How Robust is Human Gait to Shortened Calf Muscles?

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Contents

- Introduction
- Aims
- Methods
- Results
- Discussion
- Future Work
- Conclusions
- References

Introduction

Movement abnormalities, which are symptoms of many neuromuscular diseases and conditions, can be caused by aberrations in one or several of the parameters governing muscle excitation, activation, and mechanics. Of the parameters governing muscle mechanics, the optimal fiber length of a muscle is the most important mechanical parameter in determining how much isometric force it can produce [1].

Contracture is a condition in which muscle fibers are permanently shortened to 45% or more of normal length [5]. Several populations, including post-stroke victims, patients with Muscular Dystrophy, and patients with Cerebral Palsy suffer from contracture [8,2]. Contracture is often present alongside muscle weakness and abnormal muscle excitation.

The individual impact of each of these abnormalities on movement impairment has not been extensively studied. Current advances in musculoskeletal modeling software offer the unprecedented opportunity to examine the effects of these abnormalities in isolation. A study that has recently been conducted on the effect of muscle weakness on human gait revealed that of all of the muscles and muscle groups important to human gait, the triceps surae, or the medial and lateral gastrocnemius and soleus muscles, are among those most sensitive to muscle weakness [7].

Aims

This study aims to perform a similar sensitivity analysis on human gait, wherein the parameter of interest is optimal fiber length. This study examines the effects of isolated contracture of the gastrocnemius (both medial and lateral) as well as contracture of the entire triceps surae (gastrocnemius and soleus), as these are two conditions frequently encountered in Cerebral Palsy patients, and two situations meritting different tendon transfer surgical strategies [5].

Questions to answer:

1. What is the effect of calf muscle shortening on tracking normal walking gait kinematics?
2. What are the compensatory activation strategies of the remaining muscles?
3. How much do total muscle forces and total muscle stresses increase with calf muscle shortening?

Methods

The Gait2392 OpenSim full-body muscle model was used along with Chand John walking data [3]. This experimental data is provided through OpenSim resources as an example for use with the Gait2392 model. An Umberger metabolic probe was added to the model for later analysis. The study process described below is also outlined in Figure 1.
Scaling: The generic musculoskeletal model was scaled to the size of the subject and model markers were configured to match marker locations in the static pose of the experimental data. Sternum, head, and left and right Acromion markers were set to zero weighting so that the poor assumption that the torso is a rigid body would not add significant error to the simulation. This was also reasonable because the region of focus is on the ankle and plantarflexors, which are among the muscles and joints furthest away from the torso.

Inverse Kinematics: An inverse kinematics analysis was then performed, allowing the musculoskeletal model to reproduce the experimental data.

Residual Reduction Algorithm: A residual reduction algorithm was performed to reduce error in center of mass location in the model by reducing the residual about the torso center of mass.

Computed Muscle Control: Computed Muscle Control [6] was used to find the optimal muscle activations that would enable the model to reproduce the experimental kinematics. The gastrocnemius excitation controls were set to a maximum of 0.65 so that the simulation could circumvent limitations in the CMC Tool and more closely match measured EMG data during walking. Kinematics were filtered at 6 Hz.

Another aspect of this study was to test the OpenSim 3.1 Umberger probe as an indicator of metabolic cost. The square of the muscle force over total muscle force was calculated as has been previously done to estimate total cost of each muscle examined and of the whole body [7]. These results were then compared to the increase in metabolic costs for individual muscles and for the whole body for each scenario of contracture. Muscles of interest included the other plantarflexors and several muscles known to be important to walking [7]. For these muscles, the activations, forces, and metabolics were examined across studies. Additionally, kinematic error and reserve ankle and knee moments were tracked throughout the simulations.

Results

Notable results from the contracture studies, including passive forces, activations, reserve actuators, and metabolic costs are shown in Figures 2–5. All plots represent one gait cycle as defined by the time between consecutive right foot heel strike.
Figure 2: Passive forces along muscle fibers
**Figure 3:** Activations in select muscles

**Figure 4:** Reserve Ankle Actuator Forces. Left: GAS Contracture only. Right: GAS and SOL Contracture
Discussion

Passive forces in the calf musculotendon units increase dramatically with increasing contracture (Figure 2). As the triceps surae muscles are shortened, high passive forces stretch the shortened muscles, causing them to be further away (to the right on the force-length curve) from optimal operating lengths throughout the gait cycle. Because they are no longer able to produce the forces necessary in normal gait, the Computed Muscle Control opts not to activate these muscles at all during much of the gait cycle. Activating them is costly and increasingly ineffective.

As a first effort to compensate for excessive passive plantarflexion forces in the calf muscles, the tibialis anterior (dorsiflexor) muscle is increasingly activated (Figure 3). However, at a certain point, the tibialis anterior cannot sufficiently compensate, and the Computed Muscle Control tool must make use of the generalized ankle reserve forces to allow the diseased model to still execute the desired normal gait kinematics without large error (Figure 4). Considering that maximum required ankle moment for this simulation was about 40 Newton-meters, and that reserves greater than 5% of the total force needed are conventionally an indicator of failure to track, these results generally indicate that a patients with isolated gastrocnemius contracture beyond 20-30% and patients with combined gastrocnemius and soleus contracture beyond 10-20% are unlikely to track normal gait. However, the baseline case with no shortening utilized reserves slightly above this 5% criterion, so caution should be used in affixing “successful” or “unsuccessful” tracking to these different conditions.

What can be observed is the following. Heel strike/early stance phase (at the beginning and very end of both plots) are difficult for patients exhibiting both kinds of contracture. This effect reflects clinical observations of Cerebral Palsy patients [9]. However, tracking the foot to be flat on the ground rather than plantarflexed during the stance phase requires enormous reserves for only the combined contracture case. This implies that combined contracture is more likely than isolated contracture to lead to equinus.

Unlike the sensitivity study on weakness, metabolic cost studies did not reflect a consistent trend of increasing metabolic cost. This may be partially due to the fact that reserve forces account for an increasing proportion of the work done to execute the desired motion and also that the simulation results lose meaning when reserves are too high. The muscle stresses cost plot indicates that the tibialis anterior muscle becomes more costly with higher degrees of contracture. One might expect this muscle to fatigue in a patient with mild contracture who attempts to walk normally, but further work must be done to confirm this. Perhaps, along with tendon transfer surgery, strengthening the tibialis anterior can help compensate for the abnormal passive forces developed in the triceps surae muscles.
The Umberger metabolic probe data did not reflect the same cost distribution as the muscle stresses cost. Because this tool is still in development, more studies should be conducted in order to understand the nature of the discrepancy.

**Future Work**

Future work could address shortcomings of the current study, which include the following:

- Model mass was not decreased in conjunction with decreased optimal fiber length.
- Weakness and spasticity, which often accompany contracture, were not included in the model.
- Tendon stiffness and cross sectional area, which can increase in subjects with shorter muscles, was kept constant.
- Contracture is often present in more muscles than just the gastrocnemius and soleus, although only these muscles were considered here.
- The manual gastrocnemius constraint set to 0.65 was an estimate based off of EMG measurements of gastrocnemius activity from a different set of walking data used as a proxy [7]. For better consistency, EMG data for a specific set of gait kinematics should be applied to the Computed Muscle Control constraints.

Until recently, the ability to measure the amount of contracture present in a patient, in vivo, was not possible. With current imaging advancements that allow for in vivo sarcomere imaging, sarcomere lengths in conjunction with MRI or ultrasonography fiber imaging could give a clear picture of the amount of contracture present in a patient [4]. The potential to include this information in biomechanical simulation holds great promise in allowing for more personalized surgical treatment.

Tendon transfer surgery, which is often performed in order to restore range of motion to patients with Cerebral Palsy, can result in muscle weakening by putting muscles on a disadvantageous portion of the force-length curve. The existence of contracture can amplify this weakening side effect [5]. Therefore, the optimal balance between increased range of motion and minimal muscle weakening for equinus treatment could be better reached using information on degree and type of contracture present in a subject.

As an example, the condition of 45% contracture in both the soleus and gastrocnemius was determined previously to be 1 cm of gastrocnemius aponeurosis lengthening and 1 cm Achilles tendon lengthening. This surgery was simulated and the gait tracking kinematics study was re-run. Results, which involved dramatically decreased passive forces and use of ankle reserves, can be seen in Figure 6. Thus, further work might include building an optimizer to find a patient-specific optimal treatment strategy, given the type and degree of equinus.

Other sensitivity studies could be performed in the same manner to understand the robustness of gait to pennation angle and activation changes. Lastly, more work should be done to understand the discrepancies between the muscle stresses costs and the Umberger metabolic probe costs.

![Figure 6: Optimal tendon transfer surgery as simulated in [5], applied to tracking normal walking gait. Differential tendon lengthening dramatically reduces passive forces in the triceps surae and thus the use of ankle reserves.](image)

**Conclusions**
In conclusion, a sensitivity study was performed in order to better understand the robustness of human gait to shortened calf muscles. Combined contracture seems to inhibit normal walking more than isolated contracture when compared across similar fiber shortening percentages. Based on the relative weakness observed throughout the gait cycle, the tip toe walk (equinus), which arises from a difficulty in dorsiflexion during the stance phase of gait, is more likely a result of combined contracture than of isolated gastrocnemius contracture. The results of these simulations are available for further study on simtk.org. Further, a framework has been presented to include information on specific types and degrees of contracture into biomechanical modeling and simulation in order to enable the design of personalized tendon transfer surgery techniques for patients with movement disorders.

References