

Effect of equinus foot placement and intrinsic muscle response on knee extension during stance

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Abstract

Equinus gait, a common movement abnormality among individuals with stroke and cerebral palsy, is often associated with knee hyperextension during stance. Whether there exists a causal mechanism linking equinus foot placement with knee hyperextension remains unknown. To investigate the response of the musculoskeletal system to equinus foot placement, a forward dynamic simulation of normal walking was perturbed by augmenting ankle plantarflexion by 10° at initial contact. The subsequent effect on knee extension was assessed when the muscle forces were allowed, or not allowed, to change in response to altered kinematics and intrinsic force–length–velocity properties. We found that an increase in ankle plantarflexion at initial contact without concomitant changes in muscle forces caused the knee to hyperextend. The intrinsic force–length–velocity properties of muscle, particularly in gastrocnemius and vastus, diminished the effect of equinus posture alone, causing the abnormal knee extension to be less pronounced. We conclude that the effect of ankle position at initial contact on knee motion should be considered in the analysis of equinus gait.

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1. Introduction

Equinus gait is a common gait deviation observed in individuals with stroke or cerebral palsy [1]. The exaggerated ankle plantarflexion at initial foot contact typical of equinus gait has been attributed to stiff or spastic ankle plantarflexors, premature activity of the ankle plantarflexors, and inadequate dorsiflexion during swing

[1–3]. Equinus foot placement is often associated with subsequent knee hyperextension during stance, but whether there exists a causative relationship between equinus foot placement and abnormal knee extension is unclear.

In normal gait, lower limb stability is promoted by coupling between ankle plantarflexion moments and knee extension during midstance [4]. During stance, soleus, an ankle plantarflexor, restrains forward rotation of the tibia over the foot and helps to extend the knee without the need for quadriceps activity [2,5]. In pathological gait, this mechanism may be disturbed.

Differentiation of the factors that contribute to increased stance phase knee extension in individuals with equinus is

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complicated by changes in the musculoskeletal system triggered directly or indirectly by the injury to the central nervous system. Knee hyperextension associated with equinus has been attributed to increased ankle plantarflexor stiffness from, for example, contractures [2], altered muscle fiber and connective tissue properties [6,7] or changes in mechanical properties of sarcomeres secondary to the injury [8]. Spasticity of the quadriceps and/or plantarflexors, indicated by premature onset or prolonged activation of these muscle groups, may also impair movement [3,9,10]. Although a variety of physiological factors may contribute to increased knee extension, the potential of equinus posture alone (i.e., increased ankle plantarflexion at initial contact) to promote knee extension has not been explored.

Another confounding factor in understanding equinus gait is that the force developed by a muscle depends on its activation and the length and velocity of its fibers. As a muscle is stretched, passive and active forces develop to resist the change in length and restore the state of the musculoskeletal system (e.g., force–length property). Active force developed while a muscle is lengthening can exceed force developed during isometric or concentric contractions (e.g., force–velocity property). Because intrinsic properties of muscle provide a spring-like response to changes in fiber length and velocity, these “preflexes” form the body’s first line of defense against external perturbations (e.g., applied loads and displacements) [11]. Simulation studies have shown that the intrinsic properties of muscle contribute significantly to recovery from static and dynamic perturbations during walking [12]. The role of intrinsic muscle properties in mediating the response of the body to altered foot placement at initial contact observed in equinus gait has not been studied.

A muscle can act to accelerate all of the body segments, and these segment accelerations depend on the body configuration [13]. A muscle-actuated forward dynamic simulation has been used to identify contributions of the ankle plantarflexors to lower limb and trunk accelerations during normal gait [5], and provides an ideal framework to assess the effect of a change in ankle posture at initial contact (equinus) on muscle contributions to segment accelerations; however, such an analysis has not been performed in previous studies of pathological gait.

The purpose of the present study was to investigate the musculoskeletal response to equinus foot placement. Specifically, we addressed the following two questions: (1) does equinus foot placement alone induce greater than normal knee extension during stance phase, and (2) do intrinsic muscle properties mitigate the response at the knee to perturbed ankle position? To address the first question, we increased ankle plantarflexion at initial contact in a forward dynamic simulation of normal walking and observed the changes in knee extension during stance phase. To address the second question, we allowed muscle forces to vary in response to the perturbed ankle position in accordance with their intrinsic force–length–velocity properties and observed

the corresponding changes in knee extension during stance phase.

2. Methods

We developed a two-dimensional biped musculoskeletal model with 9 degrees of freedom consisting of rigid segments representing the trunk, pelvis and lower extremities using SIMM [14]. Musculoskeletal geometry and individual muscle properties were specified for 15 individual muscles per leg [15], grouped into nine muscle sets per leg, with muscles within each set excited with a block pattern defined by an onset, offset and magnitude. Bilateral symmetry in the excitation patterns was assumed. Contraction dynamics were governed by a Hill-type muscle model [16], and activation dynamics were modeled with a first-order differential equation [17]. The equations of motion were derived using SD/FAST (PTC Inc.) and Dynamics Pipeline [18] was used to generate a forward dynamic simulation.

Gait kinematics and ground reaction forces from five healthy males walking overground at comfortable speed (1.5 m/s) were averaged to form an experimental data set [19]. The muscle excitation patterns were determined by a dynamic optimization algorithm that minimized the error between simulated and experimental kinematics and ground reaction forces [20], with particular emphasis on replication of the normal knee kinematics. The nominal simulation reproduced the experimental stance phase knee kinematics (Fig. 1: simulated knee angle within shaded region between 0% and 60% of the gait cycle). Additional details on development of the model and testing of the simulation of normal gait have been described previously [5].

Equinus foot placement was emulated by unilaterally augmenting the ankle angle at foot contact from 10° to 20° plantarflexion. In a first simulation with equinus (equinus

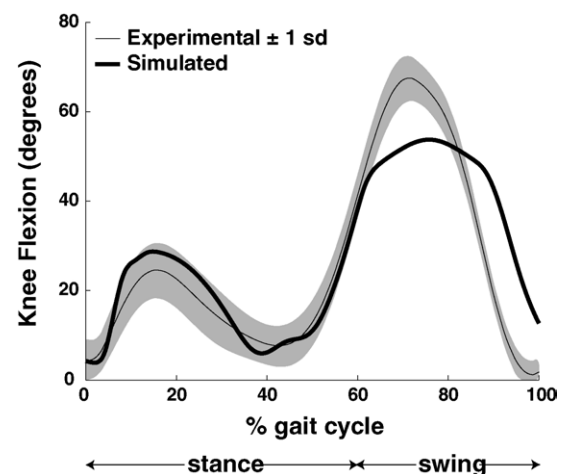


Fig. 1. Measured knee angle (thin line: shaded region = ± 1 sd) during stance (0–60% gait cycle) was reproduced by the forward dynamic simulation (thick line).

posture alone), the nominal muscle forces were applied (i.e., the forces that occurred during the unperturbed simulation of normal gait). Force production by muscles was thus, constrained to be unaffected by any change in kinematics at initial contact or during the ensuing motion. In a second simulation with equinus (equinus with intrinsic response), muscle forces were allowed to respond to changes in kinematics. Thus, this simulation accounted for intrinsic force–length–velocity properties of muscle.

Stance phase knee angle and muscle forces of the ankle plantarflexors (gastrocnemius and soleus) and knee extensors (vastus) as a result of equinus foot placement were compared to the nominal simulation. To further investigate the role of the ankle plantarflexors, the nominal force output by soleus was scaled from 75% to 125% after 10% gait cycle and the resultant effect on knee angle quantified.

3. Results

Increasing ankle plantarflexion by 10° at initial contact, while keeping muscle forces the same as in the normal gait simulation, increased stance phase knee extension dramatically, ultimately resulting in knee hyperextension (Fig. 2: compare solid and dotted lines at $\sim 30\%$ gait cycle). This occurred primarily because equinus foot placement caused the center of pressure of the ground reaction force to be located more anteriorly on the foot. The anterior center of pressure resulted in a net external knee extension moment which was not balanced by the nominal muscle forces, resulting in knee hyperextension.

When muscle forces were permitted to respond to the altered kinematics, increased knee extension occurred, but the effect was less pronounced (Fig. 2: dashed line is between dotted and solid lines at 30% gait cycle). The

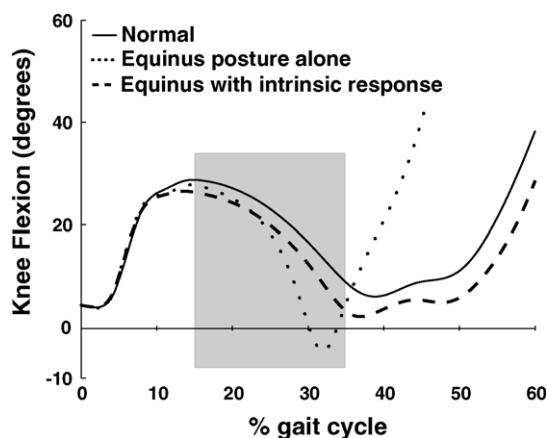


Fig. 2. Effect of equinus foot placement and intrinsic muscle response on knee flexion angle. Equinus foot placement increased stance phase knee extension (compare solid and dotted lines in shaded region). Following hyperextension ($\sim 30\%$ gait cycle), a rebound effect occurred due to passive knee ligaments, causing the knee to flex excessively. With intrinsic muscle response in the model (dashed line), knee extension was less pronounced throughout stance.

reduction in the amount of knee extension was not due to any change in muscle activation, because activation patterns were constrained to be unaltered in this simulation. Instead, the effects of perturbed ankle angle were mediated by the intrinsic force–length–velocity properties of the muscles.

Altered forces in vastus, gastrocnemius and soleus (to a lesser extent) were primarily responsible for mediating the effect of the perturbed kinematics. Increased ankle plantarflexion at initial contact caused soleus and gastrocnemius fibers to be shorter than normal, which decreased their force production. However, both muscles lengthened faster during the loading response, which increased force production. For soleus, the effect of increased lengthening velocity was nearly offset by the effect of shorter fibers, and therefore, there was a very small net increase in soleus force between 5% and 15% gait cycle. The net effect of the altered kinematics on gastrocnemius was force enhancement; which increased the knee flexion moment and reduced knee extension in early stance relative to the effect when muscle forces were not allowed to respond to the equinus posture perturbation.

Increased knee extension with equinus caused vastus fibers to be shorter than normal during concentric contraction in midstance (between 15% and 35% gait cycle); this decreased vastus force production and knee extension relative to the effect when muscle forces were not allowed to respond to the equinus posture perturbation. Thus, the net effect of intrinsic force–length–velocity properties of gastrocnemius and vastus was to reduce the knee extension moment during midstance, which partially compensated for abnormal knee extension that occurred when muscles were not allowed to respond to the equinus posture perturbation.

When soleus force was increased or decreased in isolation (i.e., all other muscle forces were retained), knee extension dramatically increased or decreased (Fig. 3a: closed and open circles, respectively). However, these effects were reduced when the other muscles were allowed to change their force production in response to the changed kinematics (compare Fig. 3b with Fig. 3a).

4. Discussion

We perturbed a forward dynamic simulation of normal walking to study the effects of altered ankle position at initial contact on musculoskeletal dynamics in the absence of changes to the muscle coordination pattern. Equinus foot placement resulted in knee hyperextension. Abnormal knee extension was mitigated substantially by the intrinsic force–length–velocity properties of muscles. Perturbation of soleus force confirmed that the soleus muscle extends the knee during stance, and revealed that complex interactions among muscles reduced the effect of the force perturbations.

Muscle coordination was constrained to the same pattern exhibited in normal walking. In this way, the sensitivity of

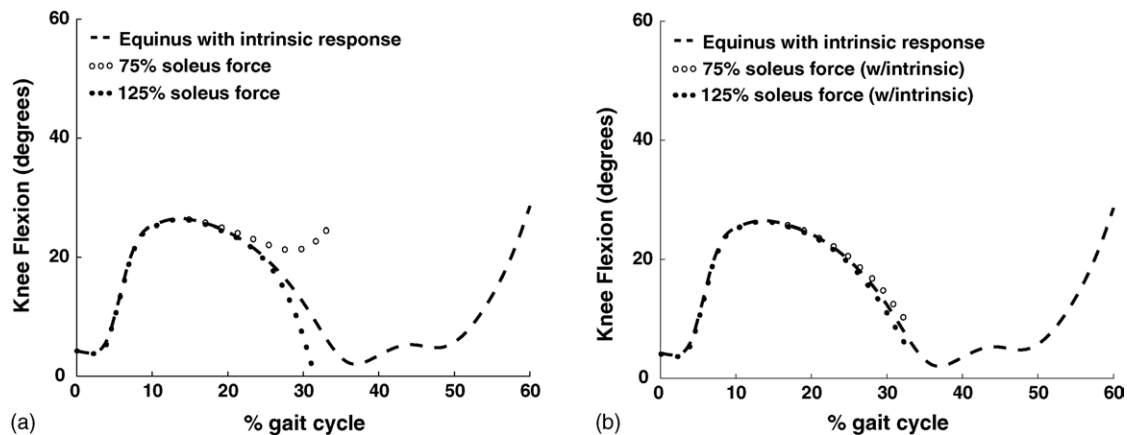


Fig. 3. Changes in knee flexion angle with altered soleus force. (a) As soleus force increased from 75% to 125% of the nominal force, with other muscle forces unaltered, knee extension increased. (b) When intrinsic response of other muscles occurred, an increase in soleus force acted to extend the knee, but the effect was less pronounced.

knee motion to changes in limb posture and muscle forces was isolated. However, maintaining the normal muscle activation pattern does not represent neural compensation (i.e., known changes to the muscle coordination pattern), which may exacerbate knee extension (e.g., spastic quadriceps) [9,21] or prevent hyperextension (e.g., act to flex the knee at initial contact) [22,23]. We believe that better understanding changes in muscle action due to equinus foot placement is a valuable first step, which logically precedes developing simulations of pathological gait to investigate the underlying muscle coordination patterns and potential compensatory strategies. Simulations investigating such patterns and strategies are of great interest to the gait community and are the focus of our ongoing work.

Since the muscle excitation pattern used in the nominal simulation reproduces steady state gait kinematics and ground reaction forces [5], the perturbations applied in this study result in non-steady state solutions. Depending on the magnitude of the perturbation, the resulting simulation may not be physically meaningful if the simulation deviates too far from observed trajectories. Thus, we chose to study the qualitative effects of a change in initial ankle posture over a short period of time after the perturbation. Because our objective was to isolate the effect of increased ankle plantarflexion at initial contact, we did not prescribe abnormal hip and knee angles, which may also be present in individuals with equinus gait [1]. Additionally, the implications of prolonged excess ankle plantarflexion, such as with equinus contracture or “toe walking”, were not addressed by the current study.

The current model utilizes generic musculoskeletal geometry [15], and does not reflect the altered properties of muscle, tendon or ligaments frequently observed in individuals with stroke or cerebral palsy. Typically, excess ankle plantarflexion at initial contact is associated with stiff ankle plantarflexors. Muscle force output is enhanced by increased stiffness due to a change in mechanical properties

of muscle or tendon. Our perturbation studies have shown that increased soleus force results in increased midstance knee extension (see Fig. 3). Although our model does not account for mechanical changes in muscle properties secondary to pathology, such alterations would exacerbate the effect of equinus foot placement on knee extension (confirmed with unpublished sensitivity study).

Previous perturbation studies have demonstrated that the force–velocity properties of muscle offer strong resistance to external perturbations, while force–length properties provide little restorative force [11,12]. In this study, intrinsic muscle responses arising from force–length–velocity properties were found to promote stability of the musculoskeletal system, prevent knee hyperextension, and restore joint motion to near normal. This partial compensation was due primarily to the increased lengthening velocity of gastrocnemius during the loading response and shortened vastus fibers in midstance. With increased force production in the presence of equinus, the gastrocnemius was capable of providing increased resistance to knee extension because of the increase in its knee flexion moment. Additionally, the force output of vastus, which acts to extend the knee, was reduced compared to normal gait. Thus, both the force–length and force–velocity properties of muscles contributed to reducing the effect of equinus foot placement on knee extension.

Abnormal stance phase knee extension associated with equinus foot placement has been attributed to spastic quadriceps, premature activity of the ankle plantarflexors, or increased passive plantarflexor stiffness. It is likely that the observed knee extension due to equinus posture alone found in this study would be exacerbated by these physiological factors. Increased quadriceps activity would increase the knee extension moment exerted by vastus, and instigate further knee extension. In addition, passive muscle adaptation that effectively stiffens soleus and augments its force output would also amplify the contribution of soleus to knee extension.

In some cases, changes in coordination patterns may exacerbate knee hyperextension; however, individuals with artificially induced equinus (taped ankles) adopted effective compensatory strategies and exhibited reduced step length and increased knee flexion at initial contact [23]. Similar gait characteristics have been observed in able-bodied subjects during toe walking [24], healthy women walking in high-heeled shoes [25], and individuals with post-stroke hemiparesis [26].

Consistent with the results of this study, restoration of heel contact for individuals with equinus may improve stance phase knee kinematics. This is commonly achieved with prescription of an ankle–foot orthosis, provided that the patient has adequate knee extensor strength [27,28]. Surgical intervention for equinus in cerebral palsy often involves lengthening of the Achilles tendon; this may reduce the effect of soleus on stance phase knee extension, but may also cause the undesirable reduction in ankle plantarflexor force production during late stance [29].

This study has isolated the effect of initial ankle posture on stance phase knee kinematics. The effect of equinus foot placement on knee extension was explored and demonstrated that distal muscle dysfunction adversely affects proximal joint and segment motion. We have shown that intrinsic force–length–velocity properties of muscle provide beneficial compensation in the presence of equinus foot placement. This simulation paradigm offers a valuable complement to experimental gait analysis by providing insight into the interaction between musculoskeletal dynamics and intrinsic muscle properties in pathological gait.

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References

- [1] Gage JR. Gait analysis in cerebral palsy. New York, NY: MacKeith Press; 1991.
- [2] Perry J. Gait analysis: normal and pathological function. Thorofare, NJ: Slack Inc.; 1992.
- [3] Knutson E, Richards C. Different types of disturbed motor control in gait of hemiparetic patients. *Brain* 1979;102:405–30.
- [4] Gage JR, Novacheck TF. An update on the treatment of gait problems in cerebral palsy. *J Pediatr Orthop B* 2001;10:265–74.
- [5] Neptune RR, Kautz SA, Zajac FE. Contributions of the individual ankle plantar flexors to support, forward progression and swing initiation during walking. *J Biomech* 2001;34:1387–98.
- [6] Dietz V, Quintern J, Berger W. Electrophysiological studies of gait in spasticity and rigidity. Evidence that altered mechanical properties of muscle contribute to hypertonia. *Brain* 1981;104:431–49.
- [7] Berger W, Horstmann G, Dietz V. Tension development and muscle activation in the leg during gait in spastic hemiparesis: independence of muscle hypertonia and exaggerated stretch reflexes. *J Neurol Neurosurg Psychiatry* 1984;47:1029–33.
- [8] Friden J, Lieber RL. Spastic muscle cells are shorter and stiffer than normal cells. *Muscle Nerve* 2003;26:157–64.
- [9] Brunnstrom S. Movement therapy in hemiplegia. New York: Harper & Row; 1970.
- [10] Dietz V, Berger W. Normal and impaired regulation of muscle stiffness in gait: a new hypothesis about muscle hypertonia. *Exp Neurol* 1983;79:680–7.
- [11] Brown IE, Loeb GE. A reductionist approach to creating and using neuromusculoskeletal models. In: Winters JM, Crago PE, editors. *Biomechanics and neural control of posture and movement*. New York, NY: Springer-Verlag; 2000. p. 148–63.
- [12] Gerritsen KG, van den Bogert AJ, Hulliger M, Zernicke RF. Intrinsic muscle properties facilitate locomotor control—a computer simulation study. *Motor Control* 1998;2:206–20.
- [13] Zajac FE, Gordon ME. Determining muscle's force and action in multi-articular movement. *Exerc Sport Sci Rev* 1989;17:187–230.
- [14] Delp SL, Loan JP. A graphics-based software system to develop and analyze models of musculoskeletal structures. *Comput Biol Med* 1995;25:21–34.
- [15] Delp SL, Loan JP, Hoy MG, Zajac FE, Topp EL, Rosen JM. An interactive graphics-based model of the lower extremity to study orthopaedic surgical procedures. *IEEE Trans Biomed Eng* 1990;37:757–67.
- [16] Schutte LM, Rodgers MM, Zajac FE, Glaser RM. Improving the efficacy of electrical stimulation-induced leg cycle ergometry: an analysis based on a dynamic musculoskeletal model. *IEEE Trans Rehabil Eng* 1993;1:109–24.
- [17] Raasch CC, Zajac FE, Ma B, Levine WS. Muscle coordination of maximum-speed pedaling. *J Biomech* 1997;30:595–602.
- [18] Delp SL, Loan JP. A computational framework for simulating and analyzing human and animal movement. *Comput Sci Eng* 2000; 46–55.
- [19] Andriacchi TP, Natarajan RN, Hurwitz DE. Musculoskeletal Dynamics, Locomotion and Clinical Applications. In: Mow VC, Hayes WC, editors. *Basic orthopaedic biomechanics*. New York, NY: Raven Press Ltd.; 1997. p. 37–68.
- [20] Neptune RR. Optimization algorithm performance in determining optimal controls in human movement analyses. *J Biomech Eng* 1999;121:249–52.
- [21] Sutherland DH, Davids JR. Common gait abnormalities of the knee in cerebral palsy. *Clin Orthop* 1993;288:139–47.
- [22] Winters TF, Gage JR, Hicks R. Gait patterns in spastic hemiplegia in children and young adults. *J Bone Joint Surg* 1987;69-A:437–41.
- [23] McMulkin ML, Barr KM, Goodman MJ, Menown JL, West JM, Vander Linde DW. Secondary gait compensations in normal adults with an imposed unilateral ankle equinus constraint. In: Eighth Annual Meeting of the Gait and Clinical Movement Analysis Society. 2003. p. 1130–114.
- [24] Kerrigan DC, Riley PO, Rogan S, Burke DT. Compensatory advantages of toe walking. *Arch Phys Med Rehabil* 2000;81:38–44.
- [25] Kerrigan DC, Todd MK, Riley PO. Knee osteoarthritis and high-heeled shoes. *Lancet* 1998;351:1399–402.
- [26] Olney SJ, Richards C. Hemiparetic gait following stroke. Part I: Characteristics. *Gait Posture* 1996;4:136–48.
- [27] Simon SR, Deutsch SD, Nuzzo RM, Mansour MJ, Jackson JL, Koskinen M, Rosenthal RK. Genu recurvatum in spastic cerebral palsy. *J Bone Joint Surg* 1978;60-A:882–94.
- [28] Lehmann JF, Condon SM, Price R, deLateur BJ. Gait abnormalities in hemiplegia: their correction by ankle–foot orthoses. *Arch Phys Med Rehabil* 1987;68:763–71.
- [29] Delp SL, Zajac FE. Force- and moment-generating capacity of lower-extremity muscles before and after tendon lengthening. *Clin Orthop* 1992;247–59.