Simulating the effect of muscle weakness and contracture on neuromuscular control of normal gait in children

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Highlights

- Normal gait may be achievable with muscle weakness and contracture.
- Altered muscle activation is required to cope with these musculoskeletal deficits.
- Reduced stiffness of the plantarflexors appears necessary to achieve normal gait.
- Improved strength and motor function of tibialis anterior also appears necessary.

ABSTRACT

Altered neural control of movement and musculoskeletal deficiencies are common in children with spastic cerebral palsy (SCP), with muscle weakness and contracture commonly experienced. Both neural and musculoskeletal deficiencies are likely to contribute to abnormal gait, such as equinus gait (toe-walking), in children with SCP. However, it is not known whether the musculoskeletal deficiencies prevent normal gait or if neural control could be altered to achieve normal gait. This study examined the effect of simulated muscle weakness and contracture of the major plantarflexor/dorsiflexor muscles on the neuromuscular requirements for achieving normal walking gait in children. Initial muscle-driven simulations of walking with normal musculoskeletal properties by typically developing children were undertaken. Additional simulations with altered musculoskeletal properties were then undertaken; with muscle weakness and contracture simulated by reducing the maximum isometric force and tendon slack length, respectively, of selected muscles. Muscle activations and forces required across all simulations were then compared via waveform analysis. Maintenance of normal gait appeared robust to muscle weakness in isolation, with increased activation of weakened muscles the major compensatory strategy. With muscle contracture, reduced activation of the plantarflexors was required across the mid-portion of stance suggesting a greater contribution from passive forces. Increased activation and force during swing was also required from the tibialis anterior to counteract the increased passive forces from the simulated dorsiflexor muscle contracture. Improvements in plantarflexor and dorsiflexor motor function and muscle strength, concomitant with reductions in plantarflexor muscle stiffness may target the deficits associated with SCP that limit normal gait.

Keywords

Biomechanics; Musculoskeletal Modelling; Equinus Gait; Cerebral Palsy; Walking

1. Introduction

Cerebral palsy (CP) is caused by a non-progressive brain lesion occurring at or near birth and is a common cause of physical disability affecting children in developed countries[1]. Although the lesion is static, the resulting muscle pathology is progressive[1]. Spastic CP (SCP) arises when this lesion causes motor cortex damage. Children with SCP exhibit altered neural control of movement, with this having a secondary effect on musculoskeletal development[2].

Muscle weakness and contracture are common musculoskeletal deficiencies associated with SCP[3-7]. Muscle weakness refers to a reduction in the capacity to produce and maintain force. The ankle muscles are commonly affected[6], with strength deficits ranging from 25%-75% compared to typically developed peers[4, 6, 8]. Contracture refers to an increase in passive muscle stiffness[9], resulting in increased resistance to stretch[2]. Contracture limits range of motion and represents a major disability for those with CP[10]. These deficiencies are often associated with abnormal gait patterns[11]. Deficiencies in the plantarflexor and dorsiflexor function can lead to equinus gait (i.e. 'toe-walking'), a common pattern associated with CP[11]. This abnormal gait likely stems from a combination of spasticity, contracture, or altered neural control that prevents heel contact with the ground at initial contact[12]. However, it is difficult to determine how these each contribute to gait abnormalities and which impairment must be targeted within interventions.

Musculoskeletal modelling has become a useful tool for evaluating pathological gait, particularly in CP[13-15]. Much of this work has utilised models with muscle properties based on unimpaired individuals, which do not accurately reflect those with SCP[2]. Studies[13-15] have primarily focused on abnormal movement patterns to examine muscle coordination, without fully considering the impact of altered muscle-tendon properties. Conducting simulations with properties that resemble the deficiencies associated with SCP may elicit further understanding of the control strategies required, or factors that limit normal gait. This may assist in developing interventions that improve gait function in children with SCP. The purpose of this study was to examine the effect of simulated muscle weakness of the plantarflexor/dorsiflexor muscles and contracture of the plantarflexor muscles on the neuromuscular requirements for achieving normal walking gait. It was hypothesised that normal gait

would be robust to weakness of the plantarflexors/dorsiflexors, however contracture of the plantarflexors would be a limiting factor in maintaining normal gait.

2. Methods

As this study aimed to examine the neuromuscular requirements for maintaining normal walking gait in the presence of musculoskeletal deficits associated with SCP, typically developing children were required as participants to obtain experimental data of the desired gait pattern. Ten typically developing children (6:4 male:female; 10.0 ± 2.1 years; 140.2 ± 22.13 cm; 28.8 ± 10.5 kg) free from neuromuscular impairment participated in this study. Ethical approval, parental consent, and participant assent were obtained prior to data collection.

Three-dimensional (3D) gait data was collected as participants walked barefoot over a 10m level walkway with three embedded force platforms at a self-selected speed (1.02 ± 0.14 $m \cdot s^{-1}$; range = 0.74–1.14 $m \cdot s^{-1}$). Participants performed ten or more practice trials to determine their starting point along the walkway for adequate contact with the force platforms. Participants were not aware of the force platforms and were instructed to look forward at all times. Ten trials were recorded, with three successful trials selected for analysis. Successful trials were defined by two consecutive force plate strikes without targeting. Step and stride times were identified from successful trials via a vertical ground reaction force (GRF) threshold of 20N. The swing phase of the gait cycle was defined when vertical GRF returned below 20N.

Motion data were collected using an eight-camera 3D motion capture system (100Hz) (Vicon, Oxford Metrics Limited, Oxford, United Kingdom). A modified version of the Plug-in-Gait marker set was used with additional marker clusters on the thigh and shank, and an additional marker on the fifth metatarsal head[16]. A two-second calibration trial was recorded with participants standing in a neutral position. Ground reaction forces (GRFs) (1000Hz) were recorded using three 510mm by 465mm in-ground force platforms (AMTI, Watertown, MA, United States). The force platforms were arranged in-line at the centre of the 10m walkway. Surface electromyography (EMG) (1000Hz) were recorded using bipolar surface electrodes (Duotrode, Myotronics, Kent, WA, United States) with an

inter-electrode distance of 2cm. EMG data were collected telemetrically (Aurion ZeroWire, Milan, Italy) from the medial (MG) and lateral gastrocnemius (LG), soleus (SOL) and tibialis anterior (TA). Electrodes were placed in accordance with SENIAM guidelines[17], with placement confirmed via palpation of the muscle belly. Processing of EMG data consisted of a 30Hz zero-lag fourth-order high-pass Butterworth filter to minimise movement artefact, full-wave rectification, and a 6Hz zero-lag fourth-order low-pass Butterworth filter to obtain linear envelopes. Data were amplitude normalised to the mean of the peaks that occurred across the trials used for analysis and interpolated to 101 points per gait cycle.

Muscle-driven simulations of a representative gait cycle for the right limb were generated using OpenSim 3.3[18]. A generic musculoskeletal model of the pelvis and lower limbs, with 16 degrees of freedom (DoF) and 86 musculotendon actuators was used. A similar model has been used in both typically developed[19] and children with CP[14, 16, 20]. Segment geometry were scaled for each participant using their calibration trial, which was also used as a reference for adjusting the positions of the markers on the model. An estimation was used for scaling muscle force in the absence of experimental strength data. Scaling muscle strength to height-squared has been use in a previous simulation study involving children[15], as height-squared has been correlated to the physiological cross-sectional area of muscle in this population[21]. Therefore, maximum isometric force of muscles were estimated and scaled by the height-squared of each participant. Joint angles and moments were calculated using filtered (6Hz) marker trajectories and GRF data within inverse kinematic and dynamics analyses. Dynamic inconsistencies between measured GRFs and model kinematics were then resolved via a residual reduction algorithm[18]. The muscle excitations and forces required to drive the simulation were then estimated using computed muscle control (CMC)[22]. Simulated muscle activation showed similar features to the experimentally measured EMG.

Additional muscle-driven simulations of gait were generated for each participant's trials with altered muscle parameters representing weakness and contracture. Separate musculoskeletal models were created for each participant representing varying degrees of weakness and contracture. Weakness was simulated by reducing the maximal isometric force of selected muscles[23]. The strengths of MG, LG,

SOL and TA were progressively reduced by 15% and 30%. Strength deficits in this range have been identified in children with SCP[4]. There are variable methods for simulating muscle contracture within musculoskeletal models, such as shortening tendon slack length[2, 24] and reducing optimal fibre length[25]. Comparisons of fibre lengths between individuals with SCP and typically developing peers show inconsistent results, with a recent review showing no significant difference in passive fascicle length [26]. Hence, within this study, muscle contracture was simulated by shortening the tendon slack lengths of select muscles[2, 24]. The tendon slack lengths of MG, LG and SOL were progressively shortened by 1.5% and 3.0%. To evaluate the appropriateness of these values; the passive mechanical properties of the plantarflexors for each model were examined by simulating passive ankle motion between 15 and 25 degrees of plantarflexion and dorsiflexion, respectively. The shift in passive torque generated at the ankle with the adjusted tendon slack length parameters were similar to that seen between typically developed and SCP populations[7]. In addition, a similar slack angle to children with SCP (i.e. the ankle angle where ankle torque exceeded 0 Nm of plantarflexion torque)[7] was observed with these changes (Supplementary Figure 1). All combinations of weakness (i.e. normal, 15% and 30%) and contracture (i.e. normal, 1.5% and 3.0%) were simulated, resulting in nine muscle-driven simulations per trial. Across all simulations, reserve actuators were included for each DoF to ensure simulations ran where muscles could not generate sufficient torques. Reserve actuators generate small torques to account for large angular accelerations. As these are highly penalised by the cost function, their contribution is normally low and negligible[23]. The contribution and change of reserve actuators to joint torques were monitored across simulations. A peak reserve actuator torque that was > 5% of the peak joint moment was used to indicate failure of the muscles to generate sufficient joint torques[23]. Residual force and moment actuators were also applied at the pelvis across all simulations.

Inspection of the activations and forces produced across simulations revealed minimal descriptive differences outside of the major ankle plantarflexor (MG, LG and SOL) and dorsiflexor (TA) muscles. Subsequent statistical analyses therefore focused on these muscles. A waveform analysis, using one-dimensional statistical parametric mapping (SPM1D), was implemented via open source

code[27] in MATLAB (The Mathworks Incorporated, Natick, MA, United States). Specific details of SPM1D are provided elsewhere[28] and hence are described briefly. The advantage of this analysis is it identifies differences at each time node across the waveforms originally sampled space[28]. A two-way (3 x 3; weakness x contracture) repeated-measures analysis of variance (ANOVA) was used to test the main and interaction effects of weakness and contracture on muscle activations and forces across the gait cycle. Where statistically significant main or interaction effects were identified, post-hoc analyses using SPM1D paired t-tests determined the direction or structure of the main and interaction effects, respectively. To retain a Type I family-wise error rate of alpha = 0.05, a Šidák corrected threshold based on the total number of comparisons being made across all statistical tests was calculated and used to denote statistical significance.

3. Results

Joint angles across all simulations reproduced those calculated by inverse kinematics with a difference of less than 1^{O}. Minimal contributions (i.e. peak reserve actuator torque < 5% of the peak joint moment) were required from reserve actuators at the hip and knee across all simulations. Increases in reserve actuator contribution to dorsiflexion torque at the ankle were observed during the initial aspect of the swing phase (~60–75% of the gait cycle) across certain simulations. Peak reserve actuator torque at the ankle greater than 5% of the peak joint moment did not occur during simulations where weakness was simulated in isolation. However, this frequently occurred during simulations where muscle stiffness was increased (i.e. shortening of tendon slack lengths by 1.5% and 3.0%) without a large concomitant reduction in muscle strength (i.e. normal strength or 15% reduced strength). Peak forces and moments of the residual actuators remained below 10N and 30Nm, respectively, across all simulations.

3.1 Main Effects: Weakness of the Ankle Plantarflexors and Dorsiflexors

Main effects of weakness of the plantarflexors and dorsiflexors on muscle activation and force requirements were identified (Figures 1 and 2).

There was a statistically significant main effect ($p < 0.001$) of weakness on activation of MG, LG, SOL and TA. Post-hoc analysis revealed an increase in activation with decreases in strength for the plantarflexors. In contrast, activation of TA was reduced with decreases in strength. Specifically; MG, LG and SOL activation increased during the mid-portion of stance (MG = 24–37%; 41–49%, LG = 43–49%, SOL = 21–50% of the gait cycle); while TA activation decreased during early stance and swing (3–12%; 73–100% of the gait cycle).

A statistically significant main effect ($p < 0.001$) of weakness on force of MG, LG, SOL and TA was also observed. Post-hoc analysis revealed a decrease in force with decreases in strength for these muscles across respective portions of the gait cycle. Specifically; MG, LG and SOL force decreased during the mid-portion of stance and throughout swing (MG = 3–16%; 66–100%, LG = 3–43%; 65–100%, SOL = 40–49%; 58–97% of the gait cycle); while TA force decreased during early stance and swing (1–8%; 60–100% of the gait cycle).

3.2 Main Effects: Contracture of the Ankle Plantarflexors

Main effects of contracture of the plantarflexors on muscle activation and force requirements were identified (Figures 3 and 4).

There was a statistically significant main effect ($p < 0.001$) of contracture on activation of MG, LG and TA. Post-hoc analysis revealed a decrease in activation with shorter tendon slack lengths (i.e. increased stiffness) for MG and LG across the mid-portion of stance (MG = 28–48%, LG = 30–52% of the gait cycle). Conversely, an increase in activation with shorter tendon slack lengths was observed for the TA during swing (73–98% of the gait cycle).

A statistically significant main effect ($p < 0.001$) of contracture on muscle force of MG, LG, SOL and TA was also observed. Post-hoc analyses revealed an increase in force with shorter tendon slack lengths for these muscles across respective portions of the gait cycle. Specifically; MG and LG force increased during early stance and swing (MG = 14–18%; 77–96%, LG = 14–23%; 65–82%; 87–100% of the gait cycle); while SOL and TA force increased during swing (SOL = 79–100%, TA = 79–100% of the gait cycle).

3.3 Interaction Effects: Weakness x Contracture

There was a statistically significant interaction effect ($p < 0.001$) of weakness and contracture on activation of TA. Post-hoc analyses within and between factors revealed TA activation increased by a progressively greater amount during early stance (0–19% of the gait cycle) and late swing (83–100% of the gait cycle) as muscle strength decreased with progressively shorter tendon slack lengths.

A statistically significant interaction effect ($p < 0.001$) of weakness and contracture on muscle force of MG, LG, SOL and TA was also observed. Post-hoc analyses within and between factors revealed a progressively smaller decrease in force as muscle strength decreased and tendon slack length decreased for MG, LG and SOL during late swing (MG = 88–100%, LG = 86–100%, SOL = 76–100% of the gait cycle); and the TA during early stance (0–14% of the gait cycle) and late swing (80–100% of the gait cycle).

4. Discussion

This study explored the neuromuscular requirements for maintaining normal gait in children with simulated weakness of the plantarflexor/dorsiflexor muscles and contracture of the plantarflexor muscles, two common deficiencies associated with SCP. Normal gait appeared robust to weakness in isolation. This suggests weakness is not necessarily a limiting factor to achieving normal gait where neural control is sufficiently adaptable. Increased activation of the plantarflexors across the mid-portion of stance was observed in response to weakness. Despite this increase, the force produced by these muscles decreased across a similar period of the gait cycle. In contrast, activation of TA during early stance and swing was decreased with weakness of the plantarflexors. Weakness in isolation did not result in large increases in reserve actuator torque at the ankle, suggesting the neuromuscular adaptations were sufficient in maintaining normal gait with the simulated strength deficits. Previous work has shown increased activation can compensate for strength deficits up to 80% in maintaining normal gait[23]. Combined with the present findings, it could be inferred that increasing activation is a sufficient strategy for coping with muscle weakness during gait. However, van der Krogt et al.[23] linked an increase in total muscle cost, a measure of the load placed on the muscle, with this strategy.

It was hypothesised that this increased load could result in fatigue, additional weakness, or damage of the muscle[23]. Increasing the strength of the plantarflexors would reduce the neural demand placed on these muscles in maintaining normal gait and avoid these potential negative consequences. Strength training in an attempt to reverse the weakness associated with SCP is likely key in promoting normal and functionally appropriate gait.

Reduced activation of MG and LG during the mid-portion of stance was observed as a response to contracture. While activation of the plantarflexors remained unchanged during early stance and swing, the force produced by these muscles increased. This suggests an increased contribution of passive forces to total force during these stages of gait. Equinus gait is characterised by excessive plantarflexion during stance and/or swing[11], and it is likely that increased stiffness and passive forces generated by the plantarflexors contribute to this gait abnormality[29]. Increased activation and force of TA during swing was observed as a response to simulated plantarflexor contracture. This response of TA was further heightened as muscle strength was decreased, with greater increases in activation required in response to increased plantarflexor muscle stiffness with a weaker TA. This appeared to be in response to the increased passive forces generated by the plantarflexors at this part of the gait cycle. Reduced strength and activation capacity of TA has been found in children with SCP[4, 6], therefore interventions targeting improvements in strength and function of this muscle may combat plantarflexor contracture and equinus gait. Despite these adaptations, maintenance of normal gait may be difficult with muscle contracture, particularly where stronger plantarflexor muscles are present. Reserve actuator torque at the ankle was highest during swing where stiffness of the plantarflexors was greatest, or when muscle stiffness was combined with normal muscle strength (Figure 5). The contribution of the ankle reserve actuator under conditions of muscle contracture without concomitant decreases in strength were at the level used to indicate simulation failure. It appears that normal gait may be difficult to attain with significant plantarflexor contracture, particularly when these muscles are capable of producing high forces. This has important implications for programs that target strength improvements in the plantarflexors for children with SCP. Improvements in plantarflexor strength stemming from these programs must be combined with

reductions in stiffness to avoid the combination of strong, stiff muscles that may make normal gait difficult to achieve. Further work identifying protocols that induce strength gains while reducing stiffness, particularly in a SCP population, are likely to be beneficial.

While neuromuscular adaptations to maintain normal gait were identified, these may not be instinctively achievable for children with SCP. Reduced activation of MG and LG during stance was observed with increased plantarflexor stiffness. Premature and greater activation of the plantarflexors is a commonly observed activation pattern during 'toe-walking'[12, 30]. Further, antagonistic coactivation of the plantarflexors has been identified in children with CP[6, 8]. Children with SCP demonstrate higher levels of coactivation in the plantarflexors compared to typically developing children when producing a dorsiflexion movement[6]. However, while coactivation may be present during gait in children with SCP, it is unknown whether the forces generated by such activation would be sufficient to alter gait. Nonetheless, the ability to 'switch-off' these muscles may not be intuitive for children with SCP. High levels of TA activation were also required during simulations, with these often reaching maximal levels. Children with CP have an inability to selectively and maximally activate their muscles[6, 8], with this deficit extending to TA[6]. Training that increases the voluntary activation capacity of muscles for children with SCP may therefore be beneficial in promoting walking capacity.

Limitations

Muscle contracture was simulated via shortening of tendon slack length in isolation, with no other changes made to muscle tendon unit properties. This method was chosen as it simulated the increase in passive muscle stiffness associated with contracture. Previous work[25] has reduced optimal fibre length to simulate this increase in passive stiffness. However, given the variable findings around differences in fibre lengths between typically developing children and those with SCP[26], we chose to leave parameters relating to fibre length unaltered. It is, however, unlikely that muscle contracture is represented by a shortening of tendon slack length in isolation. Additional changes in muscle morphology and structure (such as reduced muscle belly length, volume, cross-sectional area and thickness) have been noted in children with SCP[26]. We did conduct an analysis (not reported within

this study for brevity) on the effect of shortening fibres rather than slack length, and this resulted in very similar results to those presented here (see Supplementary Document 1). Overall, there is very little evidence suggesting the precise mechanism contributing to muscle contracture. However, altering additional relevant characteristics within musculoskeletal models of contracture may elucidate further information about how these parameters impact gait function in children with SCP.

Experimental measures of strength were not collected from participants and this may have impacted our ability to appropriately scale muscle strength and subsequent weakness within the musculoskeletal models. Due to this, an estimate of muscle strength was used in scaling the musculoskeletal models which could potentially over or underestimate the baseline strength of participants. We attempted to address this limitation by: (1) using an appropriate and valid estimate of muscle strength based on body size[21]; and (2) basing the values for weakness on normalised (i.e. percentage) deficits identified in children with SCP[4] rather than absolute strength values. Nonetheless, the accuracy of baseline and weakened models would be improved with experimental measures of strength and should thus be included in future work.

Conclusions

This study demonstrated that muscle weakness and contracture present a requirement for increased lower limb muscle activity to achieve a typical gait pattern, however their combined effect generally allowed for normal gait to be achieved within the capacity of the muscles. The practical implications of this work revolve around the design of training to promote walking capacity in children with SCP. Interventions targeting improvements in plantarflexor and dorsiflexor motor function (i.e. activation capacity) and strength[31], along with reductions in plantarflexor muscle stiffness, are likely to improve gait capacity. A focus on TA may be necessary given the high degree of activation and force required during simulations, particularly when overcoming forces associated with contracture of the plantarflexors.

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Conflict of Interest Statement

All authors declare there are no conflicts of interest associated with the production of this manuscript.

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Figure Captions

Figure 1 – Effect of plantarflexor and dorsiflexor muscle weakness on muscle activation across the gait cycle. Solid lines represent the normal strength model, with the dashed and dotted lines representing models with strength deficits of 15% and 30%, respectively. The dashed vertical line indicates the stance-swing transition, with the stance and swing phases presented as 0-59.2% and 59.2-100% of the gait cycle, respectively. Solid horizontal bars indicate where statistical parametric mapping found a statistically significant ($p < 0.001$) main effect.

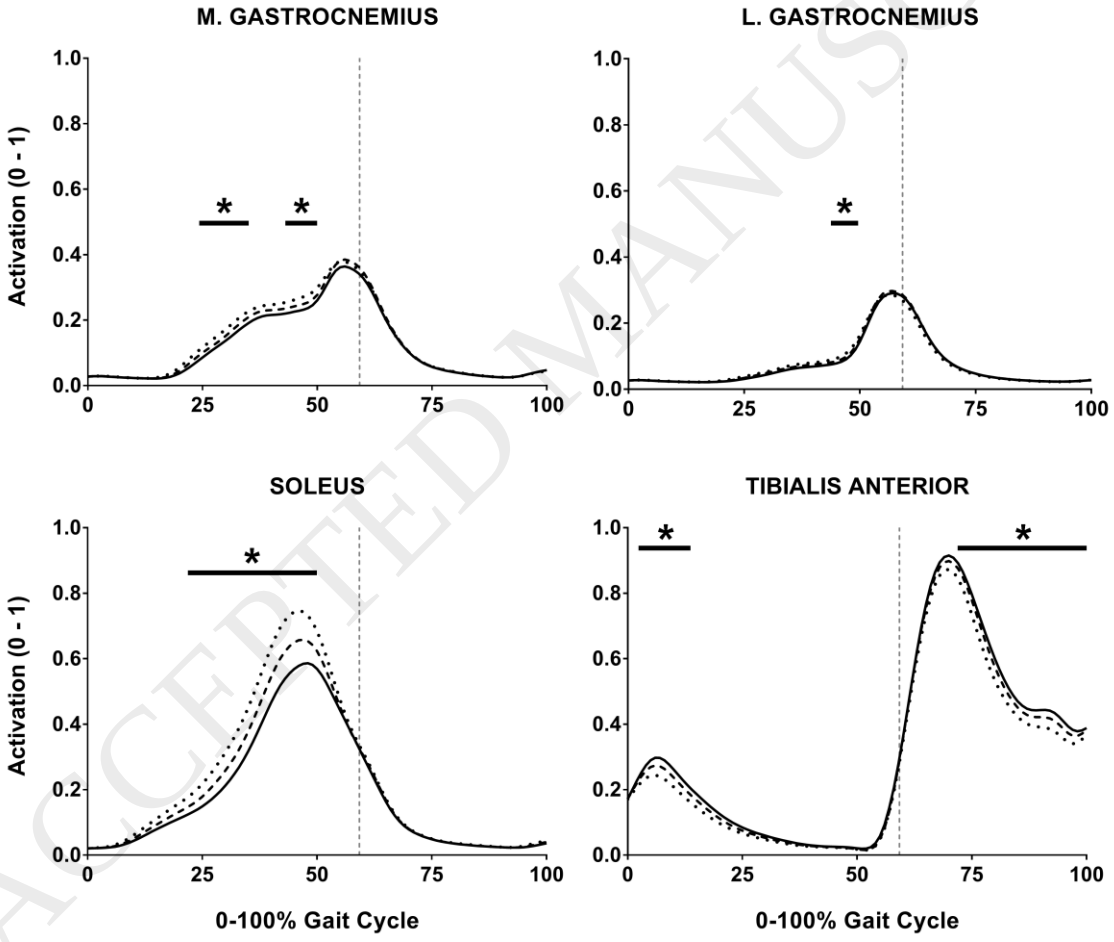


Figure 2 – Effect of plantarflexor and dorsiflexor muscle weakness on muscle forces across the gait cycle. Solid lines represent the normal strength model, with the dashed and dotted lines representing models with strength deficits of 15% and 30%, respectively. The dashed vertical line indicates the

stance-swing transition, with the stance and swing phases presented as 0-59.2% and 59.2-100% of the gait cycle, respectively. Solid horizontal bars indicate where statistical parametric mapping found a statistically significant ($p < 0.001$) main effect.

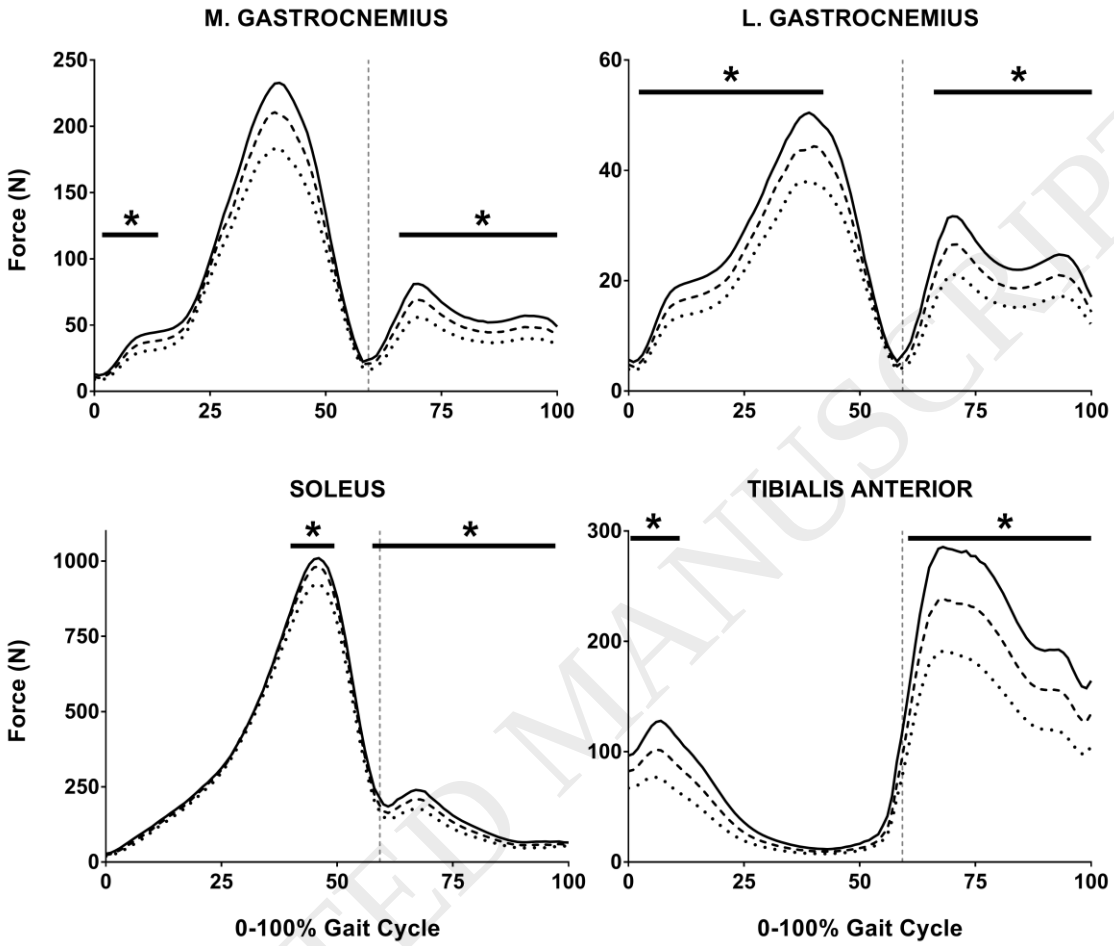


Figure 3 – Effect of plantarflexor muscle contracture on muscle activations across the gait cycle. Solid lines represent the normal tendon slack length model, with the dashed and dotted lines representing models with tendon slack length decreased by 1.5% and 3.0%, respectively. The dashed vertical line indicates the stance-swing transition, with the stance and swing phases presented as 0-59.2% and 59.2-100% of the gait cycle, respectively. Solid horizontal bars indicate where statistical parametric mapping found a statistically significant ($p < 0.001$) main effect.

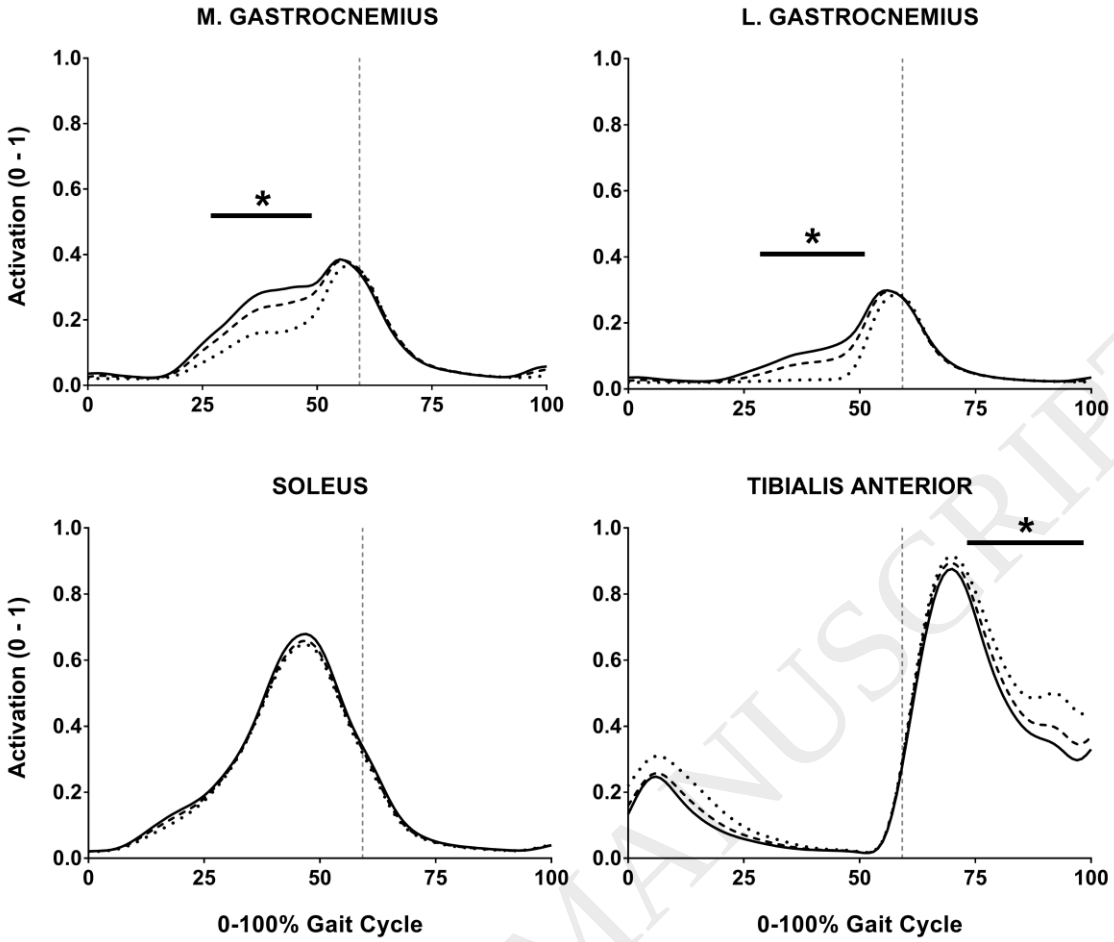


Figure 4 – Effect of plantarflexor muscle contracture on muscle forces across the gait cycle. Solid lines represent the normal tendon slack length model, with the dashed and dotted lines representing models with tendon slack length decreased by 1.5% and 3.0%, respectively. The dashed vertical line indicates the stance-swing transition, with the stance and swing phases presented as 0-59.2% and 59.2-100% of the gait cycle, respectively. Solid horizontal bars indicate where statistical parametric mapping found a statistically significant ($p < 0.001$) main effect.

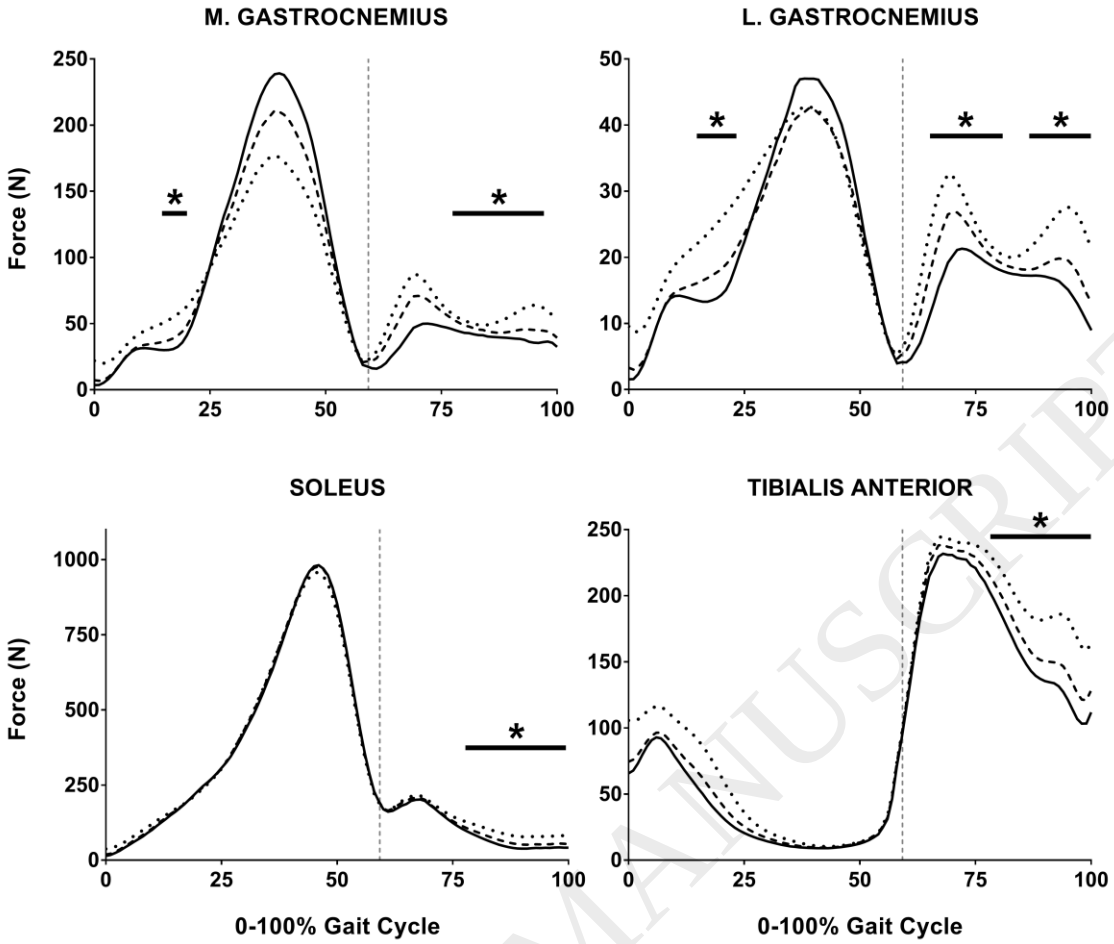
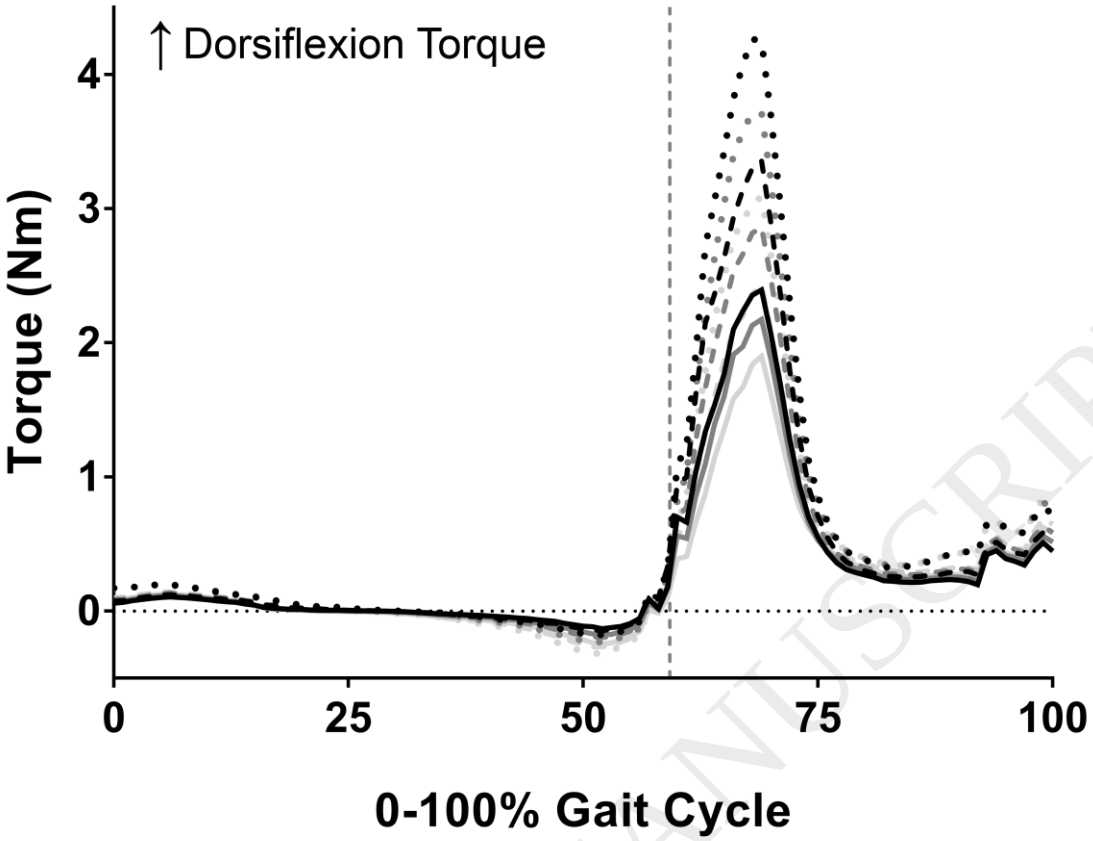


Figure 5 – Effect of plantarflexor and dorsiflexor muscle weakness, and plantarflexor muscle contracture on ankle reserve actuator torque contributions across the gait cycle. Black lines indicate normal strength models, with dark grey and light grey lines represent models with strength deficits of 15% and 30%, respectively. Solid lines represent the normal tendon slack length model, with the dashed and dotted lines representing models with tendon slack length decreased by 1.5% and 3.0%, respectively. The dashed vertical line indicates the stance-swing transition, with the stance and swing phases presented as 0-59.2% and 59.2-100% of the gait cycle, respectively. Positive values indicate the production of an internal dorsiflexor torque, while negative values indicate the production of an internal plantarflexor torque.



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