

Can Intrinsic Mechanics of Equinus Result in Knee Extension?

JS Higginson^{1,2}, FE Zajac^{1,2,3}, SA Kautz^{4,5,6}, RR Neptune⁷, SL Delp^{1,2} and CG Bugar^{1,3}

¹VA Palo Alto Rehab. R&D Ctr., Palo Alto, CA; ²Dept of Mech. Eng. and ³Div. of Physical Med. and Rehab., Stanford University, Stanford, CA; ⁴Brain Rehab. Research Ctr., Malcom Randall VA Medical Ctr., Gainesville, FL; ⁵Dept. of Physical Therapy, University of Florida, Gainesville, FL; ⁶Brooks Ctr. for Rehab. Sci., University of Florida, Gainesville, FL; ⁷Dept. of Mech. Eng., University of Texas, Austin, TX

Introduction: Equinus gait, characterized by excess ankle plantarflexion at initial contact, is common in persons with stroke and cerebral palsy. Knee hyperextension is frequently associated with equinus.¹ However, it is unclear whether knee hyperextension results from altered muscle excitation patterns, changes in muscle length and velocity, or perturbed intersegmental dynamics in response to equinus foot placement. The purpose of this study is to examine whether knee extension can be caused by the intrinsic mechanical response associated with equinus foot placement in the absence of active compensation.

Statement of Clinical Significance: Improved understanding of the effect of equinus on knee extension will provide a basis for the design of therapeutic interventions which target altered mechanics or abnormal muscle control for enhanced gait velocity, symmetry, stability and pain reduction.

Methodology: A forward dynamic simulation of normal gait² was perturbed to study the effect of equinus on musculoskeletal mechanics during stance. Optimal muscle excitations for the nominal solution actuated a 9 DOF, 11 segment musculoskeletal model.³ The equinus perturbation was introduced by augmenting the ankle angle just prior to initial contact from 1° dorsiflexion (normal) to 20° plantarflexion (equinus) while maintaining the normal muscle excitation pattern and the remaining limb configuration. The contributions of major lower extremity muscles (soleus, SOL, gastrocnemius, GAS, and vastus, VAS) to knee motion were investigated.

Results: Immediately following initial contact with equinus foot placement, SOL fibers are shorter but stretched more rapidly than normal. Due to the inherent properties of the muscle-tendon complex (i.e. force-length-velocity property), passive force of SOL during double support is increased. Since the foot is on the ground, the increased plantarflexor force restrains the tibia, causing the knee to extend slightly more than normal (Figure 1, transition to single support: thin and thick solid lines diverge). Until this transition, VAS fiber length and force generation are virtually unchanged.

In single support, excess ankle plantarflexion decreases fiber length and force generation by SOL. Despite this reduction in force, mechanical analyses reveal that SOL (and GAS) restrain forward tibia motion still more, causing the knee to flex less than normal. This increased knee extension shortens VAS fibers which reduces their force output and knee extensor moment and partially counteracts the action of the plantarflexors to extend the knee.

Because the action of the plantarflexors is so strong, the net effect due to muscles is increased knee extension throughout single support (Figure 1: thin vs. thick solid lines in single support).

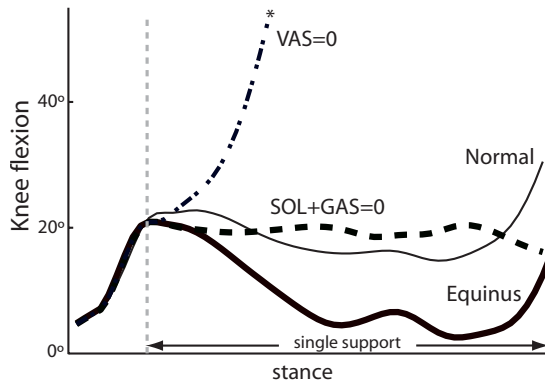


Figure 1. Knee flexion angle for normal (thin) and equinus (thick) gait during stance phase. With equinus foot placement, the knee extends more during single support. Decreased force output of the ankle plantarflexors (SOL+GAS, dashed) or knee extensors (VAS, dash-dot) results in less knee extension. Vertical line indicates transition to single support and onset of perturbation.

The relative contribution of specific muscle groups to knee motion was determined by isolating the force generated by the ankle plantarflexors and knee extensors during single support of the equinus gait simulation. When the force of each of these muscle groups (SOL+GAS, VAS) was eliminated with all other muscle forces retained, the knee flexed more compared to the equinus gait simulation (Figure 1: difference between dashed line for SOL+GAS or dashed-dotted line for VAS and thick solid line). Despite the observed reduction in SOL and VAS forces in single support of equinus gait, the new limb configuration enhances the effect of these muscles on knee extension. In summary, equinus induces a small change in limb configuration at the transition to single support which has significant effects on muscle contributions to knee extension throughout stance.

Discussion: In the presence of equinus, the fundamental mechanics of the musculoskeletal system can extend the knee more than normal. Because this study neglects active compensation that may be introduced to enhance stability or protect the joints, we can isolate the effect of intrinsic mechanical responses to equinus. It should be noted that muscle activity associated with spasticity following brain injury has not been included in the model. However, we would expect such activity to exacerbate knee extension. The results of this study clearly indicate that intrinsic mechanisms, via muscle properties and intersegmental dynamics, can have a significant effect on knee extension in the presence of equinus and should be considered in the treatment of this gait abnormality.

References:

1. Perry (1992) *Gait Analysis: Normal and Pathological Function*. Slack Inc.
2. Neptune et al. *J Biomech*, 34, 1387-98, 2001.
3. Delp et al. *IEEE Trans Biomed Eng*, 37, 757-67, 1990.

Acknowledgements: This work was supported by the Rehabilitation Research & Development Service of the Department of Veterans Affairs.