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Short communication

A biomechanical model to estimate corrective changes in muscle activation patterns for stroke patients

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ABSTRACT

We have created a model to estimate the corrective changes in muscle activation patterns needed for a person who has had a stroke to walk with an improved gait—nearing that of an unimpaired person. Using this model, we examined how different functional electrical stimulation (FES) protocols would alter gait patterns. The approach is based on an electromyographically (EMG)-driven model to estimate joint moments. Different stimulation protocols were examined, which generated different corrective muscle activation patterns. These approaches grouped the muscles together into flexor and extensor groups (to simulate FES using surface electrodes) or left each muscle to vary independently (to simulate FES using intramuscular electrodes). In addition, we limited the maximal change in muscle activation (to reduce fatigue). We observed that with the two protocols (grouped and ungrouped muscles), the calculated corrective changes in muscle activation yielded improved joint moments nearly matching those of unimpaired subjects. The protocols yielded different muscle activation patterns, which could be selected based on practical condition. These calculated corrective muscle activation changes can be used in studying FES protocols, to determine the feasibility of gait retraining with FES for a given subject and to determine which protocols are most reasonable.

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1. Introduction

Functional electrical stimulation (FES) has been used in the rehabilitation of post-stroke patients (Peckham and Knutson, 2005). It is important to know how much additional activity should be added during FES. Many control methods have been used to derive the required electrical stimulation patterns (Popovic et al., 2003; Jezernik et al., 2004). These investigators employed different machine learning algorithms to account for the nonlinear relationship between electrical stimulation and patient's kinematic output. However, these methods did not determine this relationship by directly accounting for the biomechanics of the human neuromuscular system, through which the neurological control strategies of human movement may be characterized more accurately.

We have developed electromyographically (EMG)-driven biomechanical models to estimate muscle forces and joint moments (Lloyd and Besier, 2003; Buchanan et al., 2004). Since these models determine muscle forces based on recorded EMG data, they can be used to determine how different muscle activation

patterns influence joint moments, and thus can be used to study FES protocols.

In this study, we constructed a biomechanical model to estimate the corrective increases in muscle activation patterns that would enable post-stroke patients to walk with a similar joint kinematics to that of an unimpaired person. Our goal is to provide a platform for studying FES protocols, through which the appropriate stimulation patterns can be determined to achieve the desired normal movement.

2. Methods

In this study we focused on the ankle joint model. We included the main contributors to plantar/dorsiflexion: tibialis anterior (TA), medial and lateral gastrocnemius (MG and LG), and soleus (Sol). Our model is generic and can be applied to any joint given the necessary anatomical and physiological data.

2.1. EMG-driven model to estimate muscle forces and joint moments

The EMG-driven model was developed based on a Hill-type muscle model to calculate individual muscle forces and joint moments (Lloyd and Besier, 2003; Buchanan et al., 2004). The muscle characteristics including insertion points, pennation angle, and maximum isometric forces were adapted from literature (Delp et al., 1990). Some of the parameters in our model were subject-specific and difficult to obtain, so a calibration process was used to tune these parameters for each subject (Heine et al., 2003). Once the parameters were tuned, the model could

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then be used to predict muscle forces and joint moments for new muscle activation patterns.

2.2. Optimization model to estimate corrective changes of muscle activation patterns

Using the tuned EMG-driven model, we modeled electrical stimulation by increasing the EMGs, optimally adjusting them using a parallel version of constrained simulated annealing algorithm (Wah and Wang, 1999) (Fig. 1). Data from average unimpaired subjects' gait trials were used to determine the corrected joint angles θ for stroke patients, resembling a normal kinematic pattern. We also determined the desired joint moments, M_D , through scaling the joint moment profile of an unimpaired subject by body mass, height, and walking speed (Lelas et al., 2003), which would generate the corrected joint angles θ for stroke patients. These data were used as input, and the new EMG pattern was then optimized to produce the desired joint moment. We chose to minimize the sum of Δ EMGs as the cost function, which is related to the muscle fatigue caused by electrical stimulation. (In this context, "EMG" means the normalized, rectified, and filtered EMG signal.)

2.3. Data collection and model calculations

Data were collected from two post-stroke patients who could walk without assistance (Table 1). EMG, joint position, and force plate data were collected during four walking trials for subjects 1 and 2. EMGs were collected using surface electrodes from each muscle. Maximum voluntary contraction (MVC) trials were collected for normalization of EMG. The experimental protocol was approved by

the Human Subjects Review Board of University of Delaware, and the subjects gave an informed consent before the data collection.

The parameters in the EMG-driven models were tuned using the initial walking trial for each subject. The calibrated models were then used to predict the other walking trials of the subjects. This paradigm is a good test of the model's predictability of novel trials, because the EMG data were variable from trial to trial, unlike data for healthy subjects.

The new EMG patterns were then calculated for the initial trials of each subject using the optimization model. Two EMG changing algorithms were implemented. In Algorithm 1, the EMGs of every muscle were altered separately, representing the FES protocol of using epimysial and intramuscular electrodes to stimulate each individual muscle. In Algorithm 2, the EMGs of the muscles were grouped together as either dorsiflexors or plantar flexors and altered simultaneously, representing the FES protocol of using surface electrode to stimulate one muscle group. In addition, a constraint on Δ EMG limit ($0 \leq \Delta$ EMG < 0.6) was added. This was done to avoid too large Δ EMG values, which may cause the patient's discomfort for intense electrical stimulation and induce fatigue. Different upper limit may be used depending on the patient's comfortable tolerance.

3. Results

The predicted joint moments of our EMG-driven model were close to inverse dynamic joint moments, which verified that the calibrated model could be used to predict novel trials (Table 2).

Stroke subjects' 1 and 2 ankle joint angles in walking trial 1 were different from those of the average unimpaired subject (Winter, 1990; Fig. 2). We compared the original ankle joint moment of each post-stroke patient with the desired unimpaired patterns (Fig. 3 for subject 1). It was determined that the ankle joint moment should be altered during stance phase to match the unimpaired profile. We added the late swing phase as a buffering interval to account for the effect of electromechanical delay. Therefore, late swing phase and stance phase were chosen as the data of interest.

The original EMG of TA for subject 1 was small (Fig. 4). After adding the calculated Δ EMG (Fig. 5), the new moment was close to the desired moment (Fig. 3), and the kinematics of the subject would shift from Line "Stroke subject 1" to Line "Average healthy" in Fig. 2. The increase of EMG in dorsiflexor (TA) during late swing phase and early stance phase was needed to achieve a normal dorsiflexion moment. We observed similar results on subject 2. The statistical results for subjects 1 and 2 demonstrated that both algorithms generated similar results of joint moment profiles (Table 3).

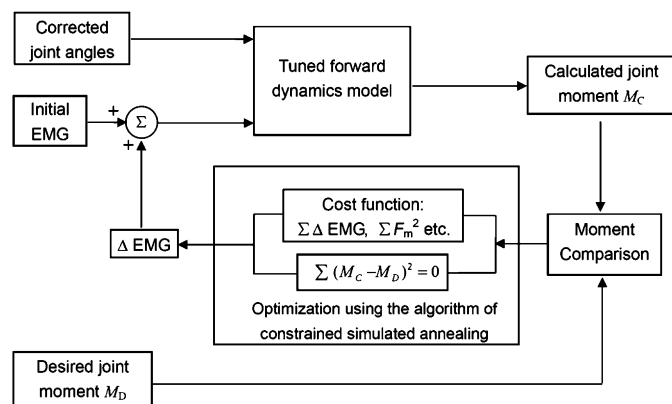


Fig. 1. Theoretical flow of the optimization model. In this model, corrected joint angles and desired joint moment were the inputs, and we used constrained simulated annealing algorithm to calculate the Δ EMGs, which were required to achieve the desired joint moment profile for the stroke subjects. The new EMG profiles were generated using a cubic spline interpolation algorithm with 16 control nodes evenly distributed across the time period.

Table 1
Information of the two subjects

Subject	Gender	Age (years)	Time since stroke (years)	Hemiplegic side	Modified lower extremity Fugl-Meyer Score
1	M	77	4	Right	32/36
2	M	58	3	Right	28/36

Table 2
Statistical results of calibration and prediction on walking trials of stroke subjects' 1 and 2

Subject		Trial	R^2 value	RMS error (N m)	Normalized RMS error (%)
1	Calibration	1	0.973	1.53	2.48
	Prediction	2–4 (mean (SD))	0.884 (0.041)	6.86 (1.05)	10.33 (1.83)
2	Calibration	1	0.971	3.32	3.03
	Prediction	2–4 (mean (SD))	0.933 (0.016)	10.89 (5.41)	11.93 (5.67)

We calculated the R^2 value, root mean square (RMS) error, and normalized RMS error (normalized to peak-to-peak joint moment) to compare the calibrated and predicted forward dynamic joint moment profiles with the inverse dynamic joint moment profiles.

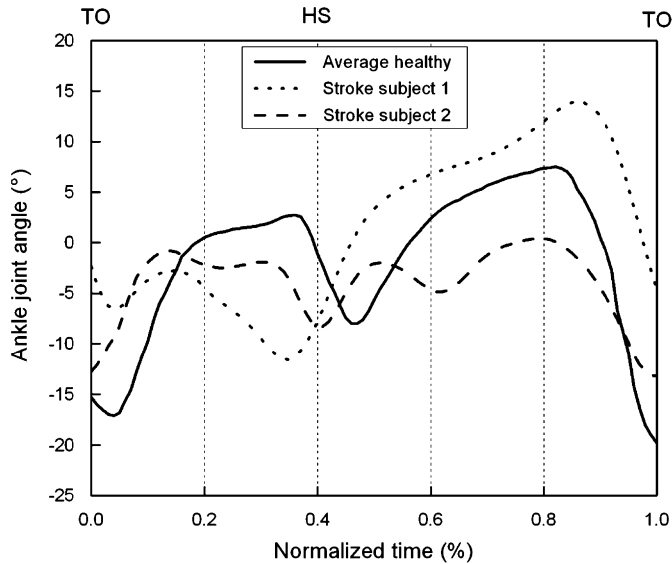


Fig. 2. Ankle joint angles during gait of average unimpaired subject and two stroke subjects. The time axis was normalized to one gait cycle, HS denotes heel strike, and TO denotes toe off. Positive joint angle indicates dorsiflexion. Note that the two stroke subjects had drop-foot during swing phase and smaller plantar flexion during late stance phase.

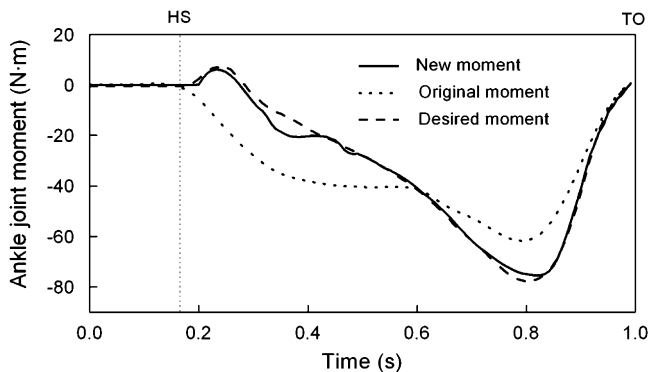


Fig. 3. The results of joint moments using Algorithm 1 for subject 1. The *new moment* is the moment calculated from the new EMG, the *original moment* is the original moment for the stroke patient, and the *desired moment* is derived from the unimpaired subject's trial. Positive moments indicate dorsiflexion. Note that subject 1 lacked a dorsiflexion peak during early stance phase, which needed to be corrected by adding stimulation. After the early dorsiflexion was restored to normal, stronger plantar flexion moment was needed to plantarflex the ankle and finish the push-off by adding stimulation.

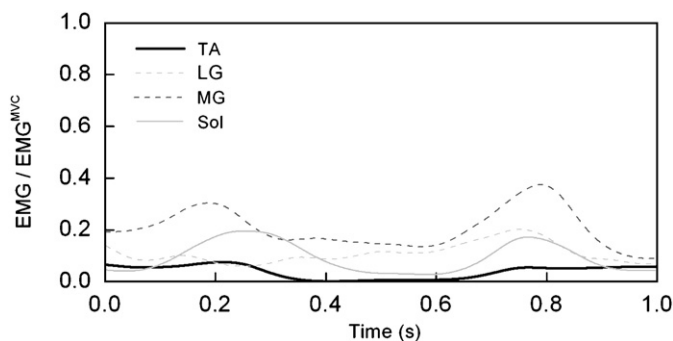


Fig. 4. The original normalized EMG patterns of subject 1. Note the subject had normal TA activity during swing phase, and the activation of TA was small during early stance phase, which supports the demand of using electrical stimulation to activate this weak muscle.

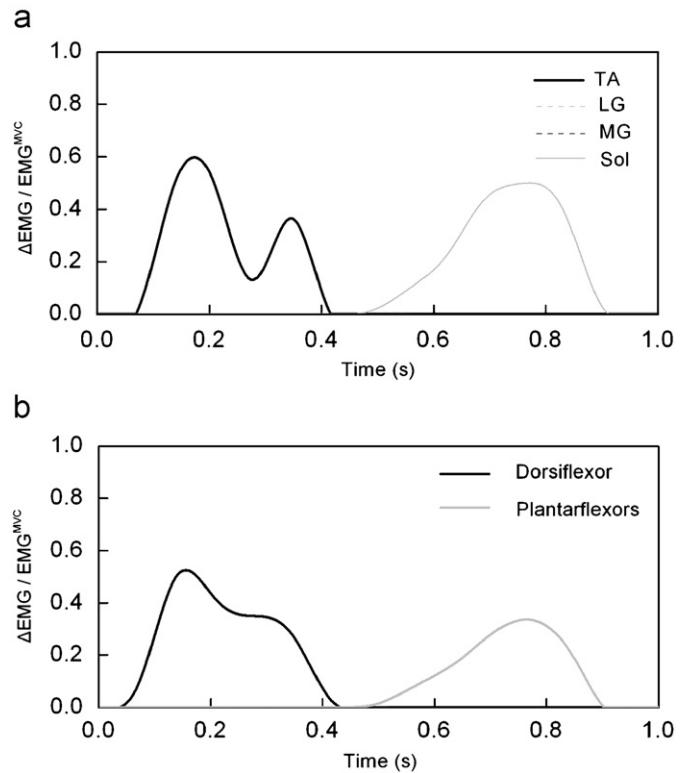


Fig. 5. The results of normalized ΔEMG for subject 1: (a) Algorithm 1 and (b) Algorithm 2. The TA needed to be stimulated over the late swing phase and early stance phase for both algorithms. The soleus was chosen as the only plantar flexor to be stimulated in Algorithm 1, because it would require a smaller amount of stimulation than the gastrocnemius, and thus reduces fatigue.

Table 3

Statistical results of different optimization protocols for stroke subjects 1 and 2

Subject	Protocol	R^2 value	RMS error (N·m)	Normalized RMS error (%)	$\sum \Delta\text{EMG} / \text{EMG}^{\text{MVC}}$
1	Algorithm 1	0.979	2.09	2.47	332.5
	Algorithm 2	0.980	2.10	2.48	368.8
2	Algorithm 1	0.966	7.10	4.69	718.7
	Algorithm 2	0.948	10.48	6.15	641.9

We calculated the R^2 value, root mean square (RMS) error, and normalized RMS error (normalized to peak-to-peak joint moment) to compare the new joint moment profiles after adding stimulation with the desired joint moment profiles. $\sum \Delta\text{EMG} / \text{EMG}^{\text{MVC}}$ is the sum of all normalized ΔEMG for all the four muscles and 1080 time steps (our sampling rate of EMG is 1080 Hz).

4. Discussion

This optimization model was developed based on our EMG-driven model, and took advantage of the EMG-driven model's ability to predict novel trials. We employed this optimization model on two stroke patients' walking trials and got the corrective changes in EMG patterns. These calculated corrective changes could be used as reference data in both stroke patients' gait training with FES.

It was observed that the TA needed to be stimulated during late swing phase and stance phase to achieve normal ankle kinematic patterns for our post-stroke subjects. Since the volitional ankle moment during swing phase is close to zero for both stroke and unimpaired subjects, the early swing phase's

kinematic pattern will be restored to a normal pattern automatically, as long as the late swing and stance phase's kinematic and kinetic patterns have been corrected using stimulation. The TA activity during swing phase in post-stroke subjects was similar to that found in unimpaired subjects, and the patients lacked the normal second peak of TA activity at initial foot contact, which might account for their flat foot walking (Burridge et al., 2001). Our results showed similar results that the TA activity needed to be corrected at heel strike to avoid drop-foot.

Two different optimization protocols were implemented during our calculation, providing different options for different stimulation protocols using surface electrodes and implantable epimysial and intramuscular electrodes. Different optimization protocols may be selected based on clinical judgment and practical condition.

After the corrective muscle activation changes were estimated using our model, the appropriate electrical stimulation patterns could be determined through other developed models to achieve the corrective changes (Riener et al., 1996; Soetanto et al., 2001; O'Keefe et al., 2003). After the stimulation patterns are determined, they could be used as a baseline in open-loop or hybrid (combined feed-forward and feedback) control during an FES intervention.

There are several assumptions and limitations of this study. First, our model does not attempt to balance the moments at the knee, hip as well as the ankle, and our model can be extended to a multi-joint model combining other joints in future study. Second, a ground contact model may be developed instead of our scaling procedure to directly account for the change of ground reaction force after the kinematics is restored to normal, and it can be used to calculate the desired joint moments. Third, other desired joint moment profiles could also be used for different goals during FES intervention. Fourth, we did not account for the change of muscle force–activation relationship caused by stroke, and used the calibrated subject-specific muscle parameters to account for this impaired musculature. Fifth, we also need to recruit more patients and get results on different groups of patients.

In conclusion, we have developed a method to determine changes in muscle activation patterns needed for stroke subjects to correct their gait patterns so that they match those of unimpaired people, and we have demonstrated this in two subjects. The calculated corrective muscle activation changes may provide a baseline reference in open-loop control or hybrid control during FES intervention.

Conflict of interest

None.

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