

Technical note

A new parametric approach for modeling hip forces during gait

Debra E. Hurwitz^{a,*}, Kharma C. Foucher^{a,b}, Thomas P. Andriacchi^{a,c}

^aDepartment of Orthopedic Surgery, Rush Medical College, Rush-Presbyterian-St. Luke's Medical Center, Chicago, IL 60612, USA

^bDepartment of Bioengineering, University of Illinois, Chicago, IL, USA

^cDepartment of Mechanical Engineering/Functional Restoration, Stanford University, Stanford California, USA

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Abstract

An analytical parametric model was developed to estimate the natural biological variations in muscle forces and their effect on the hip forces subject only to physiological constraints and not predefined optimization criterion. Force predictions are based on the joint kinematics and kinetics of each subject, a previously published muscle model, and physiological constraints on the muscle force distributions. The model was used to determine the hip contact forces throughout the stance phase of gait of a subject with a total hip replacement (THR). The parametrically modeled peak hip force without antagonistic muscle activity varied from 2.7 to 3.2 Body Weights (mean 2.9 Body Weights), which agreed well with published in vivo measurements from instrumented THRs in other subjects. For every 10% increase in antagonistic activity, the mean peak hip force increased by 0.2 Body Weights. The parametric model allows one to examine the effect of specific muscle weaknesses or increased antagonistic muscle activity on the hip forces. The model also provides a tool for studying the effect of gait adaptations on hip forces, as predictions are based on each individual's gait data. Differences in peak forces between subjects can then be evaluated relative to the uncertainty in not knowing the precise muscle force distributions.

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1. Introduction

While hip forces have been measured in vivo with transducers in subjects with total hip replacements (THRs), they provide no information on muscle forces and are limited to evaluating only a few subjects. Analytical models, while noninvasive and easily applied to a large number of subjects, are primarily deterministic models (Anderson and Pandy, 2001; Crowninshield et al., 1978; Glitsch and Baumann, 1997; Heller et al., 2001; Komistek et al., 1998; Patriarco et al., 1981; Rohrlé et al., 1984; Seireg and Arvikar, 1975; Simonsen et al., 1995). In contrast, the parametric model presented in this paper estimates the range of hip forces that results from variations in physiologically feasible muscle activation levels, which allows one to easily estimate natural biological variations in muscle and hip forces. This parametric model allows one to systematically assess the effect of different muscle force combinations

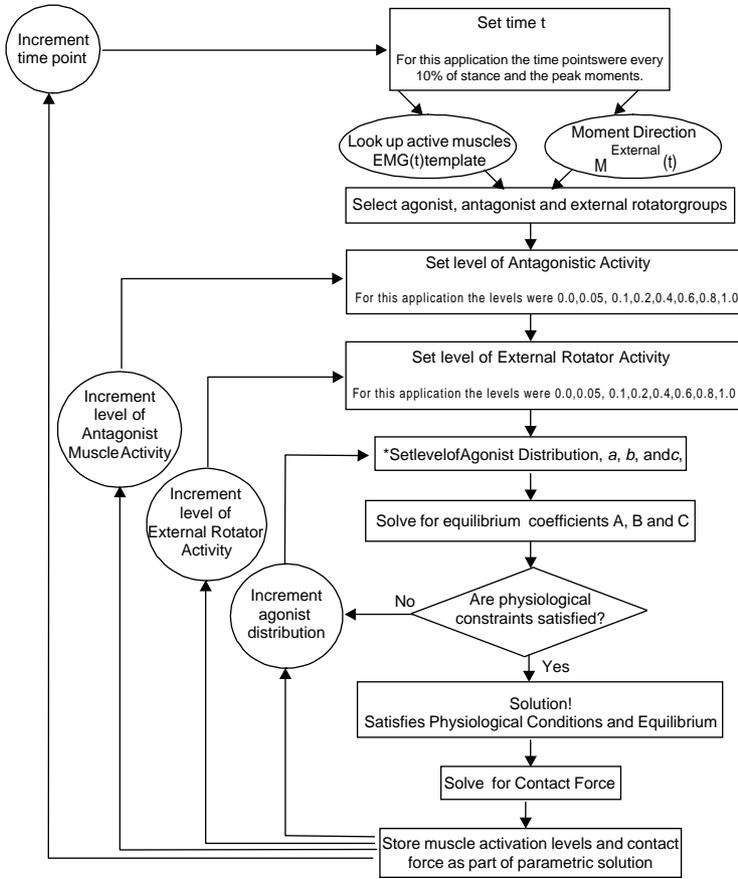
on the hip forces. The objectives of this paper are to: describe the parametric model; assess the effect of antagonistic muscle activity on the peak forces in a subject with a THR; and compare modeled forces to in vivo data collected by others (Bergmann, 2001; Bergmann et al., 2001, 1993).

2. Methods

The parametric model predicts a range of hip forces at each time point of interest that results from different combinations of physiologically feasible muscle forces (Fig. 1). The external hip moments, intersegmental forces and joint angles as calculated during gait analysis (Andriacchi et al., 1997) are inputs to the model along with the maximum isometric moment and force of each hip muscle (Delp et al., 1990) and a template from published electromyographic data (University of California, 1953; Perry, 1992; Wootten et al., 1990) identifying when each muscle is active (Fig. 2). The template is individually adjusted based on the external moment patterns of each subject.

*Corresponding author. Tel.: +1-312-942-5791; fax: +1-312-942-2101.

E-mail address: dhurwitz@rush.edu (D.E. Hurwitz).



*If the muscles in the subgroup from the adjacent list are agonist muscles at time t , the relative contribution of the subgroup is parametrically varied. For this application, scaling factors (a_i, b_i , and c_i) of 1.0, 0.8, 0.6, 0.4, 0.2, 0.15, 0.1, 0.05 and 0 were used to parametrically vary the relative contribution of each muscle subgroup.

*List of muscle or muscle subgroups scaled to create parametric range	
1	Gluteus medius (anterior portion)
2	Gluteus medius (middle portion)
3	Gluteus medius (posterior portion)
4	Gluteus medius (all)
5	Gluteus minimus (all)
6	Gluteus medius (all), gluteus minimus (all)
7	Gluteus medius (all), gluteus minimus (all), tensor fasciae latae
8	Semimembranosus
9	Semitendinosus
10	Semimembranosus, semitendinosus
11	Biceps femoris (long head)
12	Semimembranosus, semitendinosus, biceps femoris (long head)
13	Semimembranosus, semitendinosus, biceps femoris (long head), adductor magnus (all)
14	Gluteus maximus (inferior and middle portions)
15	Gluteus maximus (superior portion)
16	Gluteus maximus (all)
17	Biceps femoris (long head), gluteus maximus (superior)
18	Sartorius
19	Rectus femoris
20	Sartorius, rectus femoris
21	Sartorius, rectus femoris, iliacus
22	Tensor fasciae latae
23	Gracilis
24	Iliacus
25	Psoas
26	Iliacus, psoas
27	Adductor longus
28	Adductor brevis
29	Adductor magnus (anterior)
30	Adductor magnus (middle and posterior)
31	Adductor magnus (all)
32	Adductor longus, brevis, and magnus (all)
33	Adductor longus, brevis, and magnus (all), gracilis
34	none

Fig. 1. An overview of the parametric method for determining the muscle and hip joint forces during gait. At each point of interest a range of muscle force distributions is determined by parametrically varying the relative contribution that each agonist muscle or muscle group makes towards balancing the external hip moment and a specified level of external rotator and antagonistic muscle activity.

At each time point of interest active muscles are divided into three groups of agonist muscles, a group of antagonist muscles and a group of external rotator muscles based on the role of the muscle relative to the direction of the external hip moment present (Table 1). A full range of agonist muscle forces that balance the external moments is determined at specified levels of antagonist and external rotator activity. The range of agonist muscle forces is determined by parametrically varying the relative contribution each agonist muscle makes towards balancing the external moments.

The governing equations are:

$$\begin{aligned} \vec{M}^{\text{External}} = & A \times \sum a_i \vec{M}_i^A + B \times \sum b_i \vec{M}_i^B \\ & + C \times \sum c_i \vec{M}_i^C + D \times \sum \vec{M}_i^{\text{External Rotator}} \\ & + E \times \sum \vec{M}_i^{\text{Antagonist}} \end{aligned} \quad (1)$$

$$\begin{aligned} \vec{F}^{\text{Contact}} = & A \times \sum a_i \vec{F}_i^A + B \times \sum b_i \vec{F}_i^B \\ & + C \times \sum c_i \vec{F}_i^C + D \times \sum \vec{F}_i^{\text{External Rotator}} \\ & + E \times \sum \vec{F}_i^{\text{Antagonist}} + \vec{F}^{\text{Intersegmental}} \end{aligned} \quad (2)$$

The constraint equations are:

$$\begin{aligned} 0 \leq A \times a_i \leq 1, \quad 0 \leq B \times b_i \leq 1, \\ 0 \leq C \times c_i \leq 1 \end{aligned} \quad (3-5)$$

where

- $\vec{M}^{\text{External}}, \vec{F}^{\text{Intersegmental}}$: External hip moments and intersegmental forces, respectively, from gait analysis (input data).
- $\vec{M}_i^{A,B,C,\text{external Rotator or Antagonist}}, \vec{F}_i^{A,B,C,\text{External Rotator or Antagonist}}$: Maximum isometric

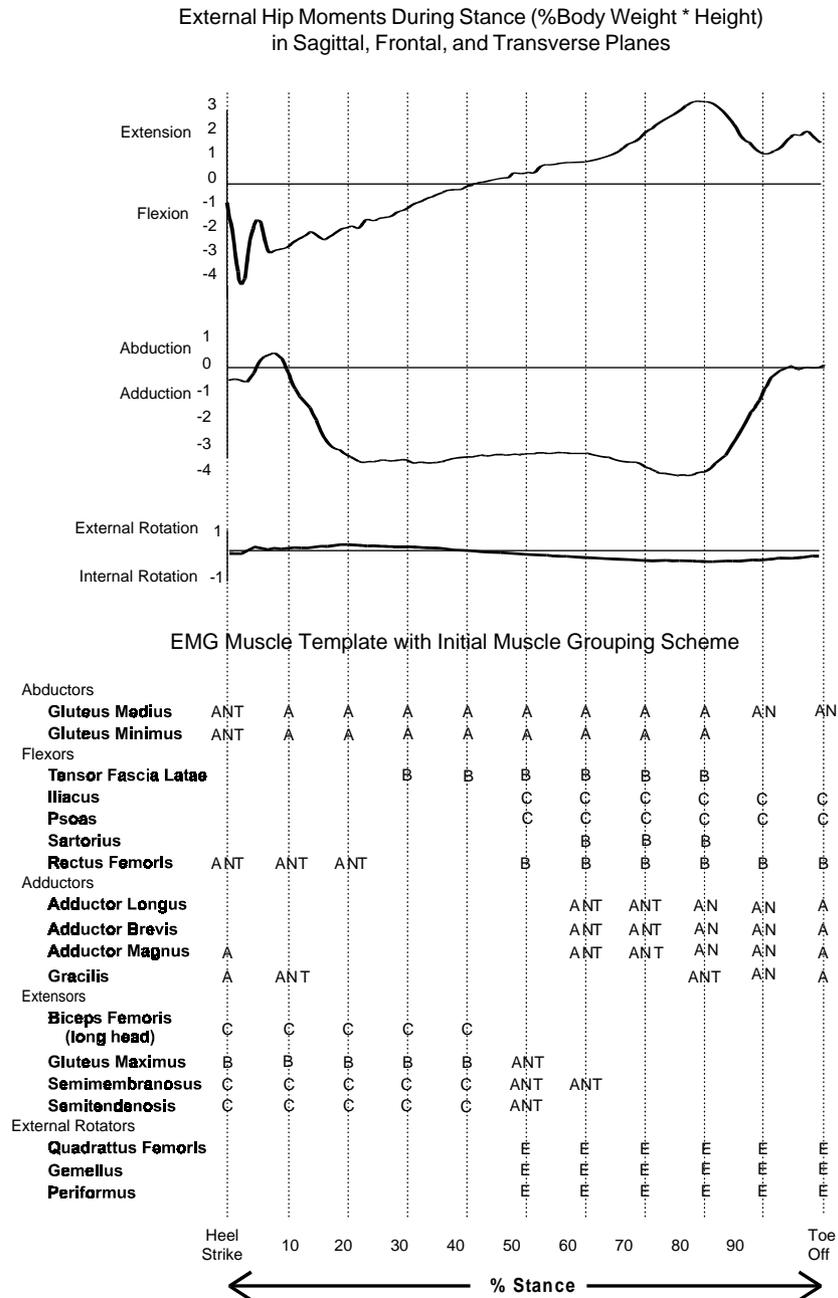


Fig. 2. An illustration of the external hip moments throughout stance along with the corresponding muscle group classification of the active muscles (i.e. *A*, *B* or *C* is agonist Muscle Group *A*, *B* or *C*, respectively; ER is an external rotator; ANT is antagonist muscle). An active muscle whose primary role is to generate a moment in the opposite direction of either the external sagittal or frontal plane moments is an agonist while an active muscle whose primary role is to generate a moment in the same direction as these external moments is an antagonist. The primary and secondary roles of the agonist muscle are then used to divide them into the three agonist groups (*A*, *B*, *C*). For example during the peak external adduction moment late in stance there is an external extension and internal rotation moment. The active muscles that are primary hip adductors or primary hip extensors are the antagonists as their primary role is to generate moments in the same direction as the external sagittal or frontal plane moments. The active muscles that are primary hip flexors or primary abductors form the three agonist groups. The primary abductors form one group of agonists, the primary flexors whose secondary role is that of abductors form a second group of agonists, while the primary flexors whose secondary role is that of adductors form a third group of agonists. Due to the lack of electromyographic information for the external rotators, they are treated as a separate group and are considered to be active only when the external transverse plane moment is an internal rotation moment.

hip moments and forces, respectively, generated by *i*th muscle of agonist group *A*, *B* or *C* or *i*th external rotator or *i*th antagonist muscle (input data).

- *D*: Selected activation level of external rotators, $0 \leq D \leq 1$ (parametrically varied).
- *E*: Selected activation level of antagonist muscle, $0 \leq E \leq 1$ (parametrically varied).

Table 1

The external hip moments and resulting muscle groupings that typically occur throughout stance. An active muscle whose primary role is to generate a moment in the opposite direction of either the external sagittal or frontal plane moments is an agonist while an active muscle whose primary role is to generate a moment in the same direction as the external sagittal or frontal plane moments is an antagonist. The primary and secondary roles of the agonist muscles are then used to divide them into the three agonist muscle groups (*A*, *B*, *C*). When the external transverse plane moment is an internal rotation moment, the external rotators are included as a separate muscle group. External rotators are treated as a separate group due to lack of electromyographic information on their activity

	External hip moment present			
	Flexion		Extension	
	External rotation		Internal rotation	
	Abduction	Adduction	Adduction	Abduction
Antagonist muscles	Primary flexor or Primary adductor	Primary flexor or Primary adductor	Primary extensor or Primary adductor	Primary extensor or Primary adductor
Agonist muscles	Primary extensor or Primary abductor	Primary extensor or Primary abductor	Primary flexor or Primary abductor	Primary flexor or Primary abductor
Group <i>A</i>	Primary adductor	Primary abductor	Primary abductor	Primary adductor
Group <i>B</i>	Primary extensor	Primary extensor	Primary flexor	Primary flexor
Group <i>C</i>	Secondary abductor	Secondary abductor	Secondary abductor	Secondary abductor
	Primary extensor	Primary extensor	Primary flexor	Primary flexor
External Rotators	Secondary abductor	Secondary abductor	Secondary abductor	Secondary abductor
	None	None	Primary external rotator	Primary external rotator

- a_i, b_i, c_i : Selected relative contribution (or scaling factor) that i th muscle of agonist group *A*, *B* or *C* contributes to maximum moment, $0 \leq a_i \leq 1$; $0 \leq b_i \leq 1$; $0 \leq c_i \leq 1$ (parametrically varied).
- *A*, *B* and *C*: Equilibrium coefficients for agonist muscle groups *A*, *B* or *C*, respectively (unknown).
- $A \times a_i, B \times b_i, C \times c_i$: Activation levels of the i th agonist muscle from groups *A*, *B* or *C*, respectively.
- $\overline{F}^{\text{Contact}}$: Hip Contact force.

The unknown equilibrium coefficients, *A*, *B* and *C*, in Eq. (1) insure that the net muscle moment is equal and opposite to the external moment. At each level of external rotator (*D*) and antagonist muscle activity (*E*), the range of agonist muscle forces is determined by parametrically varying a_i , b_i , or c_i . For example, when all muscles within agonist group *A* have the same activation level, $a_i = 1$ for all muscles in that group. However, if the relative contribution or activation level of the second muscle of group *A* is parametrically set to 60% of that of the other muscles in the group, then $a_2 = 0.6$ and $a_i = 1$ for all remaining muscles in that group. Parametrically varying a_i , b_i and c_i insures that the contact forces are determined over the full range of activation levels of each muscle ($A \times a_i, B \times b_i, C \times c_i$). This process is repeated for all agonist muscles and subgroups (Fig. 1). Constraint Eqs. (3)–(5) insure that muscle forces are physiologically feasible by checking that the maximum isometric force is not exceeded and that the role of a muscle is not reversed. Once muscle activations are determined, the corresponding contact

forces are determined (Eq. (2)). Thus, the scaling factors allow force solutions to be determined over a wide range of muscle forces and the output consists of all the physiologically feasible muscle forces and their corresponding contact forces.

The maximum isometric moment and force of each muscle at the specified limb configuration is determined from a model with a graphical interface (SIMM Software for Interactive Musculoskeletal Modeling, Evanston IL, USA). The pathways of some muscles were modified slightly from the original model (Delp et al., 1994, 1990; Delp and Maloney, 1993) to more accurately reflect the line of action of the muscle at the femoral attachment site (Hurwitz, 1994). The maximum moments generated by the hip flexors of the original model were less than experimentally measured values of others (Andersen, 1998; Cahalan et al., 1989; Inman et al., 1981). The maximum isometric force of the hip flexors were doubled so that their strength was consistent with experimentally measured values (Hurwitz, 1994).

Gait data were calculated from measurements of an optoelectronic system (CFTC—Computerized Functional Testing Corporation, Chicago IL, USA) and a multicomponent force plate (Bertec, Columbus, Ohio, USA). Inverse dynamics were used to calculate the external joint moments and intersegmental forces (Andriacchi et al., 1997). The external moments are determined from the force plate data, the location of the joint centers as determined from the marker positions in conjunction with anthropometric measurements, and the inertia forces. The external moments are equal and

opposite to the internal moment created by the muscles, soft tissues and contact forces. The complete details for calculating the external moments have been previously published (Andriacchi et al., 1997; Hurwitz et al., 1998).

Resultant hip forces are presented in detail for a representative subject with a THR (male, 73 years, 1.80 m and 119 kg, 1 year postoperative) whose peak forces when walking at about 1 m/s were close to the average values of a larger group of 17 subjects with THRs (66 ± 8 years, 1.72 ± 0.09 m, 95 ± 19 kg, 15 ± 3 months postoperative) (Foucher et al., 1999). For this subject, once antagonist muscle activity exceeded 40%, physiological solutions no longer existed at both peak forces. There were 693–20,196 different solutions at each time point depending on the level of antagonistic and external rotator activity. The average increase in the contact force for this subject due to a 10% increase in antagonistic activity was computed using linear regression. In addition to comparing the modeled forces of the representative subject to in vivo data collected from instrumented THRs of other subjects (Bergmann, 2001; Bergmann et al., 2001, 1993), the magnitude and timing of the two peak forces from the larger group of 17 subjects were statistically compared to these in vivo data.

3. Results

For the representative subject, a 10% increase in antagonist muscle force resulted in the mean peak forces increasing by 0.21 BWs. This was less than the potential variations in contact force due to the agonist muscle force distributions at a specified level of antagonist activity. For example in the absence of antagonistic muscle activity, the parametrically modeled peak forces varied by 0.5 BWs, with the first peak ranging between 2.35–2.84 BWs (mean 2.59 BWs) and the second peak ranging between 2.66 and 3.18 BWs (mean 2.87 BWs) (Fig. 3). The variation in the peak forces due to the different agonist muscle force distributions was relatively constant at each level of antagonistic activity and averaged 0.46 and 0.54 BWs for the first and second peak, respectively. Total variation in the first peak force over all possible muscle force combinations was from 2.35 BWs (minimum force with no antagonist activity) to 3.62 BWs (maximum force at 40% antagonist activity) and for the second peak, 2.66–3.98 BWs.

The shape, magnitude and timing of the parametrically predicted forces were similar to those measured in vivo in other subjects (Fig. 3). The modeled peak forces of the representative subject occurred at 20% and 81% stance, respectively. The first and second peak forces measured in vivo were between 2.4–3.6 BWs (2.9 ± 0.5 BWs) and 1.9–2.6 BWs (2.3 ± 0.3 BWs), re-

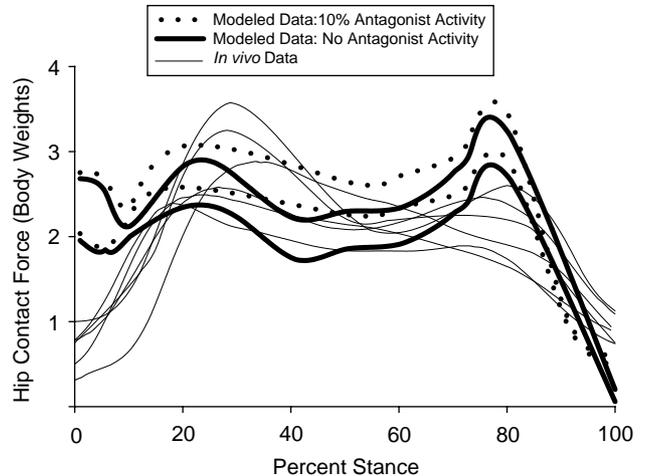


Fig. 3. The range in the parametrically modeled hip forces due to the different agonist muscle force distributions for a subject with a THR agrees well with hip contact forces measured in vivo in subjects with THRs (thin solid lines) (Bergmann, 2001; Bergmann et al., 1993). The upper thick black line represents the maximum force from the agonist muscle force distributions when there is no antagonist activity and the lower thick black line represents the minimum hip force resulting from the agonist muscle force distributions. Similarly the upper and lower dashed lines are the maximum and minimum parametrically predicted forces when there is 10% antagonist muscle activity.

spectively, and occurred between 18–33% ($26\% \pm 5\%$) and 72–80% ($75\% \pm 4\%$) stance, respectively (Bergmann, 2001; Bergmann et al., 1993).

For the group of 17 subjects with THRs, the magnitude and timing of the first peak mean force (2.8 ± 0.5 BWs, $24 \pm 7\%$ stance) and second peak mean force (2.8 ± 0.6 BWs, $75 \pm 4\%$ stance) when neglecting antagonistic muscle activity were not significantly different from these in vivo data ($p = 0.862$, $p = 0.430$, $p = 0.130$, and $p = 0.851$, respectively).

4. Discussion

The parametric model allows one to fully explore the effect of muscle force distributions on the hip forces subject only to physiological constraints and not predefined optimization criterion. Thus, the sensitivity of the hip forces to specific agonist or antagonist activation levels can be systematically evaluated.

The parametric model is ideal for studying how changes in electromyographic patterns, muscle strength, joint geometry and gait patterns affect hip forces. While for this application the generic skeleton provided with the model was used, individual variations in joint geometry and their effect on the resulting muscle moments can be easily accommodated and may be an important factor to account for when comparing forces between subjects, especially among subjects with pathologies. While for this application a generic

template indicating when a muscle was active was used, subject specific electromyographic measurements of the timing of muscle activity could be implemented to refine the template. The generic template was adjusted slightly for each individual based on the external moment pattern of the subject. For example if the transition in the external moment from flexion to extension occurred much earlier or later in stance than normally expected, the on and off times of the hip extensors and flexors in the template were adjusted accordingly. The use of a generic template even with these individual modifications would not be appropriate for subjects with external moment patterns vastly different from normal or subjects with electromyographic patterns vastly different from normal. The moment patterns of the postoperative subjects with THRs were similar to normal in terms of their shape. As with all analytical models based on gait data collected with skin based markers, errors associated with the calculation of external joint moments from not knowing the precise underlying rigid body motion of the bones exist.

The timing and magnitudes of the modeled peak forces were within the ranges reported by others from instrumented THRs. While overall modeled and in vivo data are consistent, especially at the peaks, the modeled forces at heel-strike and soon thereafter were higher for the representative subject, which most likely is a consequence of the fact that the external flexion moment at the beginning of stance for this subject was greater and earlier in stance than seen in most subjects with THRs. Thus the larger modeled forces very early in stance relative to those measured in vivo may be unique to this subject.

The parametric model allows one to estimate the potential variability in the hip forces from variations in muscle force distributions and examine, for example, the effect of specific muscle weaknesses (i.e. abductor weakness) or increased antagonistic muscle activity due to spasticity or other causes. The model also provides a tool for studying the effect of gait adaptations on hip forces, as predictions are based on each individual's gait data. Differences in peak forces between subjects can then be evaluated relative to the uncertainty in not knowing the precise muscle force distributions.

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