

# Muscle force redistributes segmental power for body progression during walking

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## Abstract

The ankle plantar flexors were previously shown to support the body in single-leg stance to ensure its forward progression [J. Biomech. 34 (2001) 1387]. The uni- (SOL) and biarticular (GAS) plantar flexors accelerated the trunk and leg forward, respectively, with each opposing the effect of the other. Around mid-stance their net effect on the trunk and the leg was negligible, consistent with the body acting as an inverted pendulum. In late stance, their net effect was to accelerate the leg and trunk forward, consistent with an active push-off. Because other muscles are active in the beginning and end of stance, we hypothesized that their active concentric and eccentric force generation also supports the body and redistributes segmental power to enable body forward progression. Muscle-actuated forward dynamical simulations that emulated observed walking kinematics and kinetics of young adult subjects were analyzed to quantify muscle contributions to the vertical and horizontal ground reaction force, and to the acceleration and mechanical power of the leg and trunk. The eccentric uniarticular knee extensors (vasti, VAS) and concentric uniarticular hip extensors (gluteus maximus, GMAX) were found to provide critical support to the body in the beginning of stance, before the plantar flexors became active. VAS also decelerated the forward motion of both the trunk and the leg. Afterwards when VAS shortens in mid-stance, it delivered the power produced to accelerate the trunk and also redistributed segmental power to the trunk by continuing to decelerate the leg. When present, rectus femoris (RF) activity in the beginning of stance had a minimal effect. But in late stance the lengthening RF accelerated the knee and hip into extension, which opposed swing initiation. Though RF was lengthening, it still accelerated the trunk forward by decelerating the leg and redistributing the leg segmental power to the trunk, as SOL does though it is shortening instead of lengthening. Force developed from highly stretched passive hip structures and active force produced by the uniarticular hip flexors assisted GAS in swing initiation. Hamstrings (HAM) decelerated the leg in late swing while lengthening and accelerated the leg in the beginning of stance while shortening. We conclude that the uniarticular knee and hip extensor muscles are critical to body support in the beginning of stance and redistribution of segmental power by muscles throughout the gait cycle is critical to forward progression of the trunk and legs. © 2003 Elsevier B.V. All rights reserved.

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## 1. Introduction

The biomechanical mechanisms used by muscles to support and ensure forward progression of the body over the gait cycle are largely unknown because of the complex

musculoskeletal system dynamics. The complexity arises because the contribution by a muscle to the ground reaction force and intersegmental joint forces produced within the body cannot be measured. A muscle's contribution to the ground reaction force and the intersegmental joint forces can cause a redistribution of power among the segments by decelerating some segments (causing those segments to lose power) while accelerating others (causing those segments to gain power). Such a redistribution of power from decelerated to accelerated segments can occur even

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when a muscle produces no power (i.e. isometric activity) or absorbs power (i.e. eccentric activity with energy dissipated by the muscle or stored in musculotendinous elastic structures) [2]. Synergistic muscle activity is often required because one muscle can rarely redistribute power among the segments in a manner compatible with the task requirements (e.g. crank acceleration in pedaling [3]; trunk support by the ankle plantar flexors during walking [1]).

Identification of the complex segmental power exchanges performed by individual muscles can only be done using forward dynamics-based analyses derived from musculoskeletal models. Specifically, the mechanisms underlying body forward progression in walking can be found by solving the equations-of-motion that causally relates muscle and non-muscular (gravity, centripetal and Coriolis) forces to the ground reaction force and the acceleration of the body segments [1,4]. For example, a muscle's contribution to trunk forward acceleration can be found by computing the muscle-induced forward acceleration of the trunk segment center-of-mass [1,5].

Forward dynamics-based studies have shown that the ankle plantar flexors can have a prominent role in supporting the body during stance, except early stance [1,6]. Neptune et al. [1] also showed that the uniaxial ankle plantar flexors (SOL) can simultaneously accelerate the trunk forward and the leg backward, and the biarticular plantar flexors (GAS) the opposite. In mid-stance, while isometric, SOL and GAS accelerated or decelerated the leg and trunk nearly the same. In late stance, while shortening, SOL continued to accelerate the trunk, but to a greater extent than in mid-stance, contributing much to its forward acceleration. The concentric biarticular plantar flexors continued to accelerate the leg, also to a greater extent than in mid-stance, contributing much to swing initiation, even in pre-swing because of the residual force produced during muscle relaxation. The summed contribution of other muscles to trunk support and forward acceleration was also found to be significant in the beginning of stance and in late stance. Although these other muscles were not identified, it is known that the muscles active in the beginning of stance or in late stance include the biarticular hamstrings (HAM), vasti (VAS), gluteus maximus (GMAX) and rectus femoris (RF) [7,8].

In the beginning of stance, VAS is active while the knee flexes. This eccentric VAS activity is commonly assumed to provide shock absorption and leg "stability" by controlling knee flexion and contributing to body support [7,8]. In addition, Hof et al. [9] observed that the eccentric VAS activity corresponded with a decrease in the trunk mechanical energy, leading them to conclude that VAS also absorbs energy from the trunk. We have suggested that VAS may create a hip intersegmental force that is directed vertically and forward in the beginning of stance, which would act to provide support and forward acceleration of the trunk [10], which might seem to dispute the conclusion by Hof et al.

[9]. But the net power effect of VAS on the trunk depends not only on the direction of the hip intersegmental force induced by VAS, but also on the motion of the trunk. If the trunk was moving upward and forward, VAS would indeed act to increase the energy of the trunk, contrary to the conclusion by Hof et al. [9]. However, if the trunk was moving downward and forward, the net power effect of VAS on the trunk could be negative or positive, depending on whether the decelerating effect on the trunk's downward motion or the accelerating effect on the trunk's forward motion dominates, respectively. Because trunk vertical motion changes from downward to upward in the beginning of stance [7,8], it may be that the effect of VAS on the trunk changes from power absorption to power delivery.

GMAX is also active in the beginning of stance but, unlike VAS, shortens because the hip is extending [7,8]. By acting to extend the hip, concentric GMAX is expected to control hip flexion, or stabilize the pelvis and prevent stance leg collapse [7,11]. Others have suggested that GMAX assists in accelerating the trunk forward [12,13]. The direction of the hip intersegmental force caused by GMAX appears to be collinear with the force caused by VAS [10]. Thus, as with VAS, the net power effect on the trunk is uncertain because of the fast changes occurring in trunk motion at that point in the gait cycle.

The effect of biarticular HAM and RF activity on leg and trunk acceleration is even more difficult to assess because each muscle has a flexor moment arm at one joint and an extensor moment arm at the other. Several studies have identified a prominent burst of RF activity in the beginning of stance [14,15] and concluded that the role of RF is to aid VAS in providing lower leg stability during the loading response (i.e. deceleration of the body). On the other hand, Perry [7] argues that RF activity in the beginning of stance would impede trunk forward progression by preventing hip extension because of its presumed action to accelerate the hip into flexion, a presumption based on its anatomical classification (i.e. a hip flexor). But recently, Neptune et al. [1] showed that residual force production in the biarticular GAS during its relaxation in pre-swing can accelerate the knee into extension, contrary to its anatomical classification, because of dynamic coupling in the lower leg [16]. Thus, RF activity in the beginning of stance, while decelerating the leg, might actually accelerate the hip into extension and the trunk forward by causing a forward-directed hip intersegmental force. RF is also active in late stance, which is primarily thought to flex the hip to accelerate the leg into swing [14,15]. However, the activity of RF in late stance may, as in the beginning of stance, actually accelerate the trunk forward while decelerating the extending leg [10].

The objective of this study was to analyze muscle-driven forward dynamic simulations of normal walking to understand the contribution of individual muscles to support and forward progression of the body, with emphasis on the contributions of non-plantar flexor muscles in the beginning and

end of stance. We hypothesized that active force generation by muscles is not only critical to support the body but also critical to the redistribution of power between the leg and trunk to accomplish body progression, and that some muscles perform this important power redistribution while undergoing concentric activity (i.e. producing positive mechanical work) and other muscles while undergoing eccentric activity (i.e. dissipating mechanical energy).

## 2. Methods

### 2.1. Musculoskeletal model

A planar bipedal forward dynamic simulation of normal walking was generated. The musculoskeletal model and the optimization framework used to produce the simulation have been described in detail [1]. Briefly, the musculoskeletal model was generated using SIMM (MusculoGraphics, Inc.) [17] and consisted of rigid segments representing the trunk and legs. Each leg consisted of a thigh, shank and foot. The trunk segment included the mass and inertial characteristics of the pelvis, torso, head and arms. The musculoskeletal geometry was based on that of Delp et al. [18]. The dynamical equations-of-motion for the model were generated using SD/FAST (Symbolic Dynamics, Inc.) and a forward dynamic simulation was produced using Dynamics Pipeline (MusculoGraphics, Inc.) [19].

The model was driven by 15 individual Hill-type musculotendon actuators for each leg. Muscles with similar anatomical classification were combined into muscle groups, with all muscles within each group receiving the same excitation signal. The nine muscle groups were defined as IL (iliacus, psoas), GMAX (gluteus maximus, adductor magnus), VAS (3-component vastus), HAM (medial hamstrings, biceps femoris long head), SOL (soleus), BFsh (biceps femoris short head), GAS (medial and lateral gastrocnemius), RF (rectus femoris) and TA (tibialis anterior). Muscle excitation was modeled as a block pattern defined by onset, duration and magnitude, except for SOL and GAS which were modeled with additional magnitude levels within the burst duration to allow for the characteristic increasing excitation pattern [7,8], and RF which was allowed to have a two-burst pattern during late swing-early stance and pre-swing. The muscle excitation patterns between the left and right legs were considered symmetric and 50% of the gait cycle out-of-phase. The muscle activation dynamics were modeled by a first-order differential equation [20] with activation and deactivation time constants of 50 and 65 ms, respectively.

The contact between the foot and the ground was modeled by 30 independent viscoelastic elements with coulomb friction attached to each foot segment [21]. Each element permitted deformation perpendicular to the floor and represented the combined mechanical properties of the shoe sole and overlying soft tissue.

### 2.2. Optimization framework

A walking simulation of 1.5 gait cycles, beginning with left heel-strike and ending with the second right heel-strike, was generated using a simulated annealing optimization algorithm [22] that found the muscle excitation patterns and initial joint angular velocities that minimized the difference between the simulated and experimentally measured walking data (“optimal tracking problem”). The objective function was formulated to minimize the squared error normalized by the inter-trial variability for each quantity “tracked”. The specific quantities evaluated in the objective function were the right and left hip, knee and ankle joint angles, net joint moments and powers, horizontal and vertical ground reaction forces and the two components ( $x$ ,  $y$ ) of the trunk translation resulting in a total of  $n=22$  tracking variables. The objective function was evaluated from right heel-strike to right heel-strike (called “the gait cycle”), and successful tracking assures an approximately steady-state solution.

The experimental data used to evaluate the objective function were collected at the Rush-Presbyterian-St. Luke’s Medical Center (Chicago, IL) from five healthy male subjects (height= $177.0 \pm 10.4$  cm; weight= $73.3 \pm 12.0$  kg and age= $22.2 \pm 2.1$  years) during normal walking. Informed consent was obtained from the subjects before participating in the data collection. The subjects walked at their self-selected pace while kinematic and ground reaction force data were collected. Intersegmental moments and powers were computed using a standard inverse dynamics approach. A single trial from each subject was processed and averaged across subjects. Further details about the data collection and processing can be found in Andriacchi et al. [23]. A similar optimal tracking technique has been used successfully in other pedaling and walking studies [1,3,24].

### 2.3. Assessing muscle function

To understand the contribution of individual muscles to support and forward progression of the body, the contribution of each muscle to the ground reaction force was determined. This contribution and its corresponding center-of-pressure (needed for subsequent analyses, see below) were determined using a two-step process [1]. First, the total ground reaction force and center-of-pressure were calculated at time step  $i$  from the viscoelastic elements based upon the current state of the system. Then, at time step  $i-1$ , all muscle forces were applied to the system except for the muscle of interest and the equations-of-motion were integrated over the time step from  $i-1$  to  $i$  ( $dt=30$  ms) and the ground reaction force and center-of-pressure were recomputed for the new state of the system. The muscle’s contribution to the ground reaction force and center-of-pressure were approximated by the difference in these quantities for the original and new system states. The process was then repeated for each muscle.

To gain additional insight into the biomechanical mechanisms and muscle synergies needed to provide support and forward progression, muscle-induced acceleration and segmental power analyses were performed over the gait cycle to quantify the role of individual muscles in contributing to the accelerations and power of individual body segments [25]. Briefly, the segment power analysis uses a state-space approach to determine the instantaneous power of each body segment based on the time derivative of the total segmental mechanical energy, which is a function of its current state (position and velocity) and its corresponding acceleration. Therefore, the mechanical power generated, absorbed or transferred by an individual muscle to or from each segment was determined by combining the current state of the segment with the instantaneous accelerations induced by that muscle [25]. The muscle-induced accelerations were determined by setting gravity and all velocities to zero, applying the one muscle force of interest and its corresponding contribution to the ground reaction force at its center-of-pressure, and computing the resulting segmental accelerations from the equations-of-motion using SD/FAST. Because a linear transformation between segment power and acceleration exists [25], the segment power analysis provides a clear interpretation of a muscle's instantaneous influence on a segment. We prefer to discuss segmental power (energy flow), as opposed to segmental energy changes, because energy changes are calculated with specific assumptions as to how the system state will evolve during the specific time period of interest. However, power is calculated directly from the equations-of-motion without these assumptions. Positive power delivered by a muscle to a segment indicates that the muscle accelerated the segment in the direction of the movement, and negative power indicates that the muscle decelerated the segment. Finally, since the right and left leg muscle coordination patterns were symmetrical, the data were analyzed only for the right leg muscles.

### 3. Results

The simulated kinematics, kinetics and EMG timing of a similar simulation were previously shown to compare well with the group-averaged experimental data [1]. Since muscle excitation timing (Fig. 1) was moderately constrained in the optimization to match published EMG data, the timing of muscle force development throughout the gait cycle can be expected to be consistent with normal human subject activity. The average simulation walking speed, stride time and stride length matched the experimental data (1.5 m/s, 1.1 s and 1.6 m, respectively) to within  $\pm 5\%$ .

Ipsilateral muscles contributed substantially to the ground reaction forces (Fig. 2). Contralateral muscles hardly contributed (not shown), even in early stance (0–10% gait cycle; first double support) and pre-swing (50–60% gait cycle; second double support). Non-muscle contributions were small (Fig. 1; dashed and dotted-dashed lines). Grav-

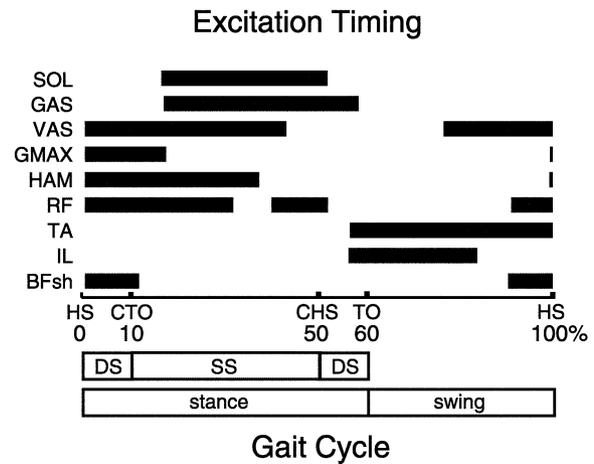


Fig. 1. Muscle excitation timing for the nominal simulation. See text for muscle abbreviations. HS, ipsilateral heel-strike; CTO, contralateral toe-off; CHS, contralateral heel-strike; TO, ipsilateral toe-off; DS, double support with the ipsilateral leg leading the contralateral leg (0–10% gait cycle; early stance) or trailing the contralateral leg (50–60% cycle; pre-swing).

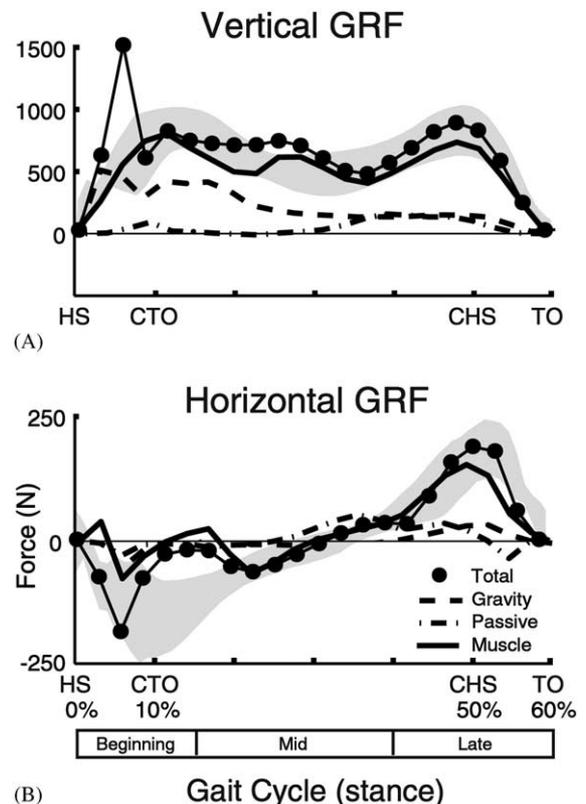


Fig. 2. Simulated vertical and horizontal ground reaction forces over the stance phase (0–60% gait cycle) and the contributions from all muscles, gravity and the passive joint structures. Shaded region indicates the experimentally measured ground reaction force (GRF)  $\pm 1$  standard deviation. Positive force accelerates body upward and forward. Beginning of stance is 0–15%; mid-stance is 15–40%; and late stance is 40–60% of the gait cycle.

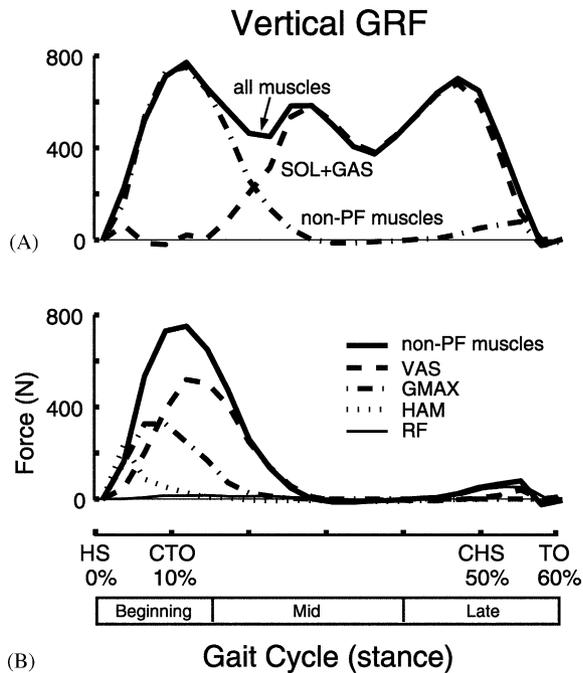


Fig. 3. Muscle contributions to the vertical ground reaction force during stance. (A) Total muscle contribution (all muscles) partitioned into the plantar flexor (SOL+GAS) and non-plantar flexor (non-PF muscles) contributions. (B) Non-PF contribution (same as in (A)) and the individual contributions from VAS, GMAX, HAM and RF. Positive force accelerates body upward.

ity force contributed substantially to the vertical ground reaction force, though its contribution diminished to about 20% body weight as stance progressed.

Though the ankle plantar flexors (SOL and GAS) were the primary muscles contributing to the vertical ground reaction force in mid-stance to toe-off, other muscles were the primary contributors in the beginning of stance (defined as 0 to ~15% gait cycle) into mid-stance (defined as ~15–40% gait cycle) (Fig. 3A; compare dashed line to dotted-dashed line). The uniaxial knee and hip extensors were by far the dominant contributors then (Fig. 3B: VAS, GMAX). HAM and RF contributed negligibly throughout stance (Fig. 3B: HAM, RF). Thus, VAS and GMAX contributed significantly to the support of the body center-of-mass in the beginning of stance.

Muscles providing body support in stance acted to extend the knee and hip, except GAS, which at times tended to flex or at other times extend the hip (Fig. 4). Thus, in the beginning of stance GMAX and VAS accelerated the knee and hip into extension with VAS continuing this action into mid-stance. In mid-stance, both the uni- (SOL) and biarticular (GAS) ankle plantar flexors accelerated the knee into extension, and SOL the hip as well.

In single-leg stance to toe-off, the ankle plantar flexors were the primary muscles contributing to the horizontal ground reaction force (Fig. 5A; dashed line). Until approximately 30% of the gait cycle, the plantar flexors decelerated the forward motion of the body center-of-mass with GAS

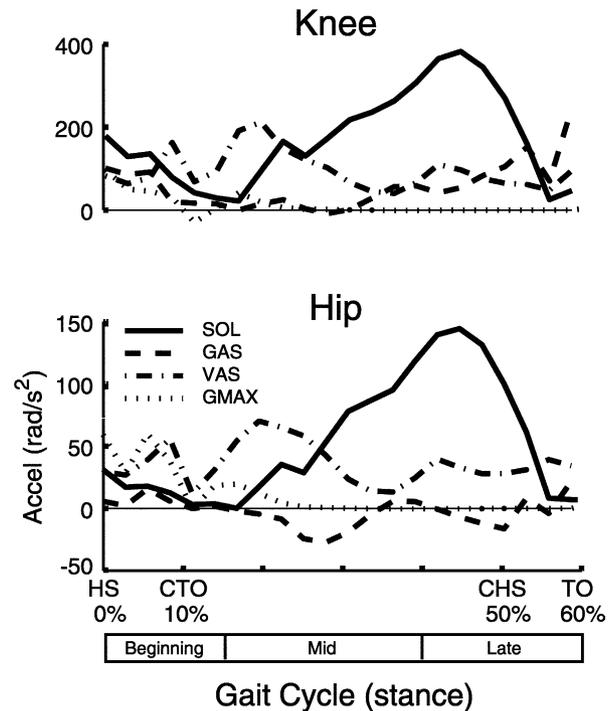


Fig. 4. Knee and hip joint angular accelerations induced by SOL, GAS, VAS and GMAX during stance. VAS and GMAX acted to accelerate the hip and knee into extension (positive acceleration) (except GMAX briefly accelerated the knee into flexion near 15% gait cycle). GAS also accelerated the knee into extension, and either had some flexor or extensor effect at the hip.

hardly contributing (Fig. 4B) because GAS offset its forward deceleration of the trunk by accelerating the leg forward, whereas SOL decelerated the forward motion of both the leg and the trunk [1]. In late stance (~40–60% gait cycle), both SOL and GAS accelerated the body center-of-mass (Fig. 5B), with SOL accelerating the trunk and GAS the leg [1].

In late stance, eccentric RF activity decelerated the forward motion of the body center-of-mass (Fig. 5C: RF) to partially oppose the summed effect of the uni- and biarticular plantar flexors. However, RF decelerated only the leg's forward motion (Fig. 6: RF, dashed line < 0); in fact, the forward motion of the trunk was not decelerated but instead was accelerated (Fig. 6: RF, dotted line > 0). Because RF was lengthening, the power lost in the leg through deceleration was more than the power gained in the trunk through acceleration (Fig. 6: RF, compare dashed line to dotted line). Thus, RF force generation allowed power to be redistributed from the leg to the trunk but redistribution was only partial because RF was dissipating power during its eccentric activity (Fig. 6: RF, solid line < 0).

In the beginning of stance, non-plantar flexor muscles were the primary contributors to the horizontal ground reaction force (Fig. 5A; dotted-dashed line), with VAS dominating the deceleration of the forward motion of the body center-of-mass (Fig. 5C: VAS). HAM countered somewhat the deceleration caused by VAS, and this acceleration of

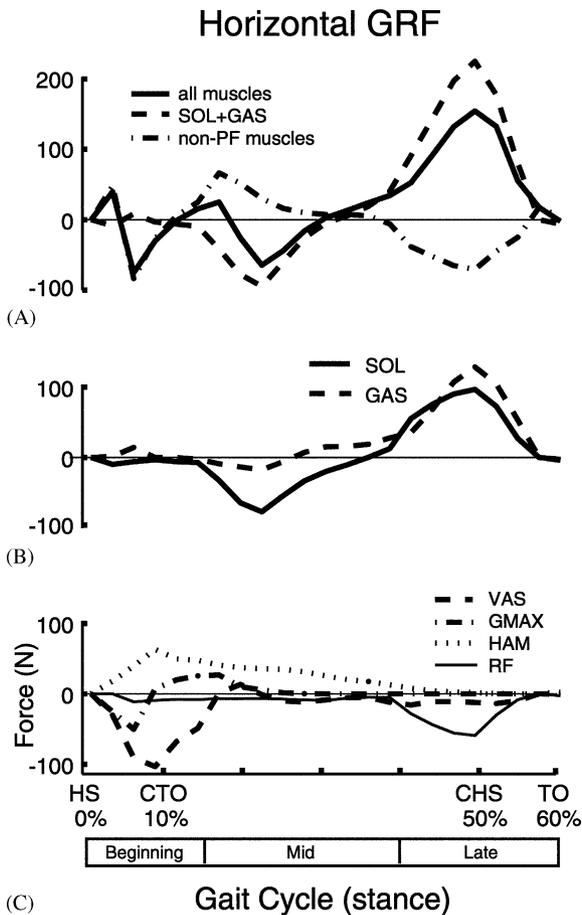


Fig. 5. Muscle contributions to the horizontal ground reaction force during stance. (A) Total muscle contribution (all muscles) partitioned into the plantar flexor (SOL+GAS) and non-plantar flexor (non-PF muscles) contributions. (B) Individual contributions from SOL and GAS. (C) Individual contributions from VAS, GMAX, HAM and RF. Positive force accelerates body forward.

the forward motion of the body center-of-mass by HAM continued further into mid-stance but lessened (Fig. 5C: HAM). GMAX contributed negatively and then positively, and RF had little effect in the beginning of stance (Fig. 5C: GMAX, RF).

VAS decelerated the forward motion of the trunk and the leg in the beginning of stance (Fig. 7: 0–15% gait cycle; dotted line < 0; Fig. 6: VAS, dashed line < 0), consistent with its deceleration of the forward motion of the body center-of-mass (Fig. 5C: VAS, 0–15% gait cycle). VAS also decelerated the downward motion of the trunk and leg very early in stance because of its action to support the trunk and leg, though afterwards VAS accelerated the upward motion of the trunk and leg (e.g. Fig. 7: 0–15% cycle; dashed line < 0 then > 0). Regardless, VAS absorbed power throughout the beginning of stance because of its strong deceleration of the forward motion of the leg, consistent with its eccentric activity (Fig. 6: VAS, 0–15% gait cycle; dashed and solid lines < 0).

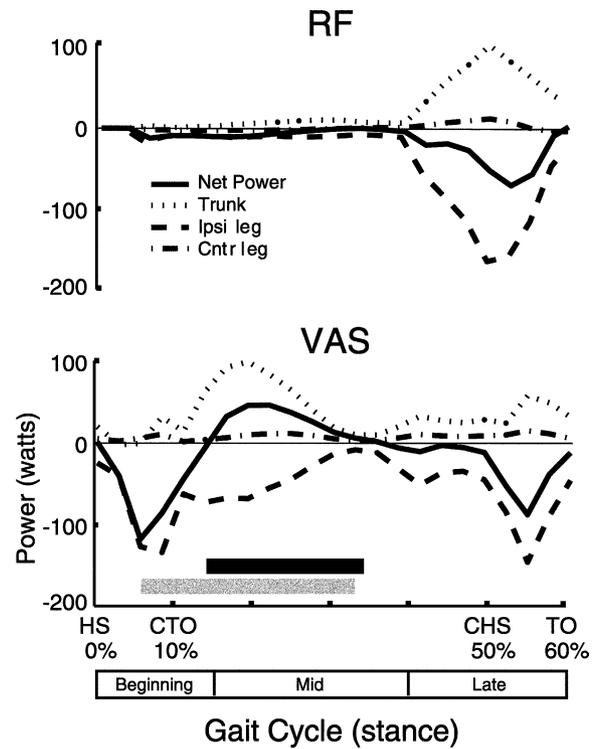


Fig. 6. Musculotendon mechanical power output by RF and VAS (Net Power) and distribution to the ipsilateral (Ipsi leg) and contralateral (Cntr leg) leg segments and trunk (Trunk) during stance. The net power is the mechanical power output from the musculotendon actuator. Positive (negative) net power indicates power production (absorption) by the actuator. Positive (negative) power to the leg/trunk indicates that the actuator is accelerating (decelerating) the leg/trunk in the direction of movement. Leg power is mostly in the horizontal direction. Horizontal bars indicate when knee is extending (black bar) and when the trunk is moving upward (gray bar).

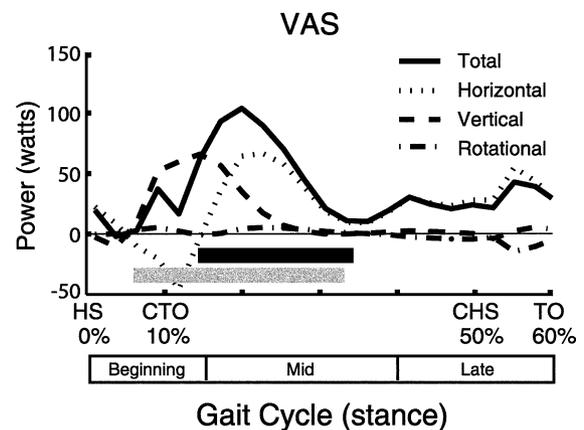


Fig. 7. Mechanical power delivered to the trunk by VAS during stance. Horizontal power (Horizontal) is the time rate-of-change of the horizontal kinetic energy; vertical power (Vertical) is the time rate-of-change of the vertical kinetic and potential energy; anterior/posterior tilting power (Rotational) is the time rate-of-change of rotational kinetic energy. Total power to or from the trunk (Total) is the sum of the horizontal, vertical and anterior/posterior tilting power. Black bar indicates when the knee is extending. Gray bar indicates when the trunk is moving upward.

VAS, by sustaining its activity further into mid-stance when the knee extends and with the trunk moving upward, continued to decelerate the forward motion of the leg but now accelerated, rather than decelerated, the forward motion of the trunk (Fig. 6: VAS, 15–35% gait cycle; dashed line  $< 0$ ; Fig. 7: 15–35% cycle; dotted line  $> 0$ ). Consequently, VAS had a negligible net effect on the forward motion of the body center-of-mass (Fig. 5C: VAS, 15–35% gait cycle). VAS continued to accelerate the upward motion of the trunk and the leg because it continued to support them (e.g. Fig. 7: 15–35% cycle; dashed line  $> 0$ ). The net effect on power in the leg by VAS was still negative because deceleration of the leg's forward motion dominated (Fig. 6: VAS, 15–35% cycle; dashed line  $< 0$ ). However, the net effect on power in the trunk by VAS rose abruptly to become positive (Fig. 6: VAS, 15–35% cycle; dotted line  $> 0$ ) because VAS accelerated rather than decelerated trunk forward motion (Fig. 7: 15–35% cycle; dotted line  $> 0$ ). Thus, VAS force generation allowed power to be redistributed from the leg to the trunk (Fig. 6: VAS, 15–35% cycle; dashed line  $< 0$  and dotted line  $> 0$ ). But because VAS was also producing power from its concentric activity (Fig. 6: VAS, 15–35% cycle; solid line  $> 0$ ), even more power was delivered to the trunk.

#### 4. Discussion

We found that force generation by stance-leg muscles supports the body and maintains its progression through the redistribution of leg and trunk segmental power, consistent with the concept of motor task execution through segmental power redistribution by muscles [2]. This redistribution of power among segments during walking resulted from both eccentric muscle activity (e.g. VAS in the beginning of stance; SOL/GAS in early single-leg stance [1]; and RF in late stance) and concentric activity (e.g. VAS in mid-stance; SOL/GAS in late stance [1]). Some muscles in stance redistributed segmental power first with their eccentric activity and immediately afterwards with their isometric and concentric activity (VAS; SOL/GAS [1]). Much redistribution was found to occur in the beginning of stance and in late stance near double-leg support, which is purported to be an efficient time to add energy to the task and which coincides with redirection of the body center-of-mass from downward and forward to upward and forward [26–28]. The eccentric-to-isometric-to-concentric activity of the uniaxial and biaxial plantar flexors in single-leg stance, with their co-functionality to support the body and their synergism to maintain body forward progression, may also be an efficient mechanism by which the body can act as an inverted pendulum [2,10,26–28].

##### 4.1. Limitations of our study

The identification of individual muscle contributions to body support and forward motion through redistribution of

segmental power was possible through a detailed analysis of a computer simulation that emulated the kinematics and kinetics of walking. Some limitations of musculoskeletal modeling and simulation generation and analysis have been described [1,2,10].

One limitation of our study is that the pelvis, torso, head and arms were combined into one trunk segment and controlled by a small number of leg muscles attached to the pelvis (cf. additional muscles attaching to both the pelvis and trunk, such as the lower back and abdominal muscles). Consequently, the large mass of the trunk segment oscillated anteriorly–posteriorly around the medial–lateral axis more than normal at impact, causing the hip to flex and GMAX to lengthen momentarily during early stance. A more complex model that includes a separate pelvis and trunk segment controlled by lower back and abdominal muscles may provide the necessary stability [4]. As a result, the single-segment trunk model used in our model required muscles predominantly crossing the hip to stabilize the trunk. To assess whether this additional requirement of muscles to stabilize the trunk would influence our interpretation of muscle function, we performed a sensitivity analysis by adding a passive viscoelastic torsional spring about the trunk center-of-mass to stabilize the trunk's anterior–posterior tilting. This analysis showed that the interpretations of muscle function using the one-segment trunk remained the same.

The one-segment trunk model used in our study does not allow pelvis rotation (rotation in the transverse plane) or pelvic listing (rotation in the coronal plane). The contribution of the hip abductors (e.g. gluteus medius) to body support can, however, be substantial in single-leg stance, as shown by Anderson and Pandey [6] in a 3D model of walking, and hip abductor activity prevents pelvic listing, as shown by Yamaguchi and Zajac [29] in another 3D modeling study. Thus, the support provided by the plantar flexors in our study, especially the support provided by SOL near mid-stance, may be overestimated because of this unaccounted contribution of the hip abductors. Pelvis rotation causes the hips to move relative to one another in the anterior–posterior direction. With respect to our analysis, we believe that this relative motion of the hips will mostly affect the relative forward acceleration of the ipsilateral and contralateral legs caused by HAM and GMAX in stance. Both muscles were found to accelerate the contralateral leg forward through appreciable muscle-induced hip intersegmental forces. Thus, HAM and GMAX accelerated both legs (e.g. see Fig. 9: HAM), causing the high power produced by them while shortening in stance to be delivered to both legs, though some power produced by GMAX also acted to accelerate the trunk (unpublished data from our simulations). Because the motion of one hip relative to the other is small during unimpaired walking compared with the forward motion of the trunk, the energy changes in the trunk and the induced ipsilateral and contralateral hip intersegmental forces from small transverse plane rotations are, however, expected to be small. We believe, therefore, that the basic muscle mechanisms

emphasized in this study and espoused to account for support and forward progression of the body will be generally unaltered when pelvic rotation and listing are allowed, with the caveat above regarding the overestimation of the plantar flexors role in providing body support near mid-stance.

Because of our desire to understand muscle function at impact, we used a slightly different simulation than in our previous study [1], which focused on the function of the ankle plantar flexors (which are not active at impact). In the present study, we constrained the simulation to include 50% of the gait cycle before impact to assure that the initial startup transients had decayed and the solution at impact was near steady-state. Nevertheless, the two simulations of a gait cycle were nearly identical and interpretations of muscle function unchanged. However, the vertical and horizontal ground reaction forces of these simulations at impact were larger in magnitude than the measured forces (Fig. 2). Though the contribution of VAS and other muscles to support and forward acceleration of the body center-of-mass near impact may therefore be less, their qualitative function in accelerating the body segments and redistributing segmental power is expected to be unaltered.

An additional difference from our previous study [1] is the integration step size used in the ground reaction force decomposition. Previously, we had focused on the ankle plantar flexors and used an integration step size of 2.2 ms after finding that those muscles were generally insensitive to variations in step size. However, in the present study, such a short integration step size leads to different qualitative functions for VAS and GMAX in very early stance (~0–7% gait cycle). For the present study, we performed a sensitivity analysis and determined that an integration step size of 30 ms was optimal using the following criteria: (1) the components of the vertical and horizontal ground reaction force approximately summed to the ground reaction forces produced in the simulation, assuming that the contributions from Coriolis and centripetal forces are negligible during normal walking, (2) the power delivered to the segments by each muscle approximately summed to equal the positive or negative power produced or absorbed by the musculo-tendon actuator, (3) the qualitative function of each muscle had reached steady state and (4) the same step size could be used to meet the previous three criteria for similar analysis of both forward and backward walking (backward walking data are not presented). We believe the reason that the 2.2 ms step size leads to a different conclusion in very early stance is because a longer time is required for the proximal muscles (e.g. VAS and GMAX) to deform the viscoelastic contact elements attached to the foot segment due to the lower foot acceleration induced by those muscles (cf. ankle plantar flexors). We previously reported that VAS and GMAX act to accelerate the trunk forward in the beginning of stance [2,10,30] and that their contribution is about the same magnitude as the summed contribution of the plantar flexors in late stance [1,10]. We now believe that in the beginning of stance VAS while lengthening (eccentric activity), and prob-

ably GMAX while shortening (concentric activity), do not accelerate the trunk forward at all. But afterwards when both VAS and GMAX shorten, they are believed to indeed accelerate the trunk forward, though their contribution is probably much less than the plantar flexors' later contribution.

#### 4.2. Support of the body by muscles

The uniarticular eccentric knee (VAS) and concentric hip (GMAX) extensors were found to provide critical support to both the leg and the trunk in the beginning of stance. This role of VAS and GMAX to support the body is consistent with the notion that the knee and hip extensors must provide body weight acceptance in early stance because of the reduction in body support provided from the contralateral leg in pre-swing [7,8]. Perry [7] has also emphasized the role of knee and hip extensors to stabilize the leg as it decelerates knee flexion. Our results support this contention since VAS acted to accelerate the knee and the hip into extension (Fig. 4). Hof et al. [9] have suggested that VAS dissipates trunk energy in the beginning of stance as it actively lengthens. Our results indicate, however, that VAS has little net effect on trunk power (Fig. 6: VAS; dotted line  $\approx 0$ ), though VAS does decelerate the trunk's forward motion while the knee extends, which is more than offset by its acceleration of the trunk's upward motion (Fig. 7; dashed line opposite to dotted line). Though we had thought that VAS would accelerate the trunk so much that a large power redistribution from the leg to the trunk would occur [2,10,30], we now believe that the major function of VAS in the beginning of stance is to decelerate the leg (Fig. 6: VAS; dashed and solid lines  $\approx 0$ ). We also found that GMAX accelerated the knee and hip into extension (Fig. 4), consistent with the suggestion by others that the net hip extensor power in the beginning of stance prevents lower leg collapse and stabilizes the trunk [7,11]. We and Anderson and Pandy [6] have found that HAM and RF do not support the body in the beginning of stance.

In mid-stance when the knee extends, we found the body support contribution by the now concentric VAS and the continuing concentric GMAX to decrease. The uniarticular (SOL) and biarticular (GAS) plantar flexors begin their activity in early single-leg stance [7,8]. As a consequence, we found that they replace the decreasing support provided by the knee and hip extensors. Consistent with continuing plantar flexor activity until contralateral heel-strike [7,8], the plantar flexors were found in our study to sustain their support of the body. Anderson and Pandy [6] also found that plantar flexor activity supports the body, though their simulation had the plantar flexors excited only in late stance. In pre-swing, we and Anderson and Pandy [6] found that the contribution of the plantar flexors subsides, coincident with muscle relaxation, and that the contribution of RF is negligible.

The muscle and gravity contributions to the vertical ground reaction force found by us agree in large part with those found by Anderson and Pandy [6]. However, some

differences exist and probably arise from the dissimilar models used to represent the body, the different solutions to muscle excitation patterns afforded by muscle redundancy and the dissimilar methods used to compute the individual muscle contributions to the ground reaction force [31]. One difference is the contribution of muscles in single-leg stance, which Anderson and Pandy [6] found to be dominated by gluteus medius/minimus and gravity. Because our model is a 2D model and the contribution of the pure hip abductors to vertical support is not included, the computed plantar flexor contribution in single-leg stance may be overestimated as noted above. Another reason our estimate may be high is that we use a single foot segment, which demands a higher ankle plantar flexor moment. On the other hand, because they constrained the foot to be flat on the ground in single-leg stance [6,31] and their simulated plantar flexor excitation did not begin until 40% of the gait cycle [4], their computed contribution from gravity may be overestimated and their muscle contributions underestimated, especially from SOL. Another difference is the contribution of muscles in very early stance. We found that VAS and GMAX are the primary contributors to support; they found the dorsiflexors to provide support until the foot became flat on the ground. We believe that the difference arises because our models for foot contact differ; our model has 30 viscoelastic elements distributed on the surface of the foot and theirs has only five elements. Regardless, this difference in muscle contribution to body support exists only for a brief time near heel-strike, an event most challenging to model [2].

#### 4.3. Maintenance of body forward progression by muscles

It is well-known that the fore-aft ground reaction force of the leading leg decelerates the forward motion of the body center-of-mass in the beginning of stance and then, when it becomes the trailing leg, reverses to accelerate the body forward in late stance [7,8]. We found that muscles are the primary contributors to the horizontal ground reaction force.

In the beginning of stance before the onset of plantar flexor activity [7,8], the lengthening VAS was found to dominate the deceleration of the forward motion of the trunk and leg, with GMAX also contributing to leg forward deceleration (unpublished data). RF activity may also assist VAS in decelerating the leg in the beginning of stance. Many studies have shown that a prominent burst of RF activity appears in beginning of stance [8,14,15,32]. But Perry [7], who does not find such activity, has suggested that RF activity should be non-existent in the beginning of stance because it would impede ongoing hip extension. However, our simulation data show that RF actually acts to accelerate the hip into extension, though barely (Fig. 8) because of its low excitation, due to the dynamic coupling associated with the effect from its knee extensor moment. We further analyzed this finding by performing a sensitivity analysis on the magnitude and duration of RF activity in the beginning of stance and on its hip flexor moment arm. These other simulations showed

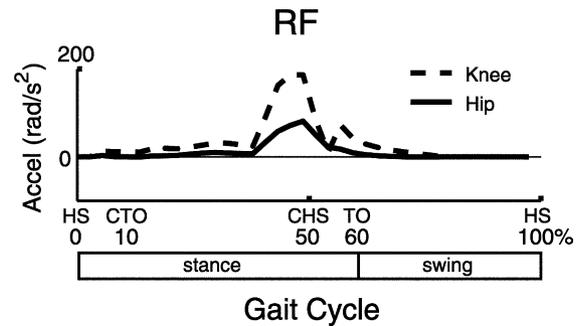


Fig. 8. Hip and knee joint angular accelerations induced by RF over the gait cycle. Extension acceleration is positive. RF accelerated the hip into extension, even though RF is anatomically defined as a hip “flexor” muscle, because of dynamic coupling (i.e. the acceleration of the hip into extension by its knee extensor moment was greater than the hip flexion acceleration induced by its hip flexor moment).

that RF indeed accelerates the hip into extension regardless of the magnitude and duration of its activity and for any hip flexor moment arm less than 4 cm (i.e. a value larger than suggested in the literature). However, we found that the effects of RF, which were to accelerate the trunk forward and decelerate the leg, were small unless RF was near full excitation. Thus, RF activity in the beginning of stance may not be the result of cross talk, as suggested by Perry [7], but rather may be due to the variability in subject-specific motor strategies [15] made possible through muscle redundancy and the limited contribution of RF to the overall task performance.

In mid-stance when the knee extends, shortening VAS was found to continue to decelerate the leg’s forward motion as in the beginning of stance but, instead of continuing to decelerate the trunk, VAS accelerated the trunk forward. Thus, the net effect by VAS on the body center-of-mass was negligible. Instead, the ankle plantar flexors were found to decelerate the forward motion of the body center-of-mass in the first half of single-leg support. Our earlier study shows that SOL decelerates both the trunk and leg but GAS only the trunk [1].

In late stance, the dominant contributors to the acceleration of the body forward motion were the concentric ankle plantar flexors. This result agrees with Kepple et al. [5], who found that the net ankle extensor moment accelerates the trunk forward, and our previous simulation analysis, which showed SOL acted to accelerate the trunk forward and GAS the leg forward to initiate swing [1].

However, RF eccentric activity in late stance was found to oppose the forward acceleration of the body by the plantar flexors through its deceleration of the leg. RF is decelerating the leg while GAS is accelerating the leg forward in late stance. However, unlike GAS, RF also accelerated the trunk forward because of its forward-directed hip intersegmental joint force [10], which augments the acceleration caused by SOL [1]. Through their action to redistribute segmental power, SOL, GAS and RF may, therefore, work synergistically to ensure forward acceleration of the trunk and initiation of swing [10].

#### 4.4. Initiation of swing

Because eccentric RF activity was found to decelerate the leg in late stance including pre-swing, we believe that RF force generation does not contribute to swing initiation, contrary to the suggestions of others [7,9,15], which are based on the assumption that its hip flexor moment dominates. Our results show that RF does not act to flex the hip because its knee extensor moment, which acts to accelerate both the hip and knee into extension, has a more powerful action than its hip flexor moment, which acts to accelerate the hip and knee into flexion. Therefore, because of dynamic coupling [16], the net effect by RF is to accelerate the hip and knee into extension (Fig. 8).

In contrast, the shortening uniarticular hip flexors (IL) were found to accelerate the hip into flexion and the leg forward in pre-swing into swing (Fig. 9). However, IL also

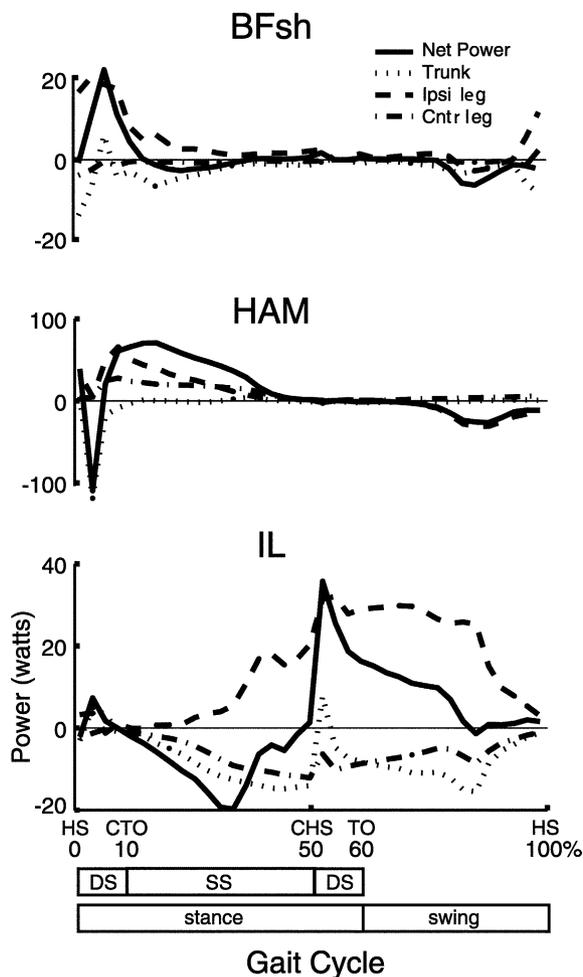


Fig. 9. Musculotendon mechanical power output by BFsh, HAM and IL, and distribution to the ipsilateral (Ipsi leg) and contralateral (Cntr leg) leg segments and trunk (Trunk) over the gait cycle. In early stance, HAM power output is briefly negative (solid line  $< 0$ ), indicating momentary lengthening, and the trunk loses power (dotted line  $< 0$ ), which probably do not exist in reality, but arise in the simulation because of anterior/posterior tilting oscillations at heel-strike (see Section 4). See Fig. 6 caption.

simultaneously redistributed power from the trunk to the leg by decelerating the trunk forward, contrary to the suggestion of others that a hip flexor moment assists trunk forward acceleration [13,33]. Any passive structures (e.g. ligaments, tendon and tissue) acting to generate a hip flexor moment in late stance into pre-swing and swing will also accelerate the leg forward by redistributing power from the trunk to the leg through deceleration of the trunk. For example, in our study the stretched elastic structures in the passive hip flexors in single support generate force that accelerates the leg forward by decelerating the trunk backwards (Fig. 9: IL, 20–50% cycle; dashed line  $> 0$  and dotted line  $< 0$ ). We also found that non-muscle elastic structures crossing the hip store a substantial amount of mechanical energy during hip extension that is recovered in pre-swing. Though some studies have also indicated that the passive moment may be contributing as an energy storage and release mechanism [34], others have suggested that the effect is negligible at normal walking speeds [35]. Regardless of the contribution of the passive structures to swing initiation, the qualitative effect of muscles on swing initiation described in this study will remain unaffected.

We conclude, therefore, that acceleration of the leg to initiate swing arises from force generation by GAS in late stance, including pre-swing because of muscle relaxation [1], IL in pre-swing into swing and passive hip structures in single-leg stance into pre-swing. It is also suggested that neural control of GAS and IL can afford effective and proper control of swing initiation. For example, when the stiffness of the passive hip structures was decreased in our model and another simulation generated, GAS and IL increased their activity and swing was again initiated well.

#### 4.5. Advancement and deceleration of the leg during swing

If BFsh should be active in late swing, as Sutherland [36] observes, our simulation analysis suggests that lengthening BFsh would assist lengthening HAM in decelerating the leg (Fig. 9: BFsh and HAM, 75–100% gait cycle; dashed and solid lines  $< 0$ ). If BFsh activity should continue into early stance [36], our results show that the now shortening BFsh would accelerate the leg, just as HAM also shortens and accelerates the leg then.

We believe that the uniarticular knee and hip flexor muscles accelerate the leg in early into mid swing. If BFsh should be active in early swing as Perry [7] suggests, our results from another simulation with the BFsh burst occurring in early swing shows that as BFsh shortens it acts co-functionally with the shortening IL to accelerate the leg, although the proportion of horizontal, vertical and rotational accelerations is different. The degree of acceleration required by BFsh and IL will depend on the kinetic energy of the leg at toe-off resulting from swing initiation. Under ideal conditions, such that the swing leg replicates “ballistic walking” [37], little activity in BFsh and IL would be expected.

Thus, BFsh can act co-functionally with IL in early swing to accelerate the leg, with HAM in late swing by dissipating energy and decelerating the leg and with HAM in early stance by producing energy and accelerating the leg. Because of this co-functionality of BFsh with other muscles, it is not unexpected that BFsh activity may be absent at times in those regions where Perry [7] and Sutherland [36] have observed activity. Differences between studies may, therefore, be related to variability in subject-specific motor strategies arising from muscle redundancy.

#### 4.6. Forward trunk acceleration by the swing leg

Though different hypotheses have appeared in the literature regarding the contribution of the swing leg to trunk forward acceleration, our results suggest that the swing leg muscles and the non-muscular sources of energy (gravity and inertia) contribute little compared with the stance-leg muscles. Hof et al. [9] suggested that the acceleration (by plantar flexors and hip flexors) and deceleration (by HAM/BFsh and possibly GMAX) of the swing leg during early and late swing, respectively, cause the greatest energy exchanges in the trunk. However, we found that the acceleration of the swing leg by uniaxial hip flexors causes a posteriorly directed hip intersegmental force, which decelerates trunk forward acceleration (Fig. 9: IL, dotted line  $< 0$ ). Further, the power reduction in the leg caused by HAM in late swing was dissipated into heat and not to accelerate the trunk (Fig. 9: HAM; dashed line  $\approx$  solid line  $< 0$ ). However, both of these results may be sensitive to the trunk model (see above). Finally, though Riley et al. [38] suggested that swing leg muscle activity is most critical to forward progression and that the stance leg contributes very little, methodological issues with that study [39] have called into question their results, which have been contradicted by this study and that of Anderson and Pandy [6].

## 5. Concluding remarks

Body support and forward progression was found to occur to a large extent because of the redistribution of power between the legs and trunk by the force generation of muscles, regardless of whether force was produced from concentric, isometric or eccentric activity. We used a dynamics-based analysis to quantify the contribution of individual muscles to body progression. Muscle function was often found to be non-intuitive because of the redistribution of power from one body segment to another caused by muscle force generation. Precise quantification of a muscle's contribution to body support and forward progression resolved many of the conflicting interpretations of muscle function that were based on correlation between muscle EMG timing and the ongoing gait mechanics. Our study lays the foundation for understanding how gait is limited by abnormal muscle timing in different populations with pathology. Further stud-

ies will be able to examine the potential of compensatory mechanisms to replace lost or impaired muscle function and identify promising rehabilitation strategies.

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