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# Muscles that support the body also modulate forward progression during walking

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## Abstract

The purpose of this study was to characterize the contributions of individual muscles to forward progression and vertical support during walking. We systematically perturbed the forces in 54 muscles during a three-dimensional simulation of walking, and computed the changes in fore–aft and vertical accelerations of the body mass center due to the altered muscle forces during the stance phase. Our results indicate that muscles that provided most of the vertical acceleration (i.e., support) also decreased the forward speed of the mass center during the first half of stance (vasti and gluteus maximus). Similarly, muscles that supported the body also propelled it forward during the second half of stance (soleus and gastrocnemius). The gluteus medius was important for generating both forward progression and support, especially during single-limb stance. These findings suggest that a relatively small group of muscles provides most of the forward progression and support needed for normal walking. The results also suggest that walking dynamics are influenced by non-sagittal muscles, such as the gluteus medius, even though walking is primarily a sagittal-plane task.

*Keywords:* Walking; Forward dynamics; Induced accelerations; Muscle function

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## 1. Introduction

Muscles enable walking by providing vertical support and maintaining forward progression. During normal walking, the mass center of the body undergoes cyclic accelerations in both the vertical and fore–aft directions. These accelerations are directly related to the ground reaction force. In early stance, muscles contribute to a vertical ground reaction force that exceeds body weight, accelerating the body mass center upward. During midstance, the ground reaction force falls below body weight, causing the body mass center to accelerate downward. During late stance, muscles again contribute to a vertical ground reaction force that is greater than body weight, accelerating the body mass center upward. There are similar periods of acceleration and deceleration in the fore–aft direction. In the first half of stance, muscles contribute to a ground reaction force in the aft direction, slowing the forward progression of the mass center. During the second half of stance, muscles generate a ground reaction force that accelerates the mass center forward. Identification of the muscles that contribute to the vertical and fore–aft accelerations of the body is of interest to researchers involved in human movement science and to clinicians seeking to improve the walking ability of patients with neuromusculoskeletal disorders.

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Previous studies have identified muscles that may significantly contribute to forward progression. Many researchers have concluded that the plantarflexors are the primary source of forward acceleration during late stance (e.g., Gottschall and Kram, 2003; Kepple et al., 1997; Neptune et al., 2001, 2004; Pandy, 2001; Sutherland et al., 1980; Winter, 1983). Fewer studies have examined how muscles contribute to or inhibit progression during the first half of stance. Neptune et al.'s (2004) analysis of a sagittal-plane walking simulation indicates that hamstrings generate forward acceleration during the first half of stance, while vasti and gluteus maximus decelerate the body mass center. Pandy (2001) also found that the vasti group was responsible for slowing the body during early stance.

Muscle contributions to the vertical acceleration of the mass center, and thus to support of body weight, have been studied with two- and three-dimensional simulations of walking. Analysis of a two-dimensional simulation suggested that uniarticular hip and knee extensors (vasti and gluteus maximus) generate the majority of vertical support during the first half of stance, while the plantarflexors provide support for the remainder of stance (Neptune et al., 2004). Analysis of a three-dimensional simulation of normal walking revealed that the hip abductors (i.e., gluteus medius and gluteus minimus) also make substantial contributions to vertical acceleration, especially during single-limb support (Anderson and Pandy, 2003).

While walking simulations (Neptune et al., 2001, 2004; Pandy, 2001) have suggested that gluteus maximus, vasti, and plantarflexors provide support and also modulate forward progression, the potential roles of non-sagittal muscles, such as the hip abductors, in modulating forward progression remain unclear. Furthermore, while the influence of gravity on vertical support has been quantified for a complex walking simulation (Anderson and Pandy, 2003), gravity's effects on fore–aft acceleration are not well understood. In this study, we analyzed a three-dimensional simulation of walking to determine how individual muscles and gravity contribute to the fore–aft acceleration of the body mass center. We combined these data with contributions to vertical acceleration to synthesize a more complete picture of how muscles and gravity contribute to support and forward progression during normal walking.

## 2. Methods

A three-dimensional dynamic simulation of walking (Anderson and Pandy, 2001) was used to examine the contributions of muscles to forward progression and support. Forward progression and support were quantified by the fore–aft and vertical accelerations, respectively, of the body mass center. The body was modeled as a 10-segment, 23-degree-of-freedom articulated linkage actuated by 54 Hill-type musculotendon actuators (Zajac, 1989). The back joint and hip joints were modeled as ball-and-socket joints. Each knee was modeled as a hinge, each ankle–subtalar complex as a universal joint, and each metatarsal–phalangeal joint as a hinge. The directions of the knee, ankle–subtalar complex, and metatarsal–phalangeal joint axes were all based on anatomical data. Muscle parameters and path geometries were based on data reported by Delp et al. (1990). Ligaments were modeled as passive torques that prevented hyperextension or extreme flexion. The interaction between each foot and the ground was modeled using stiff spring-damper units distributed under the sole of the foot. The simulation of walking was obtained by solving a dynamic optimization problem for the muscle excitations that minimized the metabolic energy expenditure per distance traveled in the direction of progression. The joint angular displacements, ground reaction forces, and muscle excitation patterns predicted by the dynamic optimization solution were similar to those obtained from five healthy subjects who walked at an average speed of 1.35 m/s (Anderson and Pandy, 2001).

The contribution of a particular muscle to the fore–aft acceleration of the body mass center, $\ddot{x}_m(t_i)$, can be evaluated using the following expression:

$$\ddot{x}_m(t_i) = \frac{\partial \ddot{x}}{\partial F_m} F_m = \frac{\ddot{x}(F_m + \Delta F_m, t_i) - \ddot{x}(F_m, t_i)}{\Delta F_m} F_m, \tag{1}$$

where $t_i$ is the current time in the simulation, $F_m$ is the force generated by muscle $m$, $\Delta F_m$ is a constant perturbation to $F_m$, and $\ddot{x}$ is the acceleration of the center of mass in the fore–aft direction. Because acceleration depends linearly on force, forward differences is an exact expression for $\partial \ddot{x}_m / \partial F_m$ in Eq. (1). However, not evident in Eq. (1) is the fact that perturbing a muscle force will generate changes in the system reaction forces (e.g., ground reaction forces) that also contribute to the accelerations of the body segments. These induced reaction forces must be quantified and included in the final computation of the accelerations induced by a muscle. Anderson and Pandy (2003) quantified the induced reaction forces by performing a hard-constraint decomposition, and Neptune et al. (2001) did so by using an integration method.

In this study, we used a perturbation technique to evaluate Eq. (1) that implicitly accounts for the changes in the system reaction forces. Specifically, we calculated each muscle's contribution to the fore–aft acceleration of the body mass center by perturbing that muscle's force, simulating forward in time by a short interval $\Delta t$, and observing the resulting change in position of the mass center. Assuming that the acceleration induced by a muscle over this short interval is constant, the observed changes in position can be related to the accelerations using the following relations:

$$x(F_m, t_i + \Delta t) \approx x(F_m, t_i) + \dot{x}(F_m, t_i)\,\Delta t + \tfrac{1}{2}\ddot{x}(F_m, t_i)\,\Delta t^2, \tag{2}$$

and

$$\begin{aligned} x(F_m + \Delta F_m, t_i + \Delta t) &\approx x(F_m + \Delta F_m, t_i) \\ &\quad + \dot{x}(F_m + \Delta F_m, t_i)\Delta t + \tfrac{1}{2}\ddot{x}(F_m + \Delta F_m, t_i)\Delta t^2, \end{aligned} \tag{3}$$

where $x(F_m, t_i + \Delta t)$ and $x(F_m + \Delta F_m, t_i + \Delta t)$ are the unperturbed and perturbed fore–aft positions, respectively, of the mass center at $t_i + \Delta t$. Subtracting Eq. (2) from Eq. (3), recognizing that $x(F_m + \Delta F_m, t_i) = x(F_m, t_i)$ and $\dot{x}(F_m + \Delta F_m, t_i) = \dot{x}(F_m, t_i)$ because the positions and velocities cannot change instantaneously in response to a force perturbation, and grouping the acceleration terms on one side yields

$$\begin{aligned} \ddot{x}(F_m + \Delta F_m, t_i) \; - \ddot{x}(F_m, t_i) \;\; &\approx \\ 2 \cdot \frac{x(F_m + \Delta F_m, t_i + \Delta t) - x(F_m, t_i + \Delta t)}{\Delta t^2}&. \end{aligned} \tag{4}$$

Substituting Eq. (4) into Eq. (1) provides a formula for estimating the accelerations induced by a muscle suitable for use in a perturbation analysis:

$$\ddot{x}_m(t_i) \approx 2 \cdot \frac{x(F_m + \Delta F_m, t_i + \Delta t) - x(F_m, t_i + \Delta t)}{\Delta t^2 \, \Delta F_m} \; F_m. \tag{5}$$

The acceleration due to each muscle was evaluated every 0.02 s during the simulation. The forces applied by unperturbed muscles were constrained to follow their unperturbed trajectories, while foot spring forces and passive ligament torques (i.e., the system reaction forces) were allowed to change in response to the perturbed muscle force. The magnitude of the force perturbation, $\Delta F_m$, was chosen to be 1.0 N. Results were insensitive to the magnitude of this force perturbation over three orders of magnitude (i.e., 0.01, 1.0, and 10.0 N). The duration of the perturbation ($\Delta t = 0.03$ s) was short enough to prevent the kinematics from deviating significantly from their unperturbed values, but long enough to allow the foot springs to respond to a change in force. The accelerations due to muscles were somewhat insensitive to the size of $\Delta t$ in the range of 0.02 s to approximately 0.07 s. Given that similar results were obtained over this range of time windows, we selected a $\Delta t$ that was small but not the lower boundary of the stable range. The muscle contributions to fore–aft and vertical acceleration were computed with the same method.

A similar perturbation analysis was used to quantify the contributions of gravitational acceleration to fore–aft and vertical mass center accelerations. A formula analogous to Eq. (5) was used, and the nominal gravitational acceleration was perturbed by 0.01 m/s$^2$. The results for the gravitational contributions to mass center accelerations were also insensitive to perturbation size over a range of three orders of magnitude.

To evaluate how well this analysis quantified the contributions of muscles and gravity to the acceleration of the body's mass center, we compared the combined accelerations from all muscles and gravity to the fore–aft and vertical accelerations of the body mass center in the unperturbed simulation (Fig. 1). The muscle and gravity accelerations did not contain the high-frequency peaks of the unperturbed accelerations during double support (0–15% and 50–65% of the gait cycle); these peaks were due to the response of the

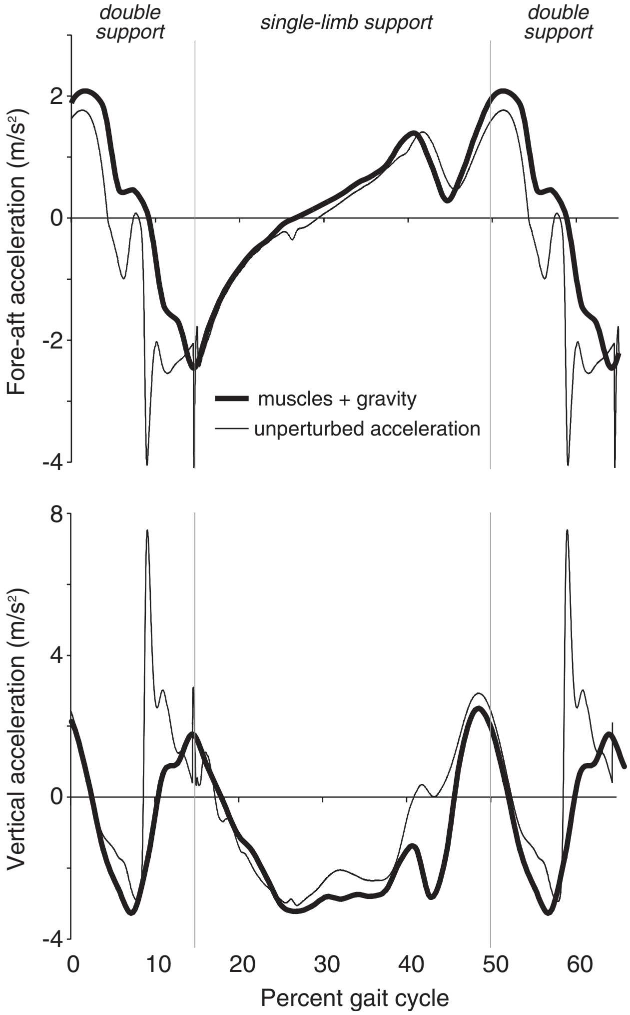

Fig. 1. Combined contributions to fore–aft (top) and vertical (bottom) accelerations from all 54 muscles and gravity compared to the acceleration of the body mass center during the unperturbed simulation. Note that the vertical acceleration of the center of mass in the simulation was not uniformly positive throughout double support as in typically observed in experiments. This was because the dorsiflexor activity in the model was below normal, resulting in both the forefoot slapping the ground at foot-flat just prior to 10% of the gait cycle and a reduced vertical velocity of the center of mass at the end of double support.

model's foot springs as floor contact changed. The ability to reconstruct the general shapes of the accelerations of the mass center lends confidence that the method used for computing the body accelerations due to muscles and gravity is sufficiently accurate.

The accelerations of the mass center due to each musculotendon actuator were calculated during the stance phase; accelerations from muscles during the swing phase were very small. Since the left and right muscle forces are symmetric, we only present data for right-side muscles. The stance phase was divided into halves at the time during single-limb support when the fore–aft acceleration of the mass center switched from negative (aft) to positive (forward). Specifically, the first half was from initial contact to midstance (0–32% of the gait cycle), and the second half was from midstance to toe-off (32–65% of the gait cycle). During each half, the muscles that generated the five greatest peak accelerations were identified.

## 3. Results

Gravity's contribution to the fore–aft acceleration of the body was small relative to the net contribution of all muscles (Fig. 2). Gravity opposed progression during early stance, aided progression for most of single-limb support, and again slowed the body after heel-off (~45% of the gait cycle). For brief periods during stance, gravity accelerated the body mass center downward at values close to $-9.8\,\mathrm{m/s^2}$ (Fig. 2, 0–7% and 45–55% of the gait cycle). This indicates that, in the absence of muscle activity, the body would have been in near free-fall during these periods. When the foot was flat on the ground, the magnitude of the vertical acceleration of the body due to gravity was substantially less than $-9.8\,\mathrm{m/s^2}$, around $-7\,\mathrm{m/s^2}$ (Fig. 2). This indicates that the bones provided some amount of passive resistance to gravity. The passive support of the bones alone was not sufficient to prevent collapse; muscles were necessary to provide support.

The net influence of stance-side muscles on the body's fore–aft acceleration was to impede progression during the first half of stance and propel the body forward during the second half of stance (Fig. 3, shaded areas in left column). The five greatest peaks for individual muscle accelerations in each half of stance were due to just four muscle groups. The vasti group was responsible for most of the fore–aft deceleration during the first half of stance, along with gluteus maximus. The dorsiflexors decelerated the body during early stance, and accelerated the body forward after foot-flat (~9% of the gait cycle); soleus had the opposite effect. From 32% to 50% of the gait cycle, the anterior and posterior compartments of gluteus medius accelerated the body forward. However, gastrocnemius and soleus produced the majority of the forward acceleration during the second half of the stance phase. The combined accelerations from vasti, gluteus maximus, dorsiflexors, gluteus medius, soleus, and gastrocnemius accounted for almost all of the net fore–aft acceleration from all stance-side muscles (Fig. 3, lower left panel).

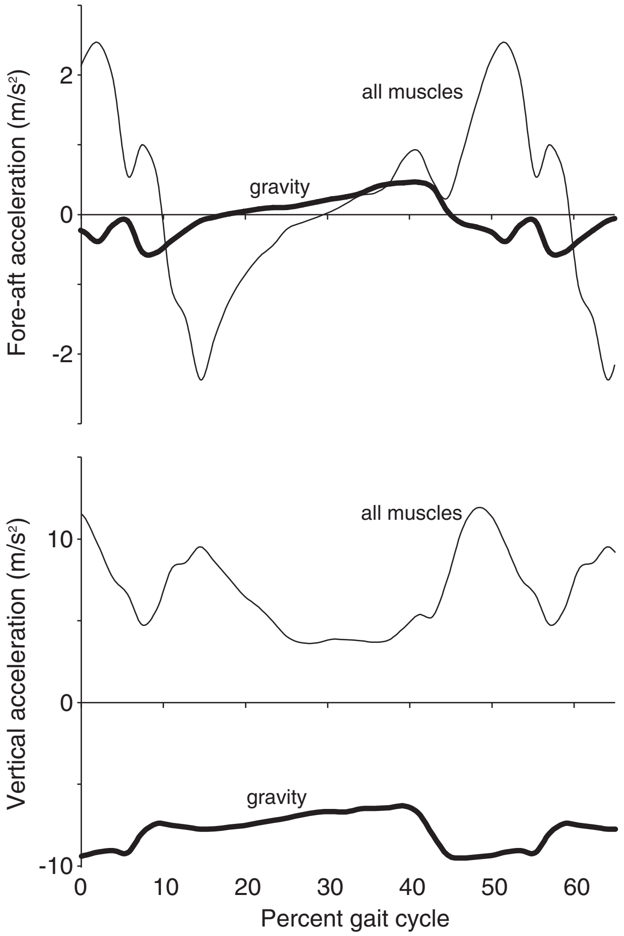

Fig. 2. Contributions of gravity to fore–aft (top) and vertical (bottom) accelerations of the body mass center compared to the contributions from all 54 muscles.

The net influence of stance-side muscles on the body's vertical acceleration was greatest near the beginning and end of single-limb support (shaded area peaks at ~15% and ~50% of the gait cycle, right column of Fig. 3). Gluteus maximus and vasti provided the most vertical support during the first half of stance, with contributions from the dorsiflexors and gluteus medius. Gluteus medius continued to accelerate the body upward through single-limb support. The second peak in vertical acceleration was due largely to gastrocnemius and soleus, with some assistance from other uniarticular plantarflexors. The combined accelerations from vasti, gluteus maximus, dorsiflexors, gluteus medius, soleus, gastrocnemius, and other plantarflexors accounted for most of the net vertical acceleration from all stance-side muscles (Fig. 3, lower right panel).

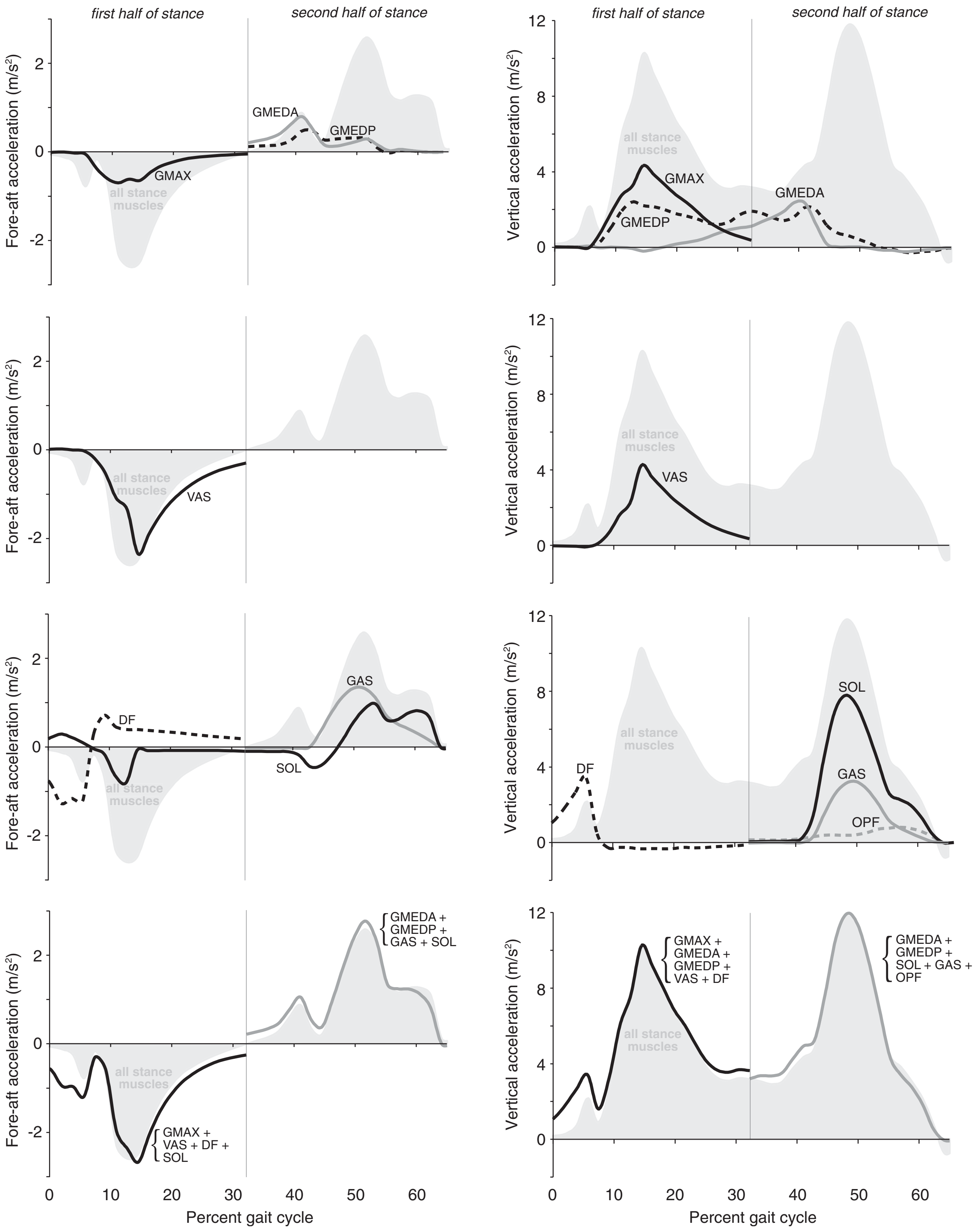

Fig. 3. Fore–aft (left column) and vertical (right column) accelerations for muscles with the greatest peak accelerations during each half of the stance phase. Hip muscles are GMAX (combined accelerations of the medial and lateral compartments of gluteus maximus), GMEDA (anterior compartment of gluteus medius), and GMEDP (posterior compartment of gluteus medius). The knee muscle group is VAS (vastus lateralis, vastus intermedius, and vastus medialis). The ankle muscles are GAS (gastrocnemius), SOL (soleus), DF (dorsiflexors), and OPF (combined accelerations of other uniarticular plantarflexors). ''All stance muscles'' (shaded area) is the combined accelerations from all 27 stance-side muscles.

In general, the same muscles that provided vertical support also modulated forward progression. Gluteus maximus and vasti made nearly equal contributions to support while slowing the body's forward acceleration during early stance (compare *y*-axis values for GMAX and VAS in Fig. 4); however, vasti's influence on forward progression was substantially larger than that of gluteus maximus (compare slopes for GMAX and VAS in Fig. 4). Gluteus medius provided support during midstance and contributed to forward acceleration during the second half of stance. Gastrocnemius and soleus both contributed to forward and vertical acceleration in late stance, but gastrocnemius generated greater forward than vertical acceleration, while soleus generated greater vertical than forward acceleration. The pattern of acceleration vectors from all stance-side muscles (bottom panel of Fig. 4) is similar to the pattern of ground reaction force vectors shown by Perry (1992).

## 4. Discussion

The model used in this analysis had over 50 musculotendon actuators, yet the fore–aft and vertical accelerations of the body during steady-state walking were largely generated by a relatively small set of muscles. This result complements previous analyses of walking based on models with very few degrees of freedom or actuators (Kuo, 2002; McGeer, 1990; Mochon and McMahon, 1980a; Pandy and Berme, 1988), which suggested that simple muscle coordination strategies are sufficient to maintain forward progression and support during walking. The use of a more complex muscle-actuated simulation provides additional insight into the specific muscles involved in these strategies. It also suggests a principle that might be deduced intuitively from a simple inverted pendulum model of stance: if a muscle provides support during the first half of stance, it will concurrently accelerate the mass center backward; if a muscle provides support during the second half of stance, it will concurrently accelerate the mass center forward. This principle applies to the actions of the plantarflexors, dorsiflexors, vasti, gluteus medius, and gluteus maximus.

It is clear that the plantarflexors are the key muscle group for generating both support and progression during late stance (Gottschall and Kram, 2003; Kepple et al., 1997; Neptune et al., 2001, 2004; Pandy, 2001; Perry, 1992; Sutherland et al., 1980; Winter, 1983). In agreement with Gottschall and Kram (2003) and Neptune et al. (2004), we found that gastrocnemius appears to be more suited to increasing walking speed than does soleus (Fig. 4, GAS vectors tilt forward more than SOL vectors). The function of the plantarflexors during the first half of stance is less clear. Neptune et al. (2004) reported that the plantarflexors, especially soleus, make large contributions to support and to the aft ground reaction force during this time. The plantarflexors did not perform such a function in our

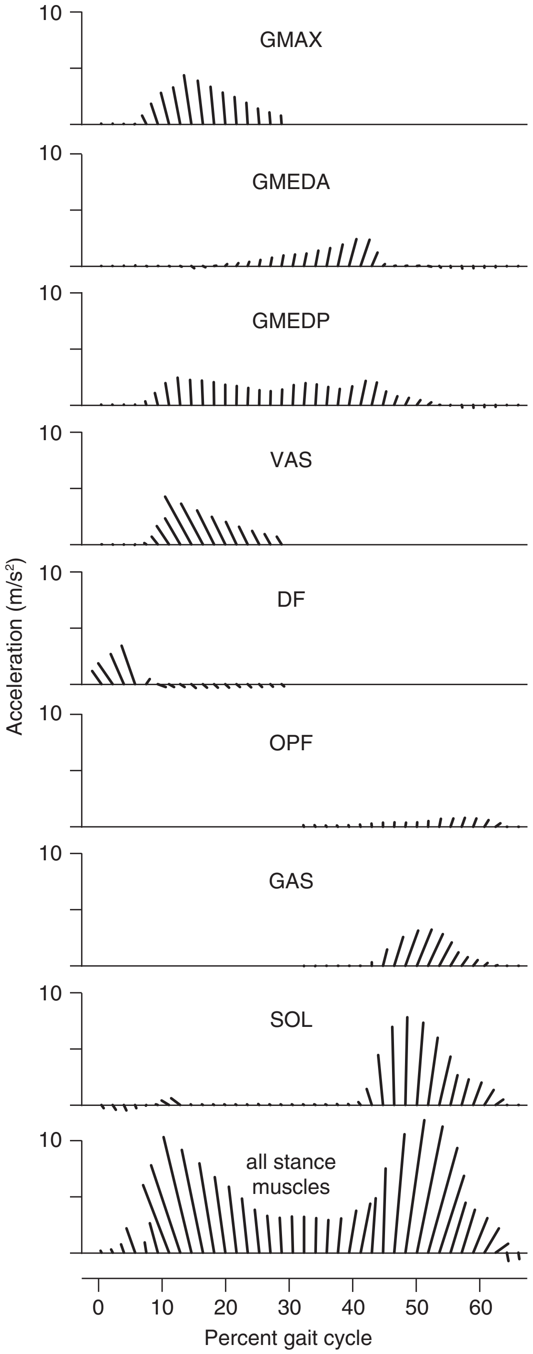

Fig. 4. The relative fore–aft and vertical accelerations for the muscles that made the largest contributions to both during stance, plotted versus percent gait cycle. Each ray represents the vector created by the fore–aft and vertical accelerations at a particular time in the gait cycle. Data for gluteus maximus, vasti, and dorsiflexors are shown only for the first half of stance because they had little influence on fore–aft or vertical acceleration after this phase. Data for gastrocnemius and for the combined accelerations of small uniarticular plantarflexors are shown only for the second half of stance because they had little influence on fore–aft or vertical acceleration prior to this. See Fig. 3 for abbreviations.

simulation. Although soleus generated force briefly before foot-flat, it was not excited during midstance in our simulation, in contrast to experimental observations (Hunt et al., 2001). Further analysis of our simulation suggested that increasing soleus excitation during midstance would have slowed the body's forward progression while contributing to vertical support, in agreement with Neptune et al. (2004).

Consistent with previous studies, we found that the hip and knee extensor muscles, primarily vasti and gluteus maximus, provide much of support in the first half of stance (Kepple et al., 1997; Neptune et al., 2004; Pandy, 2001; Winter, 1980). These muscles provide vertical support during the first half of stance as they slow forward progression.

Few simulation studies have examined the roles of muscles that act primarily outside the sagittal plane in walking. Consistent with early predictions of Mochon and McMahon (1980b), Anderson and Pandy (2003) found that gluteus medius makes large contributions to support during midstance. Since gluteus medius is a large contributor to support, our principle suggests that gluteus medius should also contribute to fore–aft acceleration. This is indeed the case. During the first half of stance, the posterior portion of gluteus medius contributed to support and slowed progression, although its influence on progression was less than other muscles (Fig. 4). In the second half of stance, both the anterior and posterior portions of gluteus medius contributed to support, and both accelerated the body mass center forward (Fig. 4). Thus, although the gluteus medius is generally not considered to be a sagittal-plane muscle, it appears to influence sagittal-plane dynamics during walking.

The dorsiflexors supported the body while slowing forward progression from initial contact to foot-flat, a combination of functions consistent with our proposed principle. That period corresponds to a well-characterized burst of activity from the pretibial muscles (Hunt et al., 2001) as they resist foot fall (Perry, 1992). After foot-flat, however, the dorsiflexors made modest reductions to support while promoting forward progression. The change in dorsiflexor contributions to support after foot-flat was also found by Anderson and Pandy (2003), who used a hard-constraint method as opposed to the perturbation analysis used here.

The hamstrings did not substantially contribute to either progression or support in our simulation. In contrast, Neptune et al. (2004) reported that hamstrings accelerated the body forward during the first half of stance and provided some support during early stance. To further investigate the function of the hamstrings group, we calculated its contributions to the body's fore–aft and vertical accelerations per unit of muscle force. This analysis showed that the hamstrings group has the potential to increase both progression and support from foot-flat to midstance (Fig. 5). From midstance to just before toe-off, hamstrings potentially reduce support while accelerating the body forward. Thus, hamstrings appears to be a unique muscle group in that it has the potential to contribute to progression throughout stance.

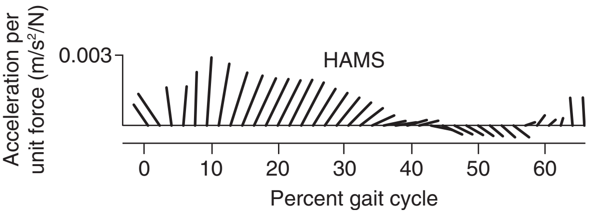

Fig. 5. The relative forward and vertical accelerations, per unit force, of the body mass center due to HAMS (semimembranosus, semitendinosus, long head of biceps femoris). Note that the accelerations per unit force are independent of the particular muscle excitation pattern that occurred during the simulation.

While our conclusions about muscle function largely agree with an intuitive interpretation of an inverted pendulum model of walking, the pattern of the fore–aft acceleration from gravity was unexpected. For an inverted pendulum, gravity would impede forward motion until the pendulum is exactly vertical (e.g., midstance), after which gravity would augment forward motion. In our more complex model, we found that gravity switched from slowing progression to assisting progression just after the end of double support, at ~18% of the gait cycle, well before midstance, which occurred at 32% of the gait cycle (Fig. 2). The anterior acceleration contributed by gravity prior to midstance must have arisen because of the dynamic interactions of the segments permitted by the joints of the leg.

Our study has important limitations. First, the results pertain only to normal walking kinematics at a single speed. Currently, little is known about the extent to which muscle function changes in movement disorders. Clarifying the influence of altered gait kinematics on muscle function will require the generation and analysis of new gait simulations. Second, from initial contact to foot-flat, the simulated vertical ground reaction force and center of mass acceleration were lower than is typically observed experimentally, allowing the body mass center to accelerate downwards during double support. This was due primarily to insufficient excitation of the dorsiflexors, which did not adequately restrain the fall of the forefoot. If the dorsiflexors had restrained the forefoot, the vertical ground reaction force would have been greater, the body's vertical acceleration would not have been negative, and the contribution of the dorsiflexors to support and progression would have been greater during double support. Third, our analysis depended on the particular excitation histories for the muscles in this simulation. While some details of our findings may change for a different set of muscle excitations (e.g., a larger contribution from soleus in midstance, or larger contributions from hamstrings and dorsiflexors in early stance), the excitation patterns used here are generally representative of normal walking.

For this study, we developed a new perturbation analysis for quantifying the contributions of muscles to the vertical and fore–aft accelerations of the body center of mass. The main shortcoming of this method is that it cannot capture high frequency detail as is possible with approaches such as the hard-constraint approach taken by Anderson and Pandy (2003). However, such high frequency accelerations cannot arise from muscles, whose physiological rate of force production is limited. So, while our perturbation analysis is not appropriate for understanding rapid changes in acceleration, as might occur from impacts, we believe it is appropriate for quantifying muscle function. Furthermore, a benefit of this analysis is that it obviates the need to explicitly decompose reaction forces. Instead, the contributions of muscles to reaction forces are computed implicitly during forward integration and automatically included in the evaluation of muscle function.

By conducting a perturbation analysis of a three-dimensional simulation, we identified individual muscles that are important for providing support and forward progression during normal walking. We found that the muscles primarily responsible for providing support are generally the same muscles primarily responsible for modulating forward progression. With the notable exception of hamstrings, if a muscle contributed to support during the first half of stance, it concurrently reduced forward progression. On the other hand, if a muscle contributed to support during the second half of stance, it concurrently increased forward progression. Future work will focus on quantifying muscle function during pathological gait.

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