What's Your Potential? The Influence of Joint Kinematics on a Muscle's Ability to Contribute to the Sit-to-Stand Transfer

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ABSTRACT

Induced Acceleration Analysis (IAA) estimates how muscles facilitate movement by supporting the center of mass (COM) against the force of gravity and contributing to the progression of the COM in the direction of movement. Prior IAA studies provide a baseline of how muscles facilitate activities of daily living; however, the underlying elements of IAA describing the mechanisms by which muscle forces contribute to movement are complex and not fully understood. While muscle force greatly impacts IAA, kinematics influence these forces, as well as muscle moment arms and contributions to movement. This kinematic component of IAA is important, as it has been suggested that for patients to utilize strength gains from rehabilitation, kinematic retraining may be necessary to reprogram the motor pattern. However, existing methods for estimating muscle contributions to movement require extensive time and experimental resources, making patient specific-IAA impractical in a clinical setting. Therefore, the purpose of this study is to develop an efficient method for determining how changes to lower extremity joint kinematics affect the potential of individual muscles to contribute to support during the sit-to-stand (STS) transfer. A 4 link 2-dimensional sagittal plane model with 4 rotational joints was used to assess the effect of changes to kinematics on the function of 23 lower extremity muscles during each task. Kinematics were varied at each joint throughout ranges that are commonly observed during the momentum transfer phase of the STS. For each

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kinematic position, a muscle's potential to contribute to support was estimated. Our new method predicted that positioning the feet more posteriorly (under the COM) during the STS increases the potential of the gluteus maximus, biceps femoris long head, vasti, and gastrocnemius to contribute to support. Additionally, a more anteriorly tilted pelvis increased the potential of the quadriceps muscles, but decreased the potential of posterior muscles. These results were verified by muscle potentials calculated using traditional experimental and simulation methods. This research represents the first effort to systematically evaluate how changes in joint kinematics affect muscle contributions to acceleration. The methods presented in this study demonstrate a mechanism for determining the cause of abnormal movement and for identifying potential rehabilitation techniques that leverage both kinematics and muscle strength to improve mobility.

INTRODUCTION

A tool in itself is not useful unless you understand how it works and how to use it in a practical application. Trying to hammer a nail into a piece of wood with the wrong side of the hammer is neither appropriate nor efficient. While the hammer is an extremely useful tool, if you don't know when and how to use it, it's not particularly useful to you.

Induced acceleration analysis (IAA) is a tool that has been used in biomechanics to understand how individual muscles facilitate movement by accomplishing two primary functions: providing vertical support and maintaining forward progression of the body's center of mass (COM) (Winter, 1980; Liu et al., 2006). Previous research has used IAA to determine which muscles are used to support, brake, and propel the COM during activities of daily living (ADLs) including walking at a single speed (Neptune et al., 2001; Anderson and Pandy, 2003; Liu et al., 2006), walking at a range of speeds (Liu et al., 2008), the sit-to-stand (STS) transfer

(Caruthers, E.J. et al., 2016), and climbing stairs (Lin et al., 2015). In general, these studies have found that the gluteus maximus, vasti muscle group, and the plantarflexors are the primary contributors to the acceleration of the center of mass during these tasks. However, individual muscle contribution demand differs between tasks due to differences in kinematics. The kinematics of the task determine the direction of the contributions from the individual muscles. For example, during walking (Liu et al., 2006) the gluteus maximus contributes to braking the body's center of mass during early stance, but when rising from a chair (Caruthers, E.J. et al., 2016) and ascending stairs (Lin et al., 2015) the gluteus maximus contributed to forward propulsion.

These studies provide an important baseline for understanding how healthy individuals use their muscles to facilitate movement; however, changes to the skeletal, muscular, and/or nervous systems due to aging, disease, or injury can affect how an individual moves and their ability to perform ADLs. For example, patients with knee osteoarthritis (KOA) have knee joint pain that is commonly accompanied by weakness of the quadriceps femoris muscles (Palmieri-Smith et al., 2010). Previous work revealed that KOA patients perform the STS slower than healthy controls, which may be due to inefficient knee extensor muscle function and greater antagonistic biceps femoris activation (Patsika et al., 2011). The authors suggest these muscle function adaptations may lead to the modified movement strategy of KOA patients compared to healthy controls (Patsika et al., 2011), which can include decreased distance between the heel and the body COM, increased forward lean of the trunk, and decreased average knee extension moment (Anan et al., 2015). Additionally, post-stroke patients often have limited ability to perform ADLs including the STS transfer (Cheng et al., 1998) and take longer to perform the STS (Boukadida et al., 2015). One study suggested that the muscle strength of the paretic limb

was an important determinant of the ability of post-stroke patients to complete the STS transfer (Lomaglio and Eng, 2005).

To address these mobility impairments, many rehabilitation programs apply muscle strengthening techniques, which aim to increase a muscle's peak isometric force – an important factor in determining a muscle's contribution to movement. For example, for patients with knee OA, the quadriceps are strengthened most frequently as doing so has been shown to reduce pain and improve function (Brakke et al., 2012). Although targeting muscle strength is the most intuitive solution, the mechanisms that govern force production are functionally complex, and muscle strengthening rehabilitation techniques may leave patients dissatisfied with their mobility. For example, current knee OA rehabilitation programs often leave a large number of patients, upwards of 40% in some cases, without significant improvement in short-term pain or ability to perform ADLs (Bennell et al., 2014). Similarly, combined functional strength training and conventional physical therapy did not lead to greater improvements in STS ability at 3 month follow-up than conventional physical therapy alone in hemiparetic stroke survivors (Kerr et al., 2016).

While muscle force is an important component of IAA, task-specific kinematics influence muscle forces, moment arms, and contributions to COM accelerations during ADLs. Movement pattern modification using repetitive task training, which addresses the kinematics component of IAA, has been suggested to be effective in improving STS after stroke (2010). Furthermore, Fisher et al. suggested that for patients to utilize improvements in muscle strength from exercise rehabilitation, movement retraining may be necessary to reprogram the locomotor pattern (Fisher et al., 1997). However, only a few studies have emphasized changes in both muscle forces and kinematics in analyses of muscle contributions to support and progression during ADLs and the

majority of these studies focused on gait (Higginson et al., 2006; Peterson et al., 2010; Hall et al., 2011; Steele et al., 2013; Thompson et al., 2013; Allen et al., 2014). Only one known study has analyzed muscle contributions to acceleration during STS in a population with altered kinematics and strength (Cullen, 2015). When provided, the therapeutic recommendations from the authors of these studies focus on changes to muscle strength and activity to improve gait function; however, potential changes to movement kinematics and their potential impact on muscle function as well as overall function are generally absent from these recommendations.

Determining how changes to movement patterns and muscle strength affect muscle function is a key step toward improved care for individuals with mobility limitations. IAA accounts for both kinematics and muscle forces, as well as muscle moment arms and the body's inertial properties including segment lengths, masses, and moments of inertia. While these variables in and of themselves are relatively straight forward, the interaction between kinematics, muscle forces, muscle moment arms, and the body's inertial properties is more complex. Joint kinematics determine a muscle's normalized fiber length and, therefore, influence the muscle's force generation capacity. Kinematics also affect a muscle's moment arm and, thus, influence the muscle's torque generation capacity. Moreover, the body's inertial properties affect how a muscle's torque accelerates the body's COM. These underlying mechanisms that determine how the large number of variables in the musculoskeletal system interact to produce movement have neither been fully explored nor explained in an accessible manner for everyone to use and understand IAA.

To develop targeted therapies that enable patients to take advantage of their strength gains and ultimately improve patient function, there is a need to understand these complex interactions between kinematics, muscle strength, and muscle function and how changes to one

or more variables, due to aging, disease, or rehabilitation, affect movement. Moreover, existing methods for determining muscle induced accelerations require extensive experimental resources and computational time, hindering the application of important insights from IAA in a clinical setting. In order to improve the identification of appropriate rehabilitation targets for improving a patient's ability to complete ADLs, a straightforward and time efficient method of understanding how possible modifications to kinematics and muscle strength could impact motion is necessary.

Therefore, the purpose of this study is to develop an efficient method for determining how changes to lower extremity joint kinematics affect the potential of individual muscles to contribute to support during the STS transfer. We hypothesize the new method will predict actual kinematic adaptations used by pathologic populations to complete the STS and that these kinematic adaptations will take advantage of the available muscle strength of the respective population. This research represents the first effort to deconstruct muscle induced accelerations to explore how the components of IAA, specifically joint kinematics, affect the potential of a muscle to contribute to COM accelerations.

METHODS

Model

A four link 2-dimensional sagittal plane model (2D model; Figure 1A) with four revolute joints was used to assess the effect of changes to kinematics on the function of 23 lower extremity muscles. The foot was modeled as a triangle, such that the three sides of the triangle were defined by vectors between three vertices located at the posterior calcaneus (C), the distal toe (T), and the ankle joint (A). A point, B, was defined along line CT such that the vector from B to A (line BA) was perpendicular to the base of the foot. For all kinematic positions, the

positive X axis pointed anteriorly and the positive Y axis pointed superiorly; however, the origin of the model depended on the task being analyzed. Link 0, formed by a rigid connection between CT and BA, represented the foot, link 1 represented the shank, link 2 represented the thigh, and link 3 represented a lumped model of the head, arms, and torso (HAT). The four revolute joints were defined between the ground and foot (joint 0), foot and shank (joint 1), shank and thigh (joint 2), and thigh and HAT (joint 3). Four generalized coordinates, θ_0, θ_1, θ_2, and θ_3, defined the degrees of freedom of the model. θ_0 was defined as the angle between the X axis and CT. Given a 90^o counterclockwise rotation from CT to OA, θ_1 was defined from OA to link 1. θ_2 was defined from the line of action of link 1 to link 2. θ_3 was defined from the line of action of link 2 to link 3. An additional angle, θ_c, was defined from the base of the foot to the line of action of the vector from the origin to the center of mass of the foot, r_0 (Figure 1A). The masses, lengths, center of mass (COM) locations, and moments of inertia of the segments were defined based on a generic 12 segment, 23 degree of freedom musculoskeletal (MSK; Figure 1B) model (Delp, 2001). Muscle lengths and moment arms were determined for each of the muscles using the muscle properties defined in the MSK model.

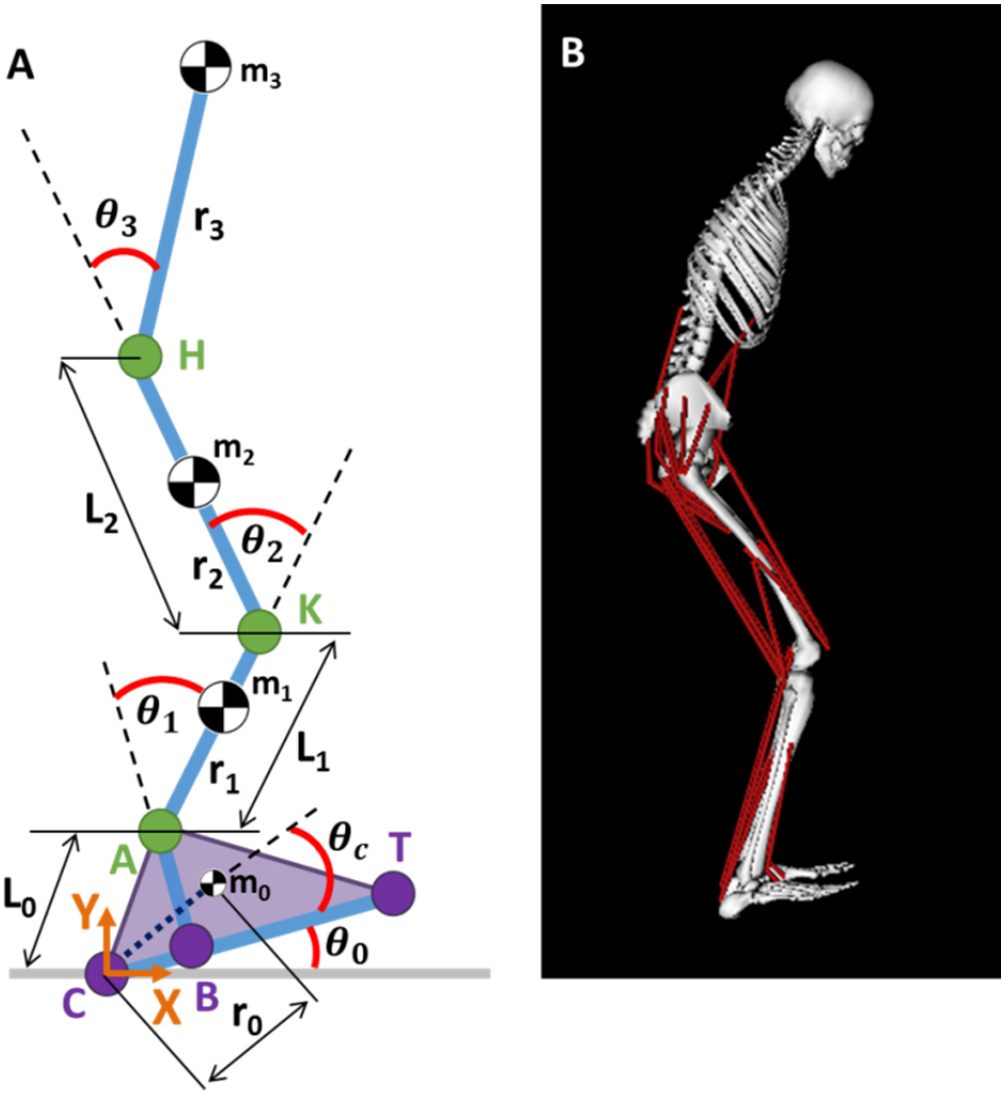

Figure 1: A) 2D Model with the origin at C and B) MSK Model

The distances, r_i, between the joint center and the COM of the segments, and the segment lengths, L_i, were determined from the MSK model. The masses of the torso and pelvis from the MSK model were lumped together in the HAT segment of the 2D model. Similarly, the mass of the foot in the 2D model was calculated as the sum of the masses of the talus, calcaneus, and toes in the MSK model. The COM of the HAT and the COM of the foot were calculated by dividing the sum of the product of the individual masses of each body and their COM locations by the total mass. The moments of inertia of the HAT and the foot were determined using the parallel axis theorem.

Calculation of Individual Muscle Potential to Contribute to COM Acceleration

Based on Lagrangian dynamics, the equations of motion of the model are defined by

$$[M]\ddot{q} = G(q) + V(q, \dot{q}) + S(q, \dot{q}) + [R]f, \qquad \text{(Equation 1)}$$

where M is the mass matrix, q are the generalized coordinates, G is the gravitational force, V is the force due to Coriolis and centrifugal effects, S accounts for intersegmental forces, and f contains the muscle forces transformed to a generalized force by R, containing the muscle moment arms. The induced acceleration of any force acting on the system is

$$\ddot{q}_i = [M]^{-1}\{F_i\}, \qquad \text{(Equation 2)}$$

where F_i is the contribution of any force (gravitation, Coriolis, contact, or muscle) to the acceleration. To determine the acceleration solely due to the application of a muscle force (muscle induced acceleration), $G(q)$, $V(q, \dot{q})$, and $S(q, \dot{q})$ can be dropped from the equation such that the equations of motion can be simplified to

$$\ddot{q}_m = [M]^{-1}[R]f_m. \qquad \text{(Equation 3)}$$

In Equation 3, $[M]$ is an *n x n* matrix, $[R]$ is an *n x m* matrix and $[f]$ is an *m x 1* matrix, where *n* is the number of coordinates in the model and *m* is the number of muscles in the model.

A muscle's potential to contribute to the support and progression of the body COM represents a muscle's contribution to acceleration per Newton of force. In this study, individual muscle potentials were calculated as

$$Potential = [M]^{-1}[R], \quad \text{(Equation 4)}$$

where $[M]$ was a *4 x 4* matrix (*n* = 4 generalized coordinates) and $[R]$ was a *4 x 23* matrix (*m* = 23 muscles).

Derivation of the Mass Matrix

The mass matrix of the model was derived for the 2D model with four revolute joints using the Lagrange formulation for defining the equations of motion (Craig, 2005). The mass matrix, [M], was obtained by

$$[M] = \sum_{i=0}^{3} m_i J_{v_i}^T J_{v_i} + J_{\omega_i}^T {}^C I_i J_{\omega_i}, \quad \text{(Equation 5)}$$

where m_i is the mass of link i, ${}^C I_i$ is the inertial tensor of link i, $J_{v_i}$is the Jacobian matrix corresponding to the linear motion of the COM of link i, $J_{\omega_i}$is the Jacobian matrix corresponding to the angular motion of link i, and $J_{v_i}^T$ and $J_{\omega_i}^T$ are the transpose of J_{v_i} and J_{ω_i}, respectively.

The Jacobian matrix corresponding to the linear motion of the COM of link 0 (${}^0J_{v_0}$) was obtained by direct differentiation of the position vector of the COM of link 0 (${}^0p_{C_0}$)

$${}^0p_{C_0} = \begin{bmatrix} r_0 \mathrm{c}\theta_{0c} \\ r_0 \, \mathrm{s}\, \theta_{0c} \end{bmatrix}, \quad \text{(Equation 6)}$$

where $\mathrm{c}\theta_{0c} = \cos(\theta_0 + \theta_c)$ and $\mathrm{s}\theta_{0c} = \sin(\theta_0 + \theta_c)$, which gives, in frame {0},

$$ {}^{0}J_{v_0} = \begin{bmatrix} -r_0 \mathrm{s}\theta_{0c} & 0 & 0 & 0 \\ r_0 \mathrm{c}\theta_{0c} & 0 & 0 & 0 \end{bmatrix}. \quad \text{(Equation 7)} $$

Similarly, the Jacobian matrix corresponding to the linear motion of the COM of link 3 (${}^{0}J_{v_3}$) was obtained by direct differentiation of the position vector of the COM of link 3 (${}^{0}p_{C_3}$)

$$ {}^{0}p_{C_3} = \begin{bmatrix} L_0\, c\,\theta_{0+90} + L_1\, c\,\theta_{01+90} + L_2\, c\,\theta_{012+90} + r_3\, c\,\theta_{0123+90} \\ L_0\, s\,\theta_{0+90} + L_1\, s\,\theta_{01+90} + L_2\, s\,\theta_{012+90} + r_3\, s\,\theta_{0123+90} \end{bmatrix}, \quad \text{(Equation 8)} $$

where $\mathrm{c}\theta_{0123+90} = \cos(\theta_0 + \theta_1 + \theta_2 + \theta_3 + 90)$ and $\mathrm{s}\,\theta_{0123+90} = \sin(\theta_0 + \theta_1 + \theta_2 + \theta_3 + 90)$, which gives, in frame {0},

$$ {}^{0}J_{v_3} = $$

$$ \begin{bmatrix} -L_0\,\mathrm{s}\,\theta_{0+90} - L_1\,\mathrm{s}\,\theta_{01+90} - L_2\,\mathrm{s}\,\theta_{012+90} - r_3\,\mathrm{s}\,\theta_{0123+90} & -L_1\,\mathrm{s}\,\theta_{01+90} - L_2\,\mathrm{s}\,\theta_{012+90} - r_3\,\mathrm{s}\,\theta_{0123+90} \\ L_0\,\mathrm{c}\,\theta_{0+90} + L_1\,\mathrm{c}\,\theta_{01+90} + L_2\,\mathrm{c}\,\theta_{012+90} + r_3\,\mathrm{c}\,\theta_{0123+90} & L_1\,\mathrm{c}\,\theta_{01+90} + L_2\,\mathrm{c}\,\theta_{012+90} + r_3\,\mathrm{c}\,\theta_{0123+90} \end{bmatrix} $$

$$ \begin{matrix} -L_2\,\mathrm{s}\,\theta_{012+90} - r_3\,\mathrm{s}\,\theta_{0123+90} & -r_3\,\mathrm{s}\,\theta_{0123+90} \\ L_2\,\mathrm{c}\,\theta_{012+90} + r_3\,\mathrm{c}\,\theta_{0123+90} & r_3\,\mathrm{c}\,\theta_{0123+90} \end{matrix} \Big] \quad \text{(Equation 9)} $$

The Jacobian matrix corresponding to the angular motion of the COM of link 0 (${}^{0}J_{\omega_0}$) was obtained by

$$ J_{\omega_0} = [\bar{\epsilon}_0 \boldsymbol{z_0} \quad \mathbf{0} \quad \mathbf{0} \quad \mathbf{0}]. \quad \text{(Equation 10)} $$

In frame {0}, this matrix is

$$ {}^{0}J_{\omega_0} = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 1 & 0 & 0 & 0 \end{bmatrix}. \quad \text{(Equation 11)} $$

Similarly, the Jacobian matrix corresponding to the angular motion of the COM of link 3 (${}^{0}J_{\omega_3}$) was obtained by

$$ J_{\omega_3} = [\bar{\epsilon}_0 \boldsymbol{z_0} \quad \bar{\epsilon}_1 \boldsymbol{z_1} \quad \bar{\epsilon}_2 \boldsymbol{z_2} \quad \bar{\epsilon}_3 \boldsymbol{z_3}]. \quad \text{(Equation 12)} $$

In frame {0}, this matrix is

$$ {}^{0}J_{\omega_3} = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 1 & 1 & 1 & 1 \end{bmatrix}. \quad \text{(Equation 13)} $$

Transformation of Potentials from Joint Space to Task Space

Equation 4 gives a *4 x 23* matrix containing the potential of each muscle to accelerate each of the four generalized coordinates, θ_{0-3}. To determine the individual muscle contributions to progression and support, defined along the X and Y Cartesian axes, respectively, the potentials were transformed from joint space to the Cartesian task space. The relationship between joint space (θ) and Cartesian space (X) can be defined using the definition of the Jacobian,

$$ \dot{X} = J\dot{\theta}. \quad \text{(Equation 14)} $$

Differentiating Equation 14, the Cartesian acceleration is calculated as,

$$ \ddot{X} = j\dot{\theta} + J\ddot{\theta}. \quad \text{(Equation 15)} $$

The potential of the muscles to contribute to joint accelerations were calculated under static conditions; therefore, the angular velocity of each joint angle is zero, giving,

$$ \ddot{X} = J\ddot{\theta}, \quad \text{(Equation 16)} $$

which was used to calculate the individual muscle potentials to contribute to progression and support for a given kinematic state.

Kinematic States

For each kinematic state, muscle moment arms were determined in OpenSim by converting the joint positions of the kinematic state from the 2D model joint angle definition to the OpenSim model joint angle definition using the following equations:

$$ \theta_{ankle} = -\theta_1 \quad \text{(Equation 17)} $$

$$\theta_{knee} = -\theta_2 \quad \text{(Equation 18)}$$

$$\theta_{hip} = \theta_2 + \theta_1 + \theta_0 - \theta_{pelvis} \quad \text{(Equation 19)}$$

$$\theta_{lumbar} = \theta_{hip} + \theta_3 \quad \text{(Equation 20)}$$

where θ_{ankle}, θ_{knee}, θ_{hip}, θ_{lumbar}, and θ_{pelvis} are the joint positions in the MSK model and θ_0, θ_1, θ_2, and θ_3 are the joint positions in the 2D model.

The initial kinematic position of the 2D model was $\theta_0 = 0^o$, $\theta_1 = 0^o$, $\theta_2 = 90^o$, and $\theta_3 = -90^o$ (Figure 2). Except for θ_0, these angles and the pelvic tilt angle of the MSK model, were adjusted in 5^o increments within specified ranges (Table 1) defined for momentum transfer phase of the STS (Schenkman et al., 1990). The kinematic states were constrained such that the foot remained flat on the floor ($\theta_0 = 0^o$ for all trials) and the lumbar angle of the MSK model was restricted to no more than 90^o of flexion. Given the joint angle ranges and constraints, we calculated individual muscle potentials for support and progression for a total of 9,025 unique momentum transfer phase states.

Table 1: Joint Angle (^{o}) Ranges for STS Trials*

Model	Angle	**Momentum Transfer Phase**	
		Min	Max
MSK Model	Ankle	0	40
	Knee	-100	-75
	Hip	25	130
	Lumbar	-90	70
	Pelvic Tilt	-30	10
2D Model	Theta 0	0	0
	Theta 1	-40	0
	Theta 2	75	100
	Theta 3	-150	-60

*Positive angles in MSK Model indicate ankle dorsiflexion, knee extension, hip flexion, lumbar extension, and posterior pelvic tilt

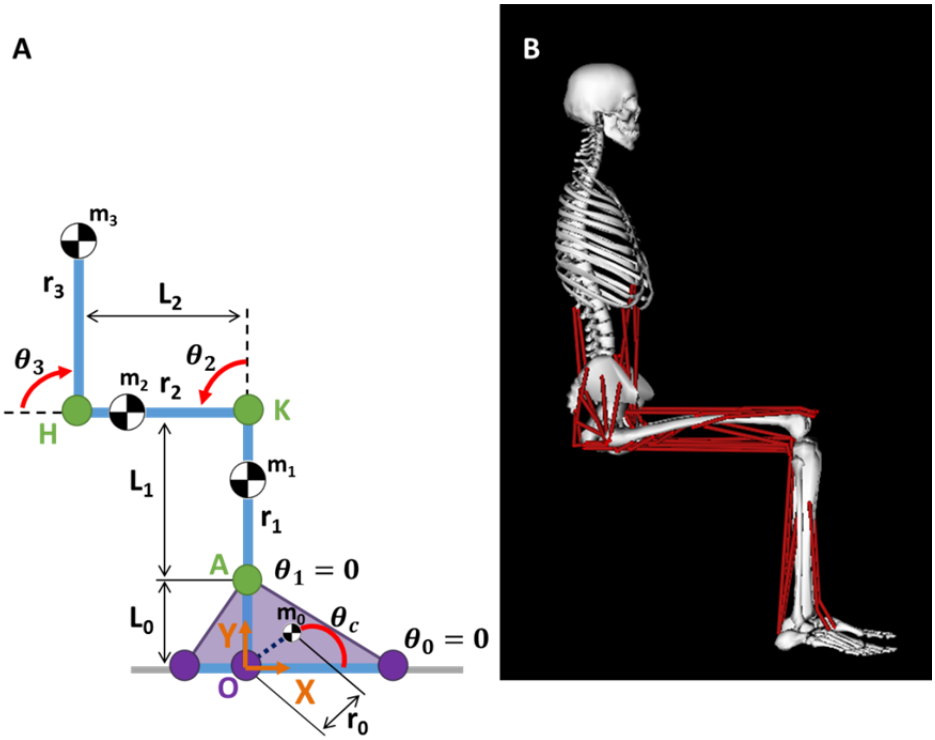

Figure 2: A) 2D Model with the origin at O and B) MSK Model in the initial kinematic position

Analysis

The time required to determine the muscle potentials for a single state was measured to estimate the computational time of the presented methods.

Based on previous research, the gluteus maximus and vasti muscles are the primary contributors to support during the momentum transfer phase of STS and the joint torques produced by these muscles, hip extension and knee extension, respectively, peak during the momentum transfer phase (Caruthers, E. J. et al., 2016). Therefore, the support potentials of 5 muscles/muscle groups crossing the knee and/or hip were chosen for further analysis: gluteus maximus, the vasti, the long head of biceps femoris, rectus femoris, and gastrocnemius. The muscle potentials predicted by the presented (new) method were verified by potentials estimated

using experimental 3D motion capture, modeling, and simulation (traditional) methods (Cullen, 2015).

RESULTS

Computational Time

For a single kinematic state, the new method calculated the support potentials of 23 muscles in 6-10s using sagittal plane joint angles. The typical frame rate of a video camera commonly used in a clinical setting to capture 2D joint angles is 60 frames (or states) per second and the average STS trial takes 1 second to complete; thus, muscle potentials could be calculated for an entire trial in 10 minutes or less.

Kinematic Modifications Affect Support Potentials

Pelvic tilt angle and foot position generally had the greatest effect on muscle potentials for support (Table 2). Support potentials were generally consistent across lumbar flexion angles for a given pelvic angle and foot position. The new method predicted that the hip extension muscles, gluteus maximus and the long head of the biceps femoris, would have greater potential for support with a more posteriorly tilted pelvis and a more posterior foot position. In contrast, the new method predicted rectus femoris, a hip flexor, would have a greater potential for support with a more anteriorly tilted pelvis and a more anterior foot position. Conversely, a more anterior foot position was predicted to increase vasti potential for support. The predicted support potential of gastrocnemius, an antagonist of the vasti and rectus femoris, was greater with a more posterior foot position. Both vasti and gastrocnemius were not greatly affected by pelvic tilt angle.

Table 2: Support potentials' relationship to pelvic tilt angle and foot position

	Agonist or Antagonist	Muscle	Increased Potential Associated With	
			Pelvic Tilt	Foot Position
Hip Extension	Agonist	Gluteus Maximus	Posterior	Posterior
		Biceps Femoris (Long Head)	Posterior	Posterior
	Antagonist	Rectus Femoris	Anterior	Anterior
Knee Extension	Agonist	Rectus Femoris	Anterior	Anterior
		Vasti	Neutral	Posterior
	Antagonist	Biceps Femoris (Long Head)	Posterior	Posterior
		Gastrocnemius	Neutral	Posterior

Predictive Capability

Consider a KOA patient with confirmed quadriceps muscle weakness. To maximize the potential of the quadriceps to contribute to support and decrease the force requirement, the new method predicts that with increasing anterior pelvic tilt the rectus femoris (and vasti, to a much smaller extent) has increasing potential for support (Figure 3). This is contrast to rectus femoris' potential for support in a more neutral pelvis position, in which it works with gravity to accelerate the COM downward. However, the new method also predicted the support potential of gluteus maximus and the long head of the biceps femoris decreases with increasing anterior pelvic tilt.

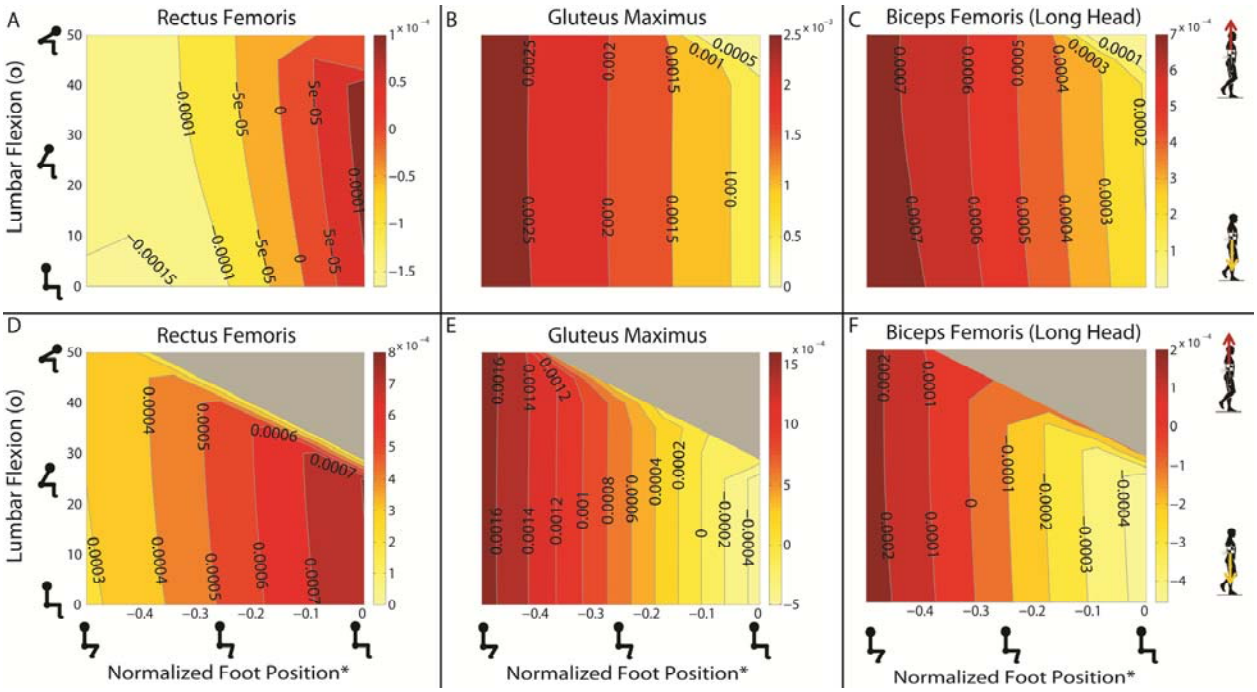

Figure 3: Rectus femoris (A,D), gluteus maximus (B, F), and the long head of the biceps femoris (C,F) support potentials at a range of kinematic states for A-C) 15^{o} and D-F) 30^{o} anterior pelvic tilt. The gray area indicates no potentials were calculated at those states due to a constraint violation (lumbar flexion angle greater than 90^{o}). *Foot position normalized to leg length.

A study comparing STS kinematics, kinetics, and muscle contributions to acceleration in KOA patients and healthy young adults (YA) (Cullen, 2015) verified the predicted relationships between kinematics and muscle potential for support. The KOA patients had greater rectus femoris support potential than YA (Figure 4A) with greater anterior pelvic tilt (KOA: 33.9^{o} ± 14.0^{o} m/Ns^{2} YA: 15.2^{o} ± 9.5^{o} m/Ns^{2}) and a slightly more posterior foot position (normalized to leg length; KOA: -0.31 ± 0.15; YA: -0.29 ± 0.11) (Cullen, 2015). As predicted, the increased anterior pelvic tilt angle of the KOA patients also led to smaller support potentials for gluteus maximus (Figure 4B) and the long head of the biceps femoris (Figure 4C) compared to the potentials of those muscles in YA.

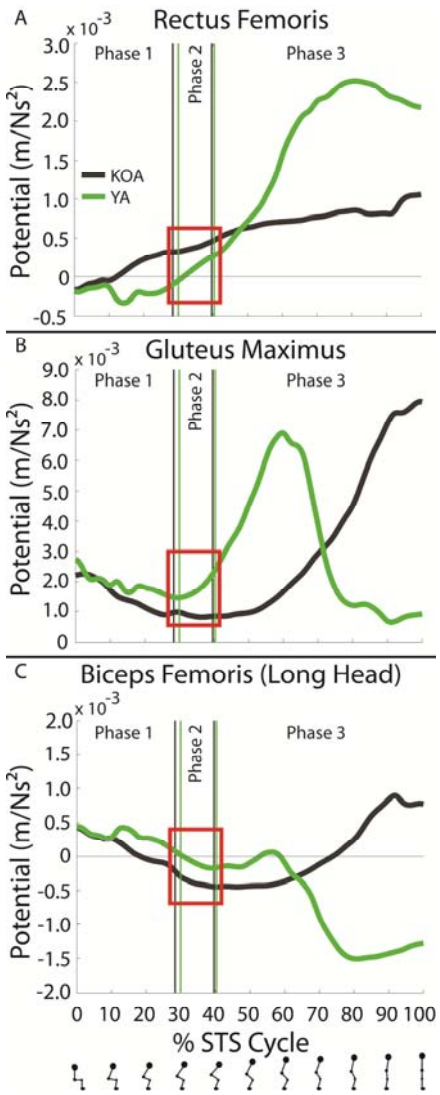

Figure 4: KOA and YA support potentials from A) rectus femoris, B) gluteus maximus, and C) the long head of the biceps femoris across one STS cycle (Cullen, 2015). The red boxes encompass data from the momentum transfer phase.

DISCUSSION

The new method predicted actual kinematic adaptations used by KOA patients to complete the STS, as confirmed by traditional simulation methods (Cullen, 2015), which often require at least 4-6 hours to obtain IAA results for a single trial. This computational time is significantly decreased by the new method, which can calculate muscle potentials in just 6-10s per kinematic position. Moreover, by eliminating the need for ground reaction force data, which

is typically required by traditional methods to perform IAA, the new method relies solely on data that can be captured in a clinical setting: sagittal plane joint angles and body anthropometrics.

Additionally, with the new method it is possible to explore a range of kinematic positions that may improve patient mobility and suggest additional strategies that could be targeted in rehabilitation. For example, KOA patients could improve overall muscle contributions to support by decreasing their anterior pelvic tilt angle by 5^{o} to increase both gluteus maximus and the long head of the biceps femoris support potentials by 50-100%. Additionally, the gluteus maximus has a larger potential for support than rectus femoris and, therefore, may be an ideal target for a muscle strengthening protocol because increasing the maximum force a muscle can produce will increase the muscle's maximum possible contribution to acceleration.

In conclusion, this work demonstrates an efficient method to systematically evaluate the effects of kinematic changes on muscle potentials, predict possible sources of pathologic movement, and identify rehabilitation strategies that leverage kinematics and muscle strength to improve mobility. The new method overcomes the time and resource limitations preventing the use of IAA in a clinical setting to determine how patients may benefit from both kinematic and muscle strength gains. Future work should investigate how altered kinematics affect muscle potentials to contribute to progression during the STS and the effect of kinematics on muscle potentials in other activities of daily living, such as gait and stair climbing.

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