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Measurement and simulation of joint motion induced via biarticular muscles during human walking

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Abstract

Gait impairments are often treated via targeted interventions performed on biarticular muscles such as the hamstrings, rectus femoris and gastrocnemius. Computational gait models suggest that biarticular muscle function can be non-intuitive, and at times inconsistent with assumptions that underlie current treatment strategies. However, the accuracy of gait model predictions has not yet been systematically established, which limits the influence of models on treatment. In this paper, we describe the use of electrical stimulation experiments to directly measure how biarticular muscles induce movement during walking. These measurements are compared to predictions made by forward dynamic simulations of gait. The results highlight the importance of carefully considering foot-floor contact and neuromusculoskeletal dynamics when using gait simulations to predict the influence of muscles on joint motion.

Keywords: Forward Dynamics, Gait, Functional Electrical Stimulation, Muscle Induced Motion

1. Introduction

Biarticular muscles are often implicated and treated in gait abnormalities. For example, Hamstring tightness and/or over-activation are considered contributors to crouch gait, which is characterized by excessive knee flexion in stance. Spasticity in the rectus femoris is cited as a cause of stiff-knee gait, i.e. diminished knee flexion during swing. Gastrocnemius contractures can contribute to crouch and/or equinus (toe-walking) gait. Treatments for these gait disorders can include botulinum toxin injections to diminish spasticity, tendon transfers to alter muscle moment arms or lengthening procedures to mitigate

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contractures [1]. However, clinical outcomes of such interventions remain variable, which in part reflects the challenges in identifying the specific causes of abnormal gait when planning treatment.

Zajac and Gordon [2] were the first to emphasize the importance of considering inter-segmental dynamics when assessing muscle function. In particular, they showed that biarticular muscles have the potential to induce joint motion in directions opposite to their anatomical classification. Since that time, dynamic simulations of walking have been used to estimate the contributions of muscles to joint motion, vertical support and forward propulsion [3-8]. However, there are a number of assumptions and limitations inherent in gait simulations which make it challenging to translate simulation results to clinical treatment. First of all, most models usually rely on generic descriptions of musculoskeletal geometry, which does not account for individual variations. Secondly, estimates of induced motion are sensitive to the specifics of the model formulation [9]. Finally, muscle function is often quantified by induced acceleration analysis, which represents the instantaneous capability of a muscle to induce motion. In contrast, clinicians typically observe movement at the position level, which will necessarily occur later, and reflect neural and biomechanical interactions within the system.

Muscle stimulation experiments have recently been performed to acquire direct *in vivo* measurements of muscle-induced motion [10-13]. For example, Stewart et al. [13] showed that the biarticular hamstrings can extend the knee in a crouched posture. We have shown that the rectus femoris can extend the hip in a swing phase limb posture [12]. However, these assessments were done in static postures, which does not account for the changing limb configuration and constraints that occur in gait. We have since established a well-controlled paradigm for measuring biarticular muscle function during walking, and used it to investigate rectus femoris, hamstring and gastrocnemius function. In this paper, we describe the approach and what we have observed in the hamstrings. Critical comparisons with gait model predictions provide insights into issues that should be addressed to improve model fidelity.

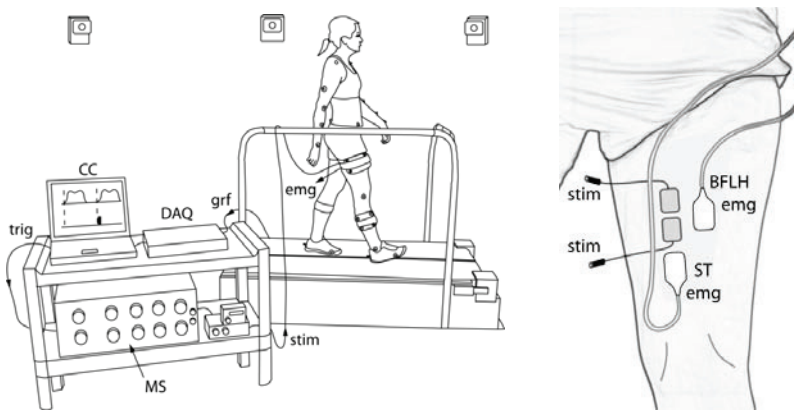
2. Methods

2.1. Empirical measures of muscle-induced motion

We place stimulating surface electrodes over the motor point of the muscle of interest. The motor point is located by moving surface stimulating electrodes over the skin until a maximum twitch response is observed. The electrode positions are then cleaned with alcohol and prepped with conductive gel prior to the placement of self-adhering surface electrodes (approximately 1.25”x 1.5”). A dual-channel, current-controlled stimulator (Grass S88, Astro-Med, Inc., West Warwick, RI) is used to stimulate the muscle. The current magnitude ($\leq$ 50 mA) is set for each subject to a level that is both tolerable and able to elicit joint motion in a relaxed posture. Subjects are then asked to perform a series of 90 second walking trials on a split-belt instrumented treadmill (Bertec Corp., Columbus, OH). A LabView (National Instruments, Austin, TX) program is used to detect heel strikes in real-time by monitoring the vertical ground reactions under each foot. The user sets the percentage of the gait cycle at which to stimulate a muscle. Based on the average stride period of the preceding 3 strides, the labview program then triggers the stimulator to send a pulse train to the muscle at the appropriate time. A 90 ms long stimulation pulse train (four 300-μs pulses delivered at 33 Hz) is used. A minimum of five non-stimulated strides will follow, with the stimulation again randomly introduced in one of the five subsequent strides. .

Whole body kinematics are recorded throughout using an 8-camera motion capture system (Motion Analysis, Santa Rosa, CA) to track 44 reflective markers placed on the upper and lower body segments (Fig. 1). All kinematic data are low-pass filtered at 6 Hz. Joint angles are then computed using inverse kinematics analysis on a whole body model that includes 23 segments and 21 lower extremity degrees of freedom (dof) to represent the low back, hip, knee and ankle joints [14]. The pelvis is the base segment

in the model with 6 dof. Each lower limb includes a 3-dof ball-and-socket representation of the hip, a 2-dof ankle with non-interesecting talocrural and subtalar joints, and a 1-dof knee where translations and non-sagittal rotations are functions of knee flexion [15]. Segment lengths are scaled to each subject using anatomical marker positions measured in a standing upright trial. We use a global optimization inverse kinematics routine to compute pelvic position and joint angles that minimize the discrepancy between measured and body-fixed marker positions at each time frame [16]. Lower extremity EMG activities, the ground reaction forces from the treadmill and the stimulator's trigger signal are sampled synchronously with the kinematics at 2000 Hz. The EMG data are used in a post-hoc fashion to evaluate potential stimulus spill-over and reflex activity in non-stimulated muscles.



(a) (b)

Fig. 1. (a) A custom labview program is used to monitor heel strikes in real time, and then to initiate stimulation of the medial hamstrings during either terminal swing or early stance of a random gait cycle; (b) Stimulating surface electrodes are used to induced hamstring contractions, while recording surface electrodes are used to monitor EMG activity.

The stimulation-induced motion is determined by comparing the joint angle trajectories in the stimulated strides to the corresponding ensemble average joint angle trajectories from non-stimulated strides. Induced motion is defined as the change in joint angles between non-stimulated and stimulated strides at 100 ms intervals after the stimulation onset, with statistically significant changes identified using paired t-tests. In this paper, we report our results on hamstring function in 19 healthy young adults (age 19-39). Stimulating surface electrodes were placed over the medial hamstrings. The muscle was stimulated at either 90% (terminal swing) or 0% (heel contact) of a random gait cycle, which corresponds to periods when the muscle is normally active. The protocol was approved by the University of Wisconsin Health Sciences Institutional Review Board.

2.2. Gait model predictions of muscle-induced motion

Scaled whole body models are also used to develop simulations of subject-specific walking dynamics. To perform simulations, we add 92 musculotendon actuators to the linked segment model, representing the major muscles acting about the low back, hip, knee and ankle joints [15]. The input to each muscle is an excitation that can vary between 0 and 1. Excitation-to-activation dynamics is represented by a bi-linear differential equation with activation and deactivation time constants of 10 and 40 ms, respectively. A Hill-type musculotendon model is used to describe contraction dynamics [17]. For each subject, we generate simulations of a normal, unperturbed gait cycle. To do this, we first used a least squares forward dynamics algorithm to resolve dynamic inconsistencies between measured kinematics and ground

reactions [18]. A computed muscle control algorithm is then used to compute muscle excitations that drive the model to track these joint angle trajectories, with measured ground reactions applied directly on the feet [14]. Muscle redundancy is resolved by using a static optimization routine to minimize the weighted sum of squared muscle activations [19] at each time step. Upper extremity kinematics are prescribed to track measured values. This approach has previously been shown to produce simulations of lower extremity joint angles that are within ~1° of measurements [14].

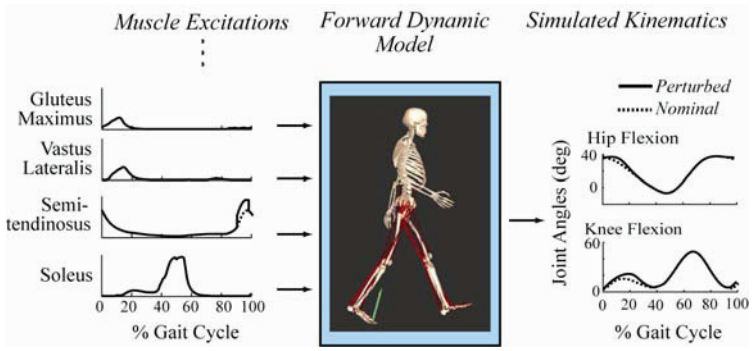


Fig. 2. A computed muscle control algorithm was used to determine muscle excitations that drove a whole body model to closely track measured gait kinematics and ground reactions. We then independently perturbed the semitendinosus or semimembranosus at either 90% or 0% of the gait, and re-generated the simulation to predict how movement would change as a result.

After generating a simulation, we then perturb individual muscle excitations to emulate the experimental study. For the case of the hamstrings, this involved perturbing the semitendinosus and semimembranosus excitation patterns at either 90% (terminal swing) or 0% (early stance) of the gait cycle. This is done by increasing the excitation level of each muscle by 0.1 units for a 100 ms period, and re-running the simulation (Fig. 2). Changes in the interactions between the stance-limb foot and the ground are accounted for by using a set of rotational and translational spring-damper units applied at the center of pressure [20]. Hence, the ground reaction forces and moments can change in response to the perturbation. As in the experimental case, the change in sagittal pelvis, hip, knee and ankle angles are determined by comparing the kinematic trajectories between the perturbed and nominal simulations.

3. Results and Discussion

3.1. Hamstring function during gait

Electrical stimulation of the hamstrings induced substantial changes in lower extremity posture during stance. Specifically, the stimulation significantly increased knee flexion (~3°) and hip extension (~1°) during stance. The hamstrings also influenced motion at the pelvis and ankle, with greater posterior pelvic tilt (~2°) and ankle dorsiflexion (~2°) observed in stance phase of stimulated strides. The directions and magnitudes of induced joint motion were similar whether the hamstrings were stimulated during late swing or starting at heel contact (Fig. 3). Induced motion measures peaked at ~300 ms after the stimulation onset, with trajectories tending to return to normative values thereafter.

3.2. Model predictions of hamstring function

The model predicted that hamstring activation in terminal swing would increase pelvic tilt, knee flexion and ankle dorsiflexion during stance. The simulated effect at the hip was small, with only a slight increase in hip extension. While there are differences in magnitude, the direction of predicted motion is

qualitatively similar to that seen in the empirical data. Interestingly, these predictions are not entirely consistent with prior gait modeling studies which suggest the hamstrings have greater influence at the hip, and may actually induce knee extension during stance [1-2]. An important distinction that could account for this difference is the fact that we investigated the relationship between muscle excitation and joint angles, which allowed hamstring activation in terminal swing to affect knee flexion in stance. In contrast, muscle function is often characterized by induced accelerations, which would represent the instantaneous relationship between muscle force and joint accelerations. Hence it is possible that the hamstrings may instantaneously induce knee extension in stance (e.g. see semimembranous in Fig. 3), but this effect may not be relevant at the joint angle level until sometime later.

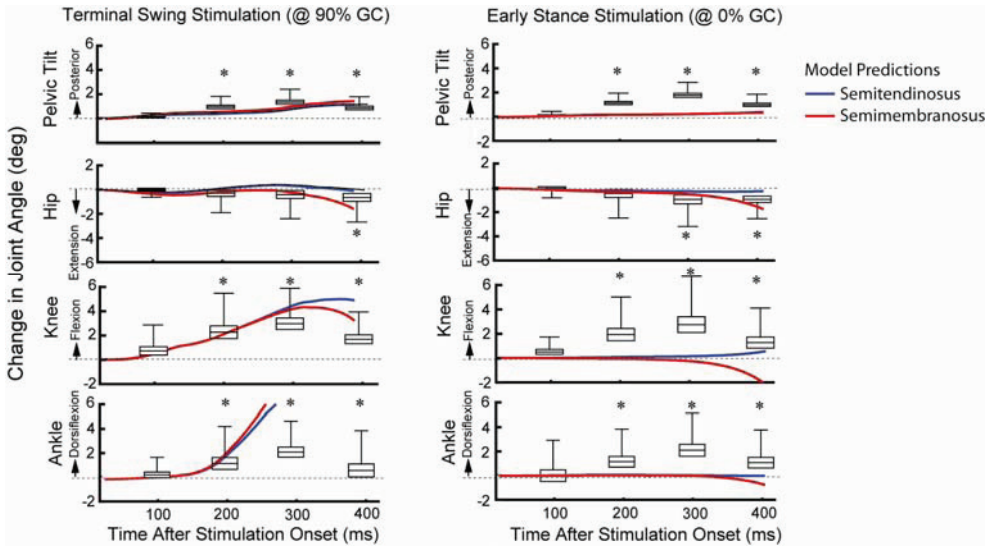


Fig. 3. Box plots shown represents the change in pelvic, hip, knee and ankle angles induced immediately after hamstring stimulation was introduced. Significant changes in joint angles ($p<0.05$) are denoted. The continuous curves represent the gait model predictions in response to perturbing the nominal semitendinosus and semimembranosus excitation patterns.

The model predicted that hamstring activation at heel contact would have relatively small effects on pelvic, hip, knee and ankle angles during stance. These predictions were not consistent with empirical measures, which show that hamstrings induced similar changes in motion whether the stimulation was introduced in late swing or early stance. This inconsistency highlights an important area for model refinement. In particular, it remains challenging to model foot-floor contact in a way that is appropriate for simulating muscle function. Different foot-floor contact models are used in the literature including stiff spring-damper formulations [6] and rigid constraints [4, 8]. A consequence of both model formulations is that double support phase effectively involves a closed loop constraint, which alters dynamics of the system. The result is the prediction that muscle function changes rather abruptly as the foot comes in and out of contact with the floor. Interestingly, we see no evidence in our data that hamstring-induced motion changed substantially after heel contact. Hence, there is a need for further consideration of how to appropriately model double support to reflect observed muscle function.

4. Conclusions

We have described the use of electrical stimulation protocols to empirically evaluate dynamic muscle function in gait. A rigorous comparison with gait model predictions shows the models to be reasonably

accurate in predicting the influence of swing phase activation on stance limb motion. However, the model was not robust at predicting the influence of hamstring activity on stance limb motion during double support. The results highlight the importance of re-evaluating foot-floor contact assumptions, and accounting for the considerable influence that dynamic processes have on induced motion. These rigorous comparisons between model predictions and experimental measures are essential for gait models to be used to identify the underlying causes of gait impairments and predict clinical outcomes.

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