predictions of lower limb posture arising from perturbations of the semitendinosus (100 ms duration, +50% excitation) at 90% and 0% of the gait cycle. Stimulation at 90% of the gait cycle increases knee flexion at heel contact, with subsequent increases in hip extension and ankle dorsiflexion. Stimulation at heel contact induces an increase in posterior pelvic tilt, knee flexion and ankle dorsiflexion at mid-stance.

evidence in our experimental EMG analysis (Fig. 6). Induced reflex activities are another potential concern, and become more relevant the longer the duration after the initial stimulation. However, we observed similar directions of induced motion at both 100 and 200 ms time points after stimulation onset (Fig. 5). Hence we are fairly confident that the induced motion represents the direct stimulation effect. Finally, we stimulated the hamstrings in normal gait only at a preferred speed. It is expected that muscle function will change with posture and speed in pathological gait (Steele et al., 2010), and further experimental work is needed to assess these sensitivities.

In conclusion, we have shown that hamstring stimulation during the swing-to-stance transition will induce an increase in posterior pelvic tilt, knee flexion and ankle dorsiflexion during stance, while having relatively less influence on hip motion. Hence, overactive hamstrings have the potential to induce the limb to move toward a crouch gait posture.

#### Conflict of interest

There are no conflicts of interest to disclose regarding this study.

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