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Compensatory strategies during normal walking in response to muscle weakness and increased hip joint stiffness

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Abstract

Compared to young adults, older adults exhibit a slower walking speed, smaller step length, shorter swing phase time and decreased range of motion in their lower extremity joints. The underlying mechanisms causing these gait adaptations is not well understood, with various musculoskeletal parameters being put forth as contributing factors, including increased joint stiffness and decreased isometric muscle strength. The objective of this study was to identify the necessary compensatory mechanisms to overcome such musculoskeletal deficits and regain a normal walking pattern. Understanding these mechanisms has important implications for designing effective rehabilitation interventions for older adults that target specific muscle groups and properties (e.g., isometric strength versus joint stiffness) to improve gait performance. Muscle-actuated forward dynamics simulations of normal walking were analyzed to quantify compensatory mechanisms in the presence of muscle weakness in specific muscle groups and increased hip joint stiffness. Of particular importance were the compensatory mechanisms provided by the plantar flexors, which were shown to be able to compensate for many musculoskeletal deficits, including diminished muscle strength in the hip and knee flexors and extensors and increased hip joint stiffness. This importance was further highlighted when a normal walking pattern could not be achieved through compensatory action of other muscle groups when the uniarticular and biarticular plantar flexor strength was decreased as a group. Thus, rehabilitation or preventative exercise programs may consider focusing on increasing or maintaining plantar flexor strength, which appears critical to maintaining normal walking mechanics.

Keywords: Gait; Muscle work; Musculoskeletal model; Forward dynamics simulation

1. Introduction

Falls are the leading cause of accidental death in older adults and lead to approximately 185,000 hip fractures each year [1,2]. Fallers tend to have a slower self-selected walking speed compared to non-fallers [3] and are more susceptible to falling laterally, which increases their potential for hip fractures [4]. Compared to young adults, older adults exhibit a slower preferred walking speed, smaller step length, shorter swing phase time and decreased range of motion in their lower extremity joints [5–8]. The underlying mechanisms causing these gait adaptations, however, are not well understood, with various musculoskeletal parameters being put forth as contributing factors including increased joint stiffness and decreased isometric strength [5–7,9–11].

Decreased isometric strength appears to be one of the more important musculoskeletal deficits that limits walking speed in older adults [6,7]. Judge et al. [7] showed that ankle plantar flexor work in older adults remains relatively constant at increasing speeds, which is in contrast to young adults that systematically increase their ankle work output with walking speed [8]. These results suggest that reduced ankle plantar flexor strength may be a limiting factor in achieving higher walking speeds [7]. Other studies have suggested that decreased hip extensor strength may be the limiting factor. Burnfield et al. [6] showed that hip extensor torque is an independent predictor of walking speed, stride length and cadence in older adults, and suggested that

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increasing the torque capacity of the hip extensor muscles may be an effective intervention to improve walking performance.

The decreased stride length and range of hip extension observed in older adults during walking may be the result of increased hip flexor stiffness (or even contractures in extreme cases), suggesting that passive, rather than active alterations at the hip joint can cause reduced gait speed in older adults [9,11]. The development of hip flexor stiffness or contractures is a common musculoskeletal disorder in older adults due to decreased physical activity [9]. The necessity of compensatory strategies to overcome hip flexor stiffness is uncertain, as the increased joint stiffness has the potential to store and release elastic energy in the gait cycle that may provide a locomotor advantage [12].

Due to muscle redundancy, various neuromotor strategies may exist to compensate for decreased muscle strength and joint stiffness. In addition, dynamic coupling allows muscles to accelerate joints and segments they do not span and biarticular muscles can accelerate joints in the opposite direction to their anatomical classification [13]. Thus, identifying the necessary compensatory strategies for specific musculoskeletal deficits is extremely difficult and often counter intuitive. Understanding these compensatory strategies has important implications for designing effective rehabilitation interventions for older adults that target specific muscle groups and properties (e.g., isometric strength versus joint stiffness) to improve gait performance.

Therefore, the overall objective of this study was to use forward dynamics simulations and dynamic optimization to analyze walking at a nominal speed and identify the necessary compensatory mechanisms to overcome specific musculoskeletal deficits and regain a normal walking pattern. Forward dynamics simulations provide the unique advantage that individual musculoskeletal properties within the model can be varied in isolation, which allows one to identify causal relationships between the property of interest and the task performance. The specific properties of interest were individual muscle maximum isometric strength and passive hip joint stiffness.

2. Methods

2.1. Musculoskeletal model

A bipedal musculoskeletal model was generated using SIMM (MusculoGraphics Inc.), which consisted of rigid segments representing the trunk and two legs (Fig. 1). The trunk was assigned the combined mass and inertial characteristics of the head, arms, torso and pelvis, and each leg consisted of a thigh, shank, patella and foot. Regression equations were used to determine the inertial properties of the body segments [14,15], and the musculoskeletal geometry and muscle lines of action were based on the work of Delp et al. [16]. Contact between the foot and the ground was modeled using 30 independent viscoelastic elements with Coulomb friction attached to the bottom of each foot segment [17]. The model's equations-of-motion were derived using SD/FAST (Parametric Technology Corp.), and a forward dynamics simulation was produced using Dynamics Pipeline (MusculoGraphics Inc.).

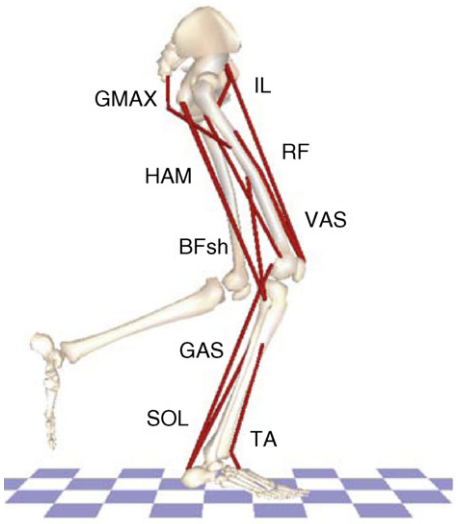

Fig. 1. The 2D musculoskeletal model consisting of the HAT (head, arms, torso and pelvis) and right and left legs (femur, tibia, patella and foot). Nine muscle groups per leg were used to drive the model including: GMAX (gluteus maximus, adductor magnus), IL (iliacus, psoas), HAM (biceps femoris long-head, medial hamstrings), VAS (3-component vasti), RF (rectus femoris), BFsh (biceps femoris short head), TA (tibialis anterior), GAS (medial and lateral gastrocnemius) and SOL (soleus). Muscles within each group received the same excitation signal and the muscle excitation-contraction dynamics were governed by Hill-type muscle properties.

The trunk was allowed to translate and rotate in the sagittal plane, and the hip and ankle joints were modeled as frictionless revolute joints. The knee joint had a moving center-of-rotation for flexion and extension, and the patella had a prescribed trajectory relative to the femur, both defined as functions of the knee flexion angle [16]. Passive joint torques were used to represent stiffness of ligaments, connecting tissue and other structures surrounding the joint based on Davy and Audu [18]. These passive torques are negligible during the normal range of motion, and increase exponentially as the motion approaches the joint limits. The model had a total of 9 degrees of freedom.

The musculoskeletal model was driven by 15 Hill-type musculotendon actuators per leg, which were combined into nine muscle groups based on anatomical classification (Fig. 1), with muscles within each group receiving the same excitation signal. The muscle contraction dynamics were governed by a Hill-type model formulation [19], and the activation and deactivation dynamics were modeled using first order differential equations [20]. Excitation for all muscles except the BFsh and IL groups were defined using EMG-based patterns (see Section 2.3). Since EMG data was not available for the BFsh and IL groups, they were modeled with a single block pattern with timing based on Perry [21].

The muscle coordination pattern for the contralateral leg was considered symmetric and 50% of the gait cycle out-of-phase of the ipsilateral leg.

2.2. Optimization framework

A nominal simulation was generated by solving the optimal tracking problem (e.g., [22]) using a simulated annealing optimization algorithm [23] that fine-tuned the muscle controls (i.e., each muscle's excitation onset, duration and magnitude) such that the difference between experimental and simulated body kinematics and ground reaction force (GRF) data was minimized. The quantities evaluated in the objective function included the right and left hip, knee and ankle angles, horizontal and vertical GRFs, trunk translation in the horizontal and vertical direction and trunk rotation.

2.3. Experimental data collection

In order to provide initial conditions for the simulations (i.e., initial body segment orientations and velocities), tracking data and EMG patterns for the muscle controls, experimental data were collected from ten subjects (five male, five female; age 29.6 ± 6.1 years; height 169.7 ± 10.9 cm; mass 65.6 ± 10.7 kg) walking at 1.5 m/s on a split-belt instrumented treadmill (Tecmachine, France) while EMG, three-dimensional GRFs and body segment motion data from the right leg were collected using a motion capture system (Motion Analysis Corp.) at 2000, 480 and 120 Hz, respectively for 15 seconds. Prior to the data collection, all subjects provided informed consent according to the rules and regulations of the Cleveland Clinic Foundation and The University of Texas at Austin. The EMG data were collected using disposable surface bipolar electrodes (Noraxon Inc.) from the right soleus, medial gastrocnemius, tibialis anterior, gluteus maximus, vastus medialis, biceps femoris long-head and rectus femoris. The data were band-pass filtered (20–400 Hz), fully rectified and then low-pass filtered at 10 Hz using a fourth order zero-lag digital Butterworth filter (e.g., [24]). The EMG linear envelope was then normalized to its maximum value observed over all step cycles for each subject. The body segment motion data were measured using a modified Helen Hayes marker set and corresponding joint angles were determined. The GRF and motion data were filtered with a fourth order zero-lag Butterworth filter with cut-off frequencies of 20 and 6 Hz, respectively. All data were time-normalized to a full gait cycle, averaged within each subject, and then across all subjects to obtain a group average.

2.4. Compensatory strategy analysis

To identify the compensatory strategies necessary to overcome the various musculoskeletal deficits, a perturbation analysis on individual musculoskeletal properties was performed. Using the nominal simulation, the musculoskeletal parameters were systematically altered one at a time, and the muscle controls were then reoptimized to identify the compensatory strategies necessary to restore the nominal walking kinematics and GRFs. The specific musculoskeletal parameters altered included decreasing the maximum isometric strength in each muscle group and increasing the passive hip joint stiffness. The maximum isometric strength of muscles within each group was reduced by 50% with all other muscle strengths unaltered. An upper limit equal to the nominal excitation value was placed on the reduced muscle group's excitation magnitude to prevent that muscle group from compensating by increasing its own excitation. The passive hip joint stiffness was increased by 20% and the range of motion for which the passive stiffness was negligible was decreased by six degrees in hip extension [9]. After each musculoskeletal parameter was increased or decreased, the new model was re-optimized until the tracking performance was within 10% of the value in the nominal simulation. The goal was to produce simulations that all had similar kinematics and GRF data. The compensatory strategies were then quantified by identifying differences in individual musculotendon work over the gait cycle relative to the nominal simulation, which was computed by integrating the corresponding musculotendon power.

3. Results

Consistent with our previous simulation analyses (e.g., [25,26]), the nominal simulation emulated the experimental body segment kinematic and GRF data within two standard deviations of the experimental data, and the resulting optimized muscle excitation patterns yielded timing similar to experimental EMG. When the musculoskeletal properties were altered, the dynamic optimization was able to identify compensatory strategies to regain normal walking kinematics and GRFs, except for the cases of reduced SOL and HAM strength. The tracking error for the SOL weakness perturbation was 33% greater than the nominal simulation due to excessive hip flexion during swing, and the tracking error associated with the HAM weakness perturbation was 25% greater than the nominal simulation due primarily to excessive ankle dorsiflexion in late swing and early stance. The primary compensatory mechanism in all simulations was to modulate the excitation magnitude, rather than varying the excitation timing. Only minor changes were needed to compensate for TA and BFsh weakness, with positive and negative work from BFsh compensating for TA weakness, and positive work from HAM compensating for BFsh weakness. In all simulations, the total musculotendon work over the gait cycle was within $\pm 10\%$ of the work performed in the nominal simulation, with the exception of the GMAX and IL weakness and increased hip stiffness simulations, which performed less overall muscle work (see Section 4).

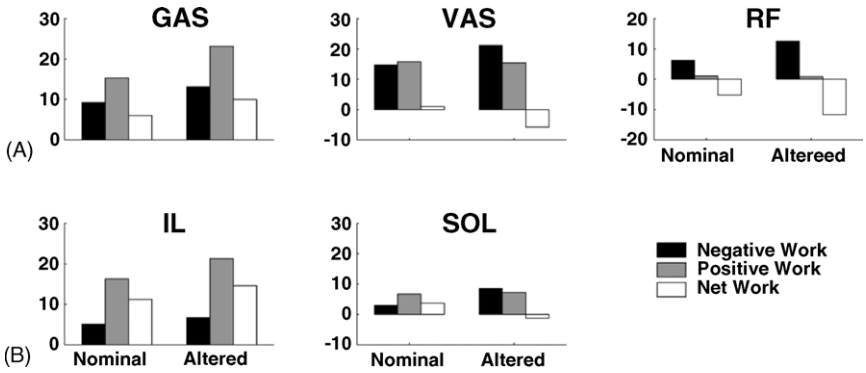

Fig. 2. Musculotendon work compensations over the gait cycle in response to (A) SOL weakness and (B) GAS weakness.

3.1. Ankle plantar flexor weakness

When SOL strength was decreased, the primary compensatory mechanism was an increase in positive work from GAS (Fig. 2A: GAS positive work) and negative work from VAS and RF (Fig. 2A: VAS and RF negative work). To a lesser extent, net work from GMAX also increased. When GAS strength was decreased, the primary compensatory mechanisms were an increase in positive work from IL and negative work from SOL (Fig. 2B: IL positive work; SOL negative work). To a lesser extent, net work from GMAX and negative work from BFsh increased.

3.2. Hip extensor and flexor weakness and hip joint stiffness

When GMAX strength was decreased, the primary compensatory mechanism was an increase in negative work from SOL (Fig. 3A: SOL negative work) and positive work from HAM (Fig. 3A: HAM positive work). To a lesser extent, positive work from GAS decreased. When RF strength was decreased, positive work increased in SOL and decreased in GAS and IL (Fig. 3B: SOL, GAS and IL positive work). When IL strength was decreased, positive work from SOL and GAS and negative work from RF increased (Fig. 3C: SOL, GAS positive work, RF negative work). To a lesser extent, positive

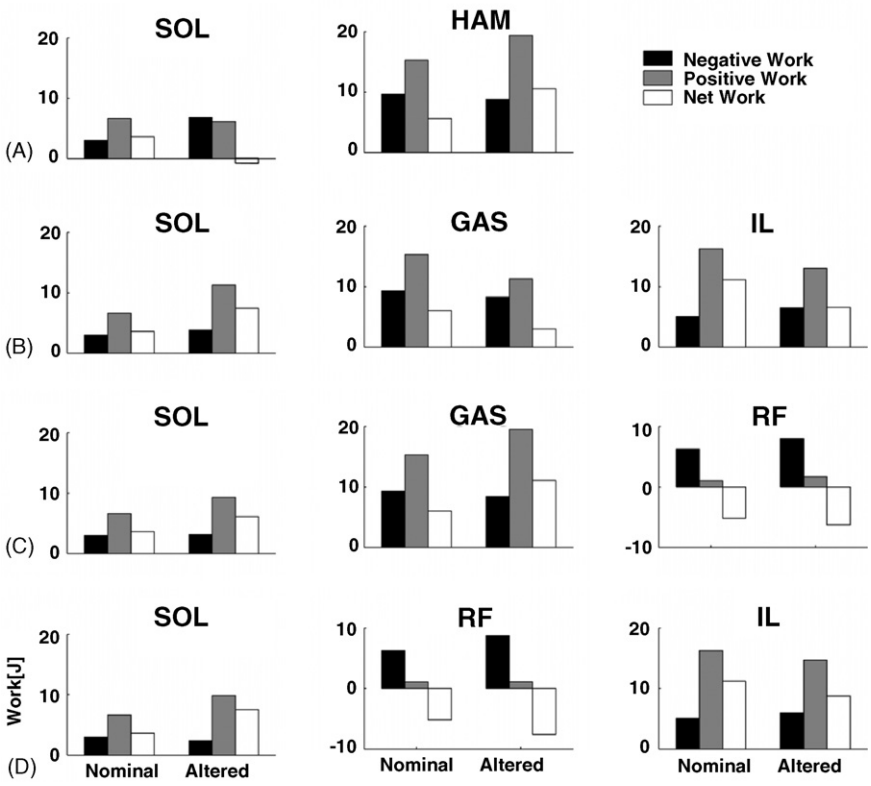

Fig. 3. Musculotendon work compensations over the gait cycle in response to (A) GMAX weakness, (B) RF weakness, (C) IL weakness and (D) increased hip stiffness.

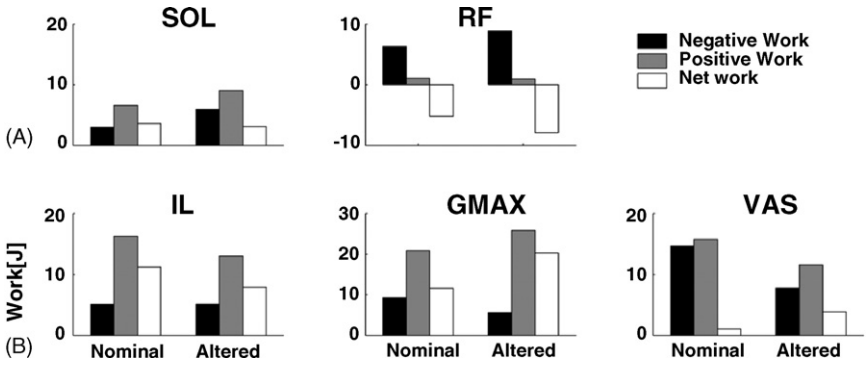

Fig. 4. Musculotendon work compensations over the gait cycle in response to (A) VAS weakness and (B) HAM weakness.

work from HAM and GMAX also decreased. When the passive hip joint stiffness was increased, positive work from SOL and negative work from RF increased (Fig. 3D: SOL positive work and RF negative work). To a lesser extent, positive work from IL decreased (Fig. 3D: IL net work), and positive work from GMAX decreased.

3.3. Knee extensor and flexor weakness

When VAS strength was decreased, the primary compensation occurred in SOL, which increased its negative and positive work output, and RF, which increased its negative work output (Fig. 4A: SOL positive and negative work and RF negative work). To a lesser extent, there was a decrease in positive work by GAS. When HAM strength was decreased, the largest changes occurred in VAS, GMAX and IL, with positive and negative work from VAS decreasing and net work from GMAX increasing, while positive work from IL decreased (Fig. 4B: VAS positive and negative work, GMAX net work and IL positive work). To a lesser extent, negative work from SOL increased and decreased in RF, while positive work from TA increased.

4. Discussion

The overall objective of this study was to use forward dynamics simulations and dynamic optimization to analyze walking at a nominal speed and identify the compensatory mechanisms necessary to regain a normal walking pattern in the presence of musculoskeletal deficits that have been proposed as impairments that limit walking performance in older adult populations. The simulations emulated treadmill walking, which has been shown to produce some kinematic differences relative to overground walking (e.g., [27]), although only minor changes have been observed in vertical GRFs and EMG (e.g., [28,29]). Since our focus is on muscle control compensations, such kinematic differences between treadmill and overground would not appear to influence on our results.

Previous modeling studies have performed similar analyses of compensatory mechanisms [30,31], but no study has analyzed the entire gait cycle. Jonkers et al. [30] used forward dynamics simulations and optimization to determine compensatory strategies present in the stance phase of gait as a result of the exclusion of SOL, GAS, HAM and GMAX. The activation of each of these muscles was systematically set to zero, and compensatory strategies were quantified by calculating changes in the activation levels of other muscles. Similar to the present study, they found that HAM and GMAX played an important role in compensating for one another as well as SOL and GAS, but that other compensations were necessary to completely compensate for the deactivation of these muscles. Komura et al. [31] used an inverse dynamics-based analysis and static optimization to examine the interactions of 13 lower extremity muscles during the stance phase of gait by systematically deactivating each muscle, which produced similar results to those of Jonkers et al. [30]. The results of our study build upon these previous analyses by examining the muscle mechanical energetics and compensatory mechanisms over the entire gait cycle associated with the clinically relevant impairments of reduced strength in specific muscle groups and increased hip joint stiffness.

A potential limitation of the musculoskeletal model used in our analysis was that the head, arms, torso and pelvis were combined into one segment and controlled by a small number of muscles crossing the hip joint (i.e., HAM, IL, RF and GMAX). As a result, GMAX, which contributes to trunk support in early stance [32,33], had an excitation level close to maximum in most of the optimizations. Consequently, the compensatory action of GMAX to provide necessary trunk support in early stance in response to muscle weakness in other groups may have been underestimated. In addition, GMAX acts to extend the hip in early stance while IL simultaneously flexes the contralateral hip in early swing [32,34,35]. Therefore, in order to provide trunk stability when the strength of GMAX was decreased, the work output of IL also decreased. Similar reductions occurred in GMAX when the strength of IL was decreased. This phenomenon also occurred in joint stiffness perturbation that resulted in larger trunk rotation deviations and a lower total work being done over the gait cycle compared to the nominal simulation. In a model with separate abdominal and lower back muscles

supporting the trunk (e.g., [36]), this may not occur. In addition, a more complex model with separate pelvis and trunk segments may allow movement of the trunk center-of-mass relative to the hip joint center that would also influence the hip flexor and extensor activity. This remains an area for further investigation.

Another potential limitation was that our model did not include the hip abductors (e.g., gluteus medius), which have been shown to provide body support in single-leg stance [33]. Thus, the necessary compensatory action of SOL in mid-stance (e.g., in response to hip or knee extensor weakness) may be overestimated due to the exclusion of the hip abductors.

The ankle plantar flexors (SOL and GAS) were shown to be able to compensate for most of the major muscle groups, which is consistent with the plantar flexors being important contributors to support, forward propulsion and swing initiation in normal walking [25]. When GMAX and VAS strength were decreased, negative work from SOL increased (Figs. 3A and 4A: SOL negative work) to provide the trunk support normally provided by VAS and GMAX in early stance [32]. Inversely, when SOL strength was decreased, positive work from GMAX and negative work from VAS increased (Fig. 2A: VAS negative work) to provide the trunk support normally provided by SOL in early stance [25]. The compensations between these muscles are possible because SOL, GMAX and VAS have all been shown to decelerate the downward motion of the trunk in early stance through their contributions to the vertical intersegmental joint forces [25,32,37].

The plantar flexors also compensated for IL weakness and increased hip stiffness. When IL strength was decreased, the largest change occurred in GAS (see also [31]), which is consistent with both IL and GAS acting to accelerate the leg into swing in late stance/pre-swing [26,32]. Inversely, when GAS strength was decreased, positive work from IL increased (Fig. 2B: IL positive work) to provide swing initiation in late stance/early swing [32]. When the hip stiffness was increased, the primary compensatory mechanism was an increase in positive work output from SOL (Fig. 3D: SOL positive work). Previous studies have shown that SOL accelerates the hip into extension [25,34,35], which would be necessary to overcome the increased hip joint stiffness as the hip extends during stance. The passive hip joint stiffness was modeled as a viscoelastic torsional element [18], which can store and release elastic energy [12]. Energy stored in the passive structures from muscles that accelerate the hip into extension in early stance (e.g., SOL, GMAX, VAS and HAM [25,32,34] would be subsequently released in late stance to assist in swing initiation. This is consistent with the observed decrease in IL work output in pre-swing and early swing with increased hip stiffness (Fig. 3D: IL net work), as this work output would be no longer necessary to help accelerate the leg into swing.

The plantar flexors also compensated for VAS and RF weakness. When VAS strength was decreased, an increase in output from SOL was observed (Fig. 4A: SOL positive and negative work) to provide the support and forward propulsion normally provided by VAS in early stance [25,32]. Similar compensations were observed in Komura et al. [31]. In the presence of RF weakness, the simulation data showed that the positive work from SOL increased (Fig. 3B: SOL positive work; see also [31]) to provide the forward propulsion normally provided by RF in late stance [32]. Inversely, when SOL strength was decreased, negative work from RF increased in late stance (Fig. 2A: RF negative work). These compensations between SOL, VAS and RF are consistent with these muscles providing trunk support (SOL and VAS, see above) and propulsion in early (VAS) and late (SOL and RF) stance through their contributions to the hip intersegmental joint force that acts to accelerate the trunk forward [25,32,37]. The increased negative work by RF, however, necessitated a synergistic increase in positive work by GAS (Fig. 2A: GAS positive work and RF negative work). In late stance and pre-swing, GAS has been shown to generate energy in the leg, while RF simultaneously transfers energy from the leg to the trunk to provide forward propulsion [25,32]. Thus, the increase in RF negative work required a corresponding increase in GAS (see also [31]). This synergistic interaction also occurred when RF strength was decreased (Fig. 3B: GAS positive work).

In our analysis, SOL and GAS strengths were decreased independently. However, it is not clear if such decreases do indeed occur independently. Therefore, we performed a post hoc analysis where we decreased SOL and GAS strength simultaneously by 50%. The optimization, however, could not find a muscle coordination pattern that generated a reasonable tracking performance. A solution could be found when the strengths were reduced by 30%, although the vigorous push-off into plantar flexion did not occur in the simulation, which resulted in an earlier rise into dorsiflexion (Fig. 5A: approximately 60–100% gait cycle) and a slower walking speed. The primary compensatory strategy was an increase in net work from GMAX in early stance and positive work from IL in pre-swing/early swing (Fig. 5B: GMAX net work, IL positive work). This strategy was consistent with observations in clinical studies. Increased hip extensor work is characteristic of elderly gait and has been proposed to compensate for a lack of trunk support in early stance [5,12,38], which is normally provided by both SOL and GAS [25], while increased hip flexor (IL) work can potentially compensate for decreased swing initiation [7,12] normally provided by GAS [25]. In addition, the decreased range of ankle motion in swing (Fig. 5 A: approximately 60–100% gait cycle) and slower walking speed with decreased plantar flexor strength are both characteristics of elderly gait mechanics [5,8]. These results are consistent with those studies suggesting that reduced ankle plantar flexor strength is a limiting factor in the gait speed of older adults [7] and important contributors to the kinematic and kinetic differences observed in elderly gait [5,8,11,12,38].

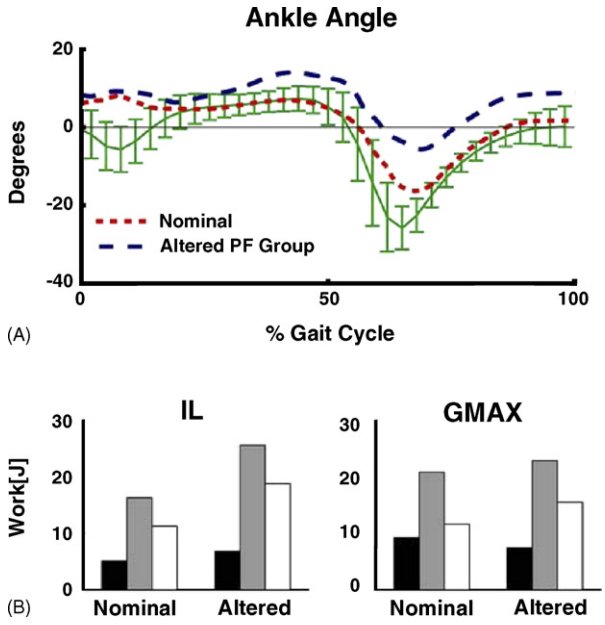

Fig. 5. (A) Right ankle angle of the combined plantar flexor weakness simulation (dashed line) compared to that of the nominal simulation (dotted line) and the subjects' group average pattern ± 2 S.D. (B) Negative (black bars), positive (gray bars) and net (white bars) musculotendon work compensations for IL and GMAX over the gait cycle when the combined plantar flexor strength was decreased by 30%.

GMAX and HAM were shown to compensate for one another, which is consistent with previous studies [30,31]. When GMAX strength was reduced, positive work from HAM increased in early stance (Fig. 3A: HAM positive work) to compensate for the reduced GMAX contribution to trunk support and forward propulsion [32]. This was consistent with HAM acting to accelerate the hip and knee into extension similar to GMAX [34,35]. When HAM strength was decreased, the primary compensatory strategy was an increase in work output from VAS and GMAX and a decrease in work from IL (Fig. 4B). Increased GMAX output in response to HAM weakness was the largest compensation observed in Komura et al. [31] and also observed in Jonkers et al. [30]. Of all the muscles, the decrease in HAM strength required the most compensation from other muscle groups.

This simulation study highlighted how redundancy in muscle contributions to the mechanical energetics of walking allows the nervous system to compensate for specific musculoskeletal deficits. Although the compensatory strategies identified by the dynamic optimization may not be unique, as other solutions may exist, of particular importance appears to be the compensatory mechanisms provided by the plantar flexors, which were shown to provide important compensations for several musculoskeletal deficits including diminished muscle strength in the hip and knee flexors and extensors and increased hip joint stiffness. This importance was further highlighted when normal kinematics and GRFs could not be achieved through compensatory action of other muscle groups when the plantar flexor strength was decreased as a group, which is consistent with clinical studies suggesting that the plantar flexors are a limiting factor in gait performance [7]. Thus, rehabilitation and preventative strategies may consider focusing on increasing or maintaining plantar flexor strength.

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