

MUSCULAR CONTROL OF STANCE PHASE KNEE EXTENSION DURING NORMAL WALKING: A STEP TOWARD IDENTIFYING THE CAUSES OF CROUCH GAIT

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INTRODUCTION

Crouch gait, characterized by excessive flexion of the knee during stance, is one of the most common abnormalities among children with cerebral palsy. In some cases, tight hamstrings and/or psoas muscles are presumed to contribute, and surgical lengthening of these muscles is performed (Bleck, 1987). In other cases, weak knee extensors, hip extensors, or plantarflexors are thought to be factors, and strengthening exercises or orthoses are prescribed (Gage, 1991). Unfortunately, treatment is not always successful, in part, because the mechanisms that control knee extension are not well understood, even during normal walking. The mechanisms are complex because motions of the knee are influenced not only by muscles that cross the knee, but also by muscles that span other joints (Zajac and Gordon, 1989). As a step toward rigorously evaluating the hypothesized causes of crouch gait, this study characterized the angular accelerations of the knee during normal gait induced by muscles that are common targets of treatment of cerebral palsy.

METHODS

The accelerations of the knee joint induced by gluteus maximus, vasti, hamstrings, soleus, gastrocnemius, and iliopsoas during stance were computed based on the dynamic optimization solution for normal gait solved by Anderson and Pandy (*in review*). The body was modeled as a 10-segment, 23-degree-of-freedom articulated linkage actuated by 54 muscles. The back joint and hips were modeled as ball-and-socket joints. The knees, subtalar, and metatarsal phalangeal joints were modeled as hinges. The force generating properties and path geometries of the muscles were based on

data reported by Delp et al. (1990). The interaction between each foot and the ground was modeled using five stiff spring-damper units distributed under the sole of the foot. The performance criterion for the dynamic optimization problem was to minimize the metabolic energy consumed per unit distance traveled by the center of mass. The muscle excitation patterns, limb kinematics, and ground reaction forces predicted by the solution were similar to experimental data obtained from five subjects (Anderson and Pandy, *in review*).

The contributions of individual muscles to the acceleration of the knee joint were computed using the equations of motion for the model (Zajac and Gordon, 1989):

$$\ddot{\bar{q}} = \bar{I}(\bar{q})^{-1} \cdot \{ \bar{C}(\bar{q}, \dot{\bar{q}}^2) + \bar{G}(\bar{q}) + \bar{R}(\bar{q}) \cdot \bar{f}_m + \bar{S}(\bar{q}) \cdot \bar{f}_s \} \quad (1)$$

where \bar{q} is the vector of generalized coordinates, $\bar{I}(\bar{q})^{-1}$ is the inverse of the system mass matrix, $\bar{C}(\bar{q}, \dot{\bar{q}}^2)$ is the vector of force terms arising from angular velocities, $\bar{G}(\bar{q})$ is the vector of gravitational forces, \bar{f}_m is the vector of applied muscle forces, $\bar{R}(\bar{q})$ is the muscle moment arm matrix, \bar{f}_s is the vector of ground spring forces, and $\bar{S}(\bar{q})$ is a matrix of measure numbers for \bar{f}_s (Kane and Levinson, 1985). Each muscle induced acceleration was calculated by setting all force terms in Eq. (1) to zero except for the muscle force in question, \bar{f}_{m_i} , and the corresponding portion of the ground reaction force caused by muscle i , $\bar{f}_s^{m_i}$:

$$\ddot{\bar{q}} = \bar{I}(\bar{q})^{-1} \cdot \{ \bar{R}(\bar{q}) \cdot \bar{f}_{m_i} + \bar{S}(\bar{q}) \cdot \bar{f}_s^{m_i} \} \quad (2)$$

$\bar{f}_s^{m_i}$ was computed by assuming rigid contact of the foot springs with the ground. To quantify the potential of a muscle to accelerate the knee, the induced acceleration *per unit force* was computed by setting

$\bar{f}_{m_i} = 1$, computing the corresponding $\bar{f}_s^{m_i}$, and substituting these quantities in Eq. (2).

RESULTS AND DISCUSSION

In early stance, both vasti and gluteus maximus produced substantial knee extension accelerations (Fig. 1A). In fact, per unit force, gluteus maximus had greater potential to extend the knee than vasti (Fig. 1B). These results support the hypotheses that weak knee or hip extensors could contribute to crouch gait.

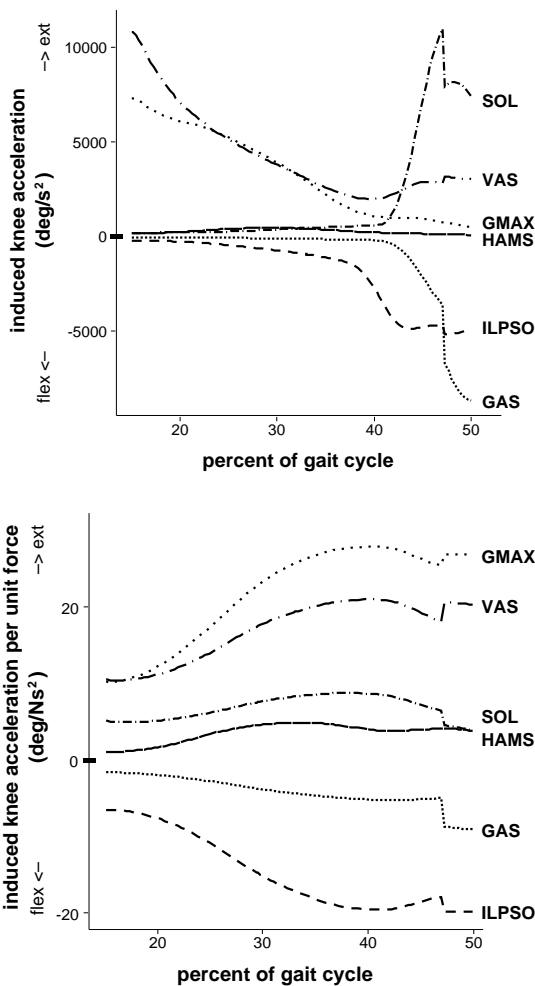


Figure 1. Angular accelerations of the knee (A) and angular accelerations per unit force (B) induced by vasti (VAS), gluteus maximus (GMAX), hamstrings (HAMS), soleus (SOL), gastrocnemius (GAS), and iliopsoas (ILPSO) during the single support phase of normal gait as computed from the dynamic optimization solution of Anderson and Pandy (*in review*).

In late stance, soleus generated large knee extension accelerations, and gastrocnemius generated large knee flexion accelerations (Fig. 1A). This suggests that decreased strength of the soleus or excessive force in gastrocnemius may contribute to reduced extension of the knee in some subjects with crouch gait. Tight iliopsoas muscles could also contribute since our analysis suggests that these muscles have the potential to flex the knee throughout stance (Fig. 1B).

Hamstrings had little effect on knee motion during stance (Fig. 1A), in part, because the forces generated by hamstrings were small. However, examination of the muscle-induced accelerations per unit force revealed that the hamstrings had the potential to *extend* the knee throughout the stance phase (Fig. 1B). This result suggests that tight or spastic hamstrings—a reputed cause of crouch gait—may *not* be the direct source of excessive knee flexion in some patients.

The non-intuitive findings, that gluteus maximus has perhaps greater potential to extend the knee than vasti and that hamstrings may actually extend the knee during gait, emphasize the need to quantify the contribution of muscles to joint motion when attempting to identify the underlying causes of movement pathologies.

REFERENCES

- Anderson FC, Pandy MG (in review). *J. Biomechanical Engineering*.
 Bleck EE (1987). *Orthopaedic Management in Cerebral Palsy*. Mac Keith Press, London.
 Delp SL et al. (1990). *IEEE Transactions on Biomedical Engineering*, **37**, 757-767.
 Gage (1991). *Gait Analysis in Cerebral Palsy*. Mac Keith Press, London.
 Kane TR, Levinson DA (1985). *Dynamics: Theory and Application*. McGraw-Hill, New York.
 Zajac FE, Gordon ME (1989). *Exercise and Sport Science Reviews*, **17**, 187-230.

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