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Leg Muscle Activation Evoked by Floor Stiffness Perturbations: A Novel Approach to Robot-assisted Gait Rehabilitation

Jeffrey Skidmore and Panagiotis Artemiadis*

Abstract— **Robotic devices have been used in a variety of rehabilitation protocols, including gait rehabilitation after stroke. However, robotic intervention in gait therapy has only produced moderate results compared to conventional physiotherapy. We suggest a novel approach to robotic interventions which takes advantage of inter-limb coordination mechanisms. We hypothesize the existence of a mechanism of inter-leg coordination that may remain intact after a hemiplegic stroke that may be utilized to obtain functional improvement of the impaired leg. One of the most significant advantages of this approach is the safety of the patient, since this does not require any direct manipulation of the impaired leg. In this paper, we focus on designing and applying unilateral perturbations that evoke contralateral leg motions through mechanisms of inter-leg coordination. Real-time control of floor stiffness is utilized to uniquely differentiate force and kinematic feedback, creating novel perturbations. We present results of repeatable and scalable evoked muscle activity of the contralateral tibialis anterior muscle through unilateral stiffness perturbations. We also present a mathematical model that accurately describes the relationship between the magnitude of the stiffness perturbation and the evoked muscle activity, that could result in model-based rehabilitation strategies for impaired walkers. The novel methods and results presented in this paper set the foundation for a paradigm shift of robotic interventions for gait rehabilitation.**

I. Introduction

Stroke is a common and serious health problem throughout the world [1], with a large percentage of stroke survivors only achieving poor functional outcome five years after the onset of stroke [2]. Neuroplasticity is the basic mechanism underlying improvement in functional outcome after stroke [3]. Robotic therapy has been proposed as a more effective alternative to conventional physiotherapy, because robots can easily facilitate the key behavioral signals of neural plasticity including repeatability, intensity, and specificity [4].

A variety of robotic rehabilitation devices have been proposed for gait rehabilitation after stroke (eg. [5]–[8]), but have only produced moderate results compared to conventional physiotherapy [9]–[12]. Currently, there is no evidence that robotic gait training is superior to conventional physiotherapy for either chronic or subacute stroke patients [13].

Due to the cyclic coordination between limbs in human walking, we hypothesize that there is a mechanism of inter-limb coordination that may remain intact after a hemiplegic stroke that may be utilized to regain functionality of the impaired leg. Utilizing the function of the unimpaired leg to provide therapy to the impaired leg provides several advantages. One of the most significant advantages is the safety of the patient, since there is no direct manipulation of the paretic leg. Moreover, stimulating a mechanism that is still intact, may elicit greater functional outcome than in stimulating the impaired mechanism.

However, before using inter-leg coordination for therapy, its functional sensorimotor mechanisms need to be investigated. Various platforms and protocols have been used to investigate bilateral reflex mechanisms during different phases of the gait cycle [14]–[16], with the majority of the experimental protocols focusing on over-ground walking and dropping of the supportive surfaces at distinct gait phases [14], [15]. During posture maintenance, experiments including powerful unilateral displacement of one leg produced bilateral responses both in adults and in healthy human infants [17]–[19]. In addition, disturbances in the load feedback as well as the length of specific muscles during walking have been associated with evoked muscular activations of the unperturbed leg [16]–[20].

However, all of the previous studies have failed to separate the mechanisms of gait from those of body weight support and balance. Moreover, most experimental protocols do not consider balance support. As a result, mechanical perturbations and sudden load changes would have likely triggered mechanisms related to body balance and posture. In fact, the latter leads to the activation of inter-limb mechanisms and therefore explains bilateral leg responses. However, little is known whether this effect is exclusively caused by the mechanisms required for body stabilization and balance maintenance, or if it is also brought about from inter-limb coordination and mechanisms of gait. This leaves a gap in our understanding sensorimotor control of gait, and consequently from engineering effective rehabilitation protocols.

Moreover, the perturbations induced by the previous studies almost exclusively focus on dropping the walking surface, which causes a disruption in both force and kinematic feedback. When the walking surface is dropped, the ankle kinematics are perturbed in addition to the force feedback that is lost when the foot loses contact with the walking surface. These types of perturbations do not provide any separation of those two feedback mechanisms, and do not allow further in-depth investigation of the role of force and kinematic feedback in gait. In order to answer important questions on inter-leg coordination and sensorimotor control, it is desirable, therefore, to differentiate force and kinematic feedback. Adjustment of the surface stiffness is a unique way to achieve this differentiation, since stepping on a low stiffness platform does not disrupt force feedback, but affects

*Corresponding author
All authors are with the Department of Mechanical and Aerospace Engineering, Arizona State University, Tempe, AZ 85287, USA {jaskidmo, panagiotis.artemiadis}@asu.edu

kinematics.

In this paper, we focus on designing and applying unilateral perturbations that evoke contralateral leg motions, taking advantage of the inter-leg coordination mechanisms. We present results of repeatable and scalable evoked muscle activity of the contralateral tibialis anterior (TA) muscle through unilateral stiffness perturbations. Moreover, we discuss the latency of the evoked muscular responses that indicates supra-spinal circuits of inter-limb coordination. We then present a mathematical model that accurately describes the relationship between the magnitude of the stiffness perturbation and the evoked TA activity. The novel methods and results presented in this paper can lead to model-based rehabilitation strategies for impaired walkers, providing a novel approach to robotic interventions for gait therapy.

The rest of the paper is organized as follows: Section II describes the experimental setup and protocol used for this study. Section III presents the effect of unilateral stiffness perturbations on the muscle activation and kinematic response of the contralateral leg for healthy subjects. Section IV discusses the implications of the results and possible medical applications. Finally, section V concludes the paper with a brief summary of the contribution.

II. Methods

A. Experimental Setup

The investigation of the effect of unilateral stiffness perturbations on the muscle activation of the contralateral leg was performed using the Variable Stiffness Treadmill (VST) system [21], [22] shown in Fig. 1. The VST provides a unique platform for investigating inter-leg coordination mechanisms by combining a variety of components into one unique system. The major components of the VST include a variable stiffness mechanism, a split-belt treadmill, a custom-built body weight support and a motion capture system, which will be discussed individually below.

1) Variable Stiffness Mechanism: Stiffness perturbations were chosen for investigating inter-leg coordination because of the inherent ability to differentiate force and kinematic feedback. Adjustment of the surface stiffness provides a unique way to achieve this differentiation during walking because the force exerted by the walker's foot on the treadmill remains the same independent of the stiffness of that surface. Since this force remains the same, a change in stiffness will cause a displacement (i.e. kinematic perturbation).

In its most simplified form, the variable stiffness mechanism is a spring-loaded lever mounted on a translational track, as shown in Fig. 2. The effective stiffness of the treadmill, located at a distance x from the pivot joint, is dependent on the coefficient of stiffness S of the linear spring and the moment arm through which it exerts a force [23]. By design, S and r remain constant, therefore, the effective stiffness of the treadmill can be controlled by changing the distance x. In the VST system, the distance x is controlled by placing the VST mechanism assembly onto the carriage of a high-capacity linear track (Thomson Linear, 2RE16-150537) which is controlled by a high-precision drive (Kollmorgen, AKD-P00606-NAEC-0000). The resolution of achievable displacement of the linear track is $0.01\,mm$. The device can change the surface stiffness from essentially infinite (non-compliant walking surface) to 61.7 N/m (extremely low stiffness) in $0.13\,s$. It can also reach any stiffness in between at a maximum resolution of $0.038\,N/m$. Therefore, the VST can create quick, high resolution stiffness perturbations of nearly any magnitude. This leads to consistent, repeatable, and unanticipated stiffness perturbations that are useful for altering kinematic feedback. For a detailed characterization of the variable stiffness mechanism and the complete VST analysis see [22].

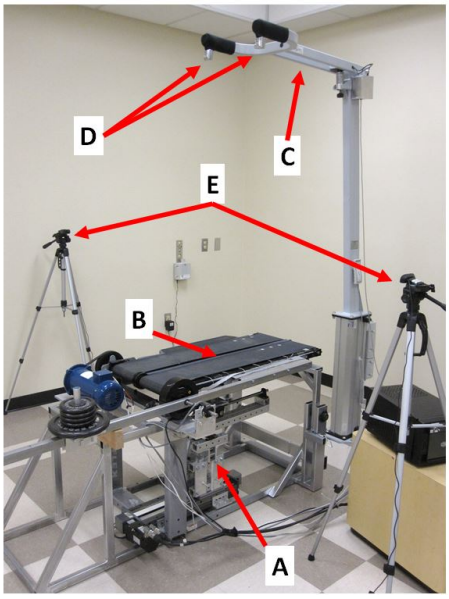

Fig. 1: The VST setup. Subsystems shown include: A) Variable stiffness mechanism, B) Split-belt treadmill, C) Custom-made harness-based body-weight support, D) BWS Loadcells, E) Motion capture system.

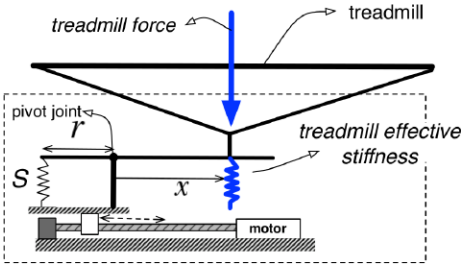

Fig. 2: Conceptual diagram of the variable stiffness mechanism.

2) Split-belt treadmill: The VST employs a split-belt treadmill configuration in order to allow each belt to deflect different amounts. The treadmill belts are supported above the floor on a frame of steel tubing that permits each belt to deflect independently. This will allow one leg to experience low stiffness perturbations while the other leg remains supported by a rigid surface. The split belt treadmill is shown in Fig. 1, part B.

3) Body weight support: Separate from the treadmill structure, there is a custom-built body weight support designed by LiteGait. By adjusting the height of the support system, full or partial body-weight support can be provided. This is an important capability to reduce activation of body stabilization and balance maintenance mechanisms. In addition, the support increases safety and extends the system's capabilities to stroke patients and other individuals with decreased mobility and stability. Two loadcells attached on the body-weight support harnesses measure the subject's weight supported by the mechanism from each side. The body weight support and loadcells are shown in Fig. 1, parts C and D, respectively.

4) Motion capture: Another important component of the VST is a low-cost and portable motion capture system comprised of infrared cameras (Code Laboratories Inc, model: DUO MINI LX) and infrared LEDs (Super Bright LEDs Inc, model: IR-1WS-850). The motion capture is important for tracking the location of the subject's foot in order to maintain the desired stiffness underneath the walker, and for precise timing of stiffness perturbations within the gait cycle. The motion capture system is also used for recording lower-limb joint angles throughout the gait cycle. The two cameras tracking the two legs are shown in Fig. 1, part E.

B. Experimental Protocol and Data Analysis

In order to understand how kinematic feedback of the foot affects inter-limb muscle coordination when body balance is not disturbed we investigated the effect of a stiffness perturbation to one leg on the muscle activity of the unperturbed leg while supplying approx. 30% body weight support.

Five healthy subjects [age 25 $\pm$ 5.4 years, weight 845 $\pm$ 156 Newtons] walked on the treadmill at a speed of $0.60\,m/s$ for over 200 gait cycles. The right leg walked on a rigid surface for the duration of the experiment. The surface under the left leg was commanded to maintain a stiffness of $1\,MN/m$, which is considered as rigid, for 30 gait cycles at the beginning of the experiment. Then, after a random number i of steps, where $i \in [3, 7]$, we immediately dropped the stiffness to 1 of 3 values: 10, 50 or $100\,kN/m$. The low stiffness perturbation began shortly after heel strike (approx. $130\,ms$) and lasted for the duration of the left leg stance phase after which the stiffness was commanded back to $1\,MN/m$ for the next i number of steps (see Fig. 3). An average of 17 $\pm$ 2.3 perturbations at each stiffness level were experienced by all subjects. Kinematic data for both legs were obtained at 140 Hz using the infrared camera system that tracked 12 (6 on each leg) infrared LEDs placed as pairs on the thigh, shank, and foot. Informed consent from the subjects was obtained at the time of the experiment, and the experimental protocol is approved by the Arizona State University Institutional Review Board (IRB ID#: STUDY00001001).

The muscle activity of the unperturbed leg was obtained using surface electromyography (EMG) via a wireless surface EMG system (Delsys, Trigno Wireless EMG) and recorded at 2000 Hz. Electrodes were placed on the tibialis anterior (TA) and soleous (SOL) of the right leg. After computing the EMG linear envelope, the data were normalized to the maximum value of that EMG signal. The EMG data corresponding to the gait cycles of walking on the rigid surface and the cycles pertaining to the three perturbations were found and normalized temporally to percent gait cycle in order to eliminate discrepancies due to natural variations in gait patterns (i.e. stride length, cycle time, etc). All cycles of walking on the surface at infinite stiffness (except for two cycles following a perturbation to eliminate any residual effects from the perturbation) are included in the unperturbed (i.e. "rigid") data set. This results in normalized EMG signals as a function of percent gait cycle, where 0% corresponds to the heel strike of the left leg.

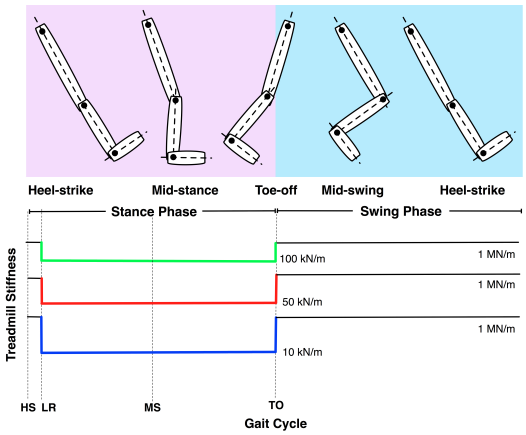

Fig. 3: Timing of stiffness perturbations within the gait cycle.

III. Results

A. Bilateral Response

The contralateral response to unilateral low stiffness perturbations for a representative subject is shown in Fig. 4. The normalized EMG amplitude for the TA and SOL, along with ankle dorsi-plantar flexion for all gait cycles pertaining to each surface stiffness is shown for the unperturbed leg. The data is plotted as a function of the gait cycle percentage, where heel-strike and toe-off of the right leg are indicated on the figure as HS and TO, respectively.

The experimental data of all subjects reveal two notable trends of 1) systematic increase in contralateral muscle activity and 2) delayed response that was consistent across subjects. The results demonstrating both of these trends will be presented below.

1) Evoked contralateral muscle activity: As can been seen in Fig. 4, low-stiffness perturbations to the left leg evoked muscle activity in the right leg that increased systematically with decreased stiffness. This is seen in the TA most prominently in mid to terminal swing phase, and during the majority of the stance phase in the SOL. As the magnitude of the perturbation increases (i.e. lower stiffness values), there is a proportional increase in TA and SOL activity. The kinematic response, which results from muscle contraction, confirms the systematic increase of muscle activity. There

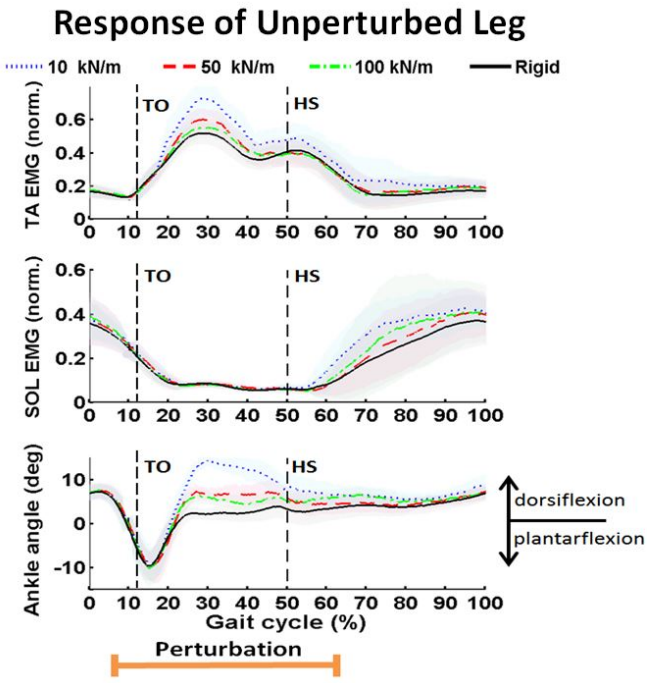

Fig. 4: Averaged muscle activity and ankle kinematics of the unperturbed leg for a representative subject. Plotted from top to bottom is the normalized TA EMG, normalized SOL EMG, and ankle dorsi (+) - plantar (-) flexion for gait cycles at each of four surface stiffness levels. Mean (darker lines) and standard deviation (lightly shaded areas) values are shown along with an indication of the timing of the perturbation. Toe- off and heel-strike for the right leg is indicated by TO and HS, respectively.

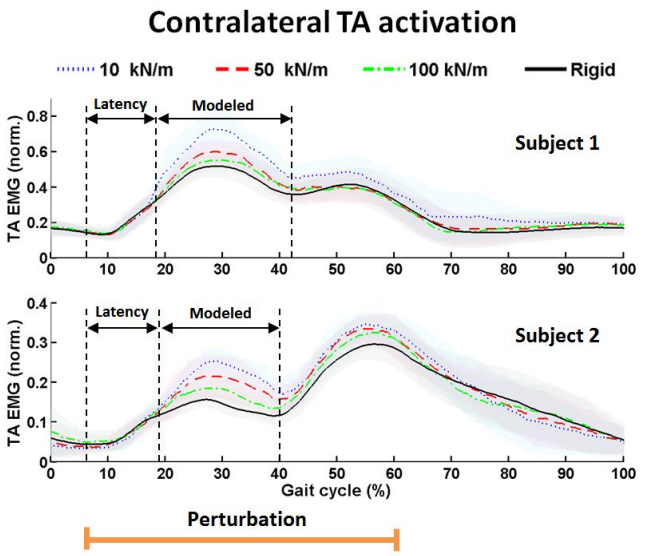

Fig. 5: Averaged TA muscle activity for gait cycles at each of the four surface stiffness levels for two subjects. Mean (darker lines) and standard deviation (lightly shaded areas) values are shown along with an indication of the timing of the perturbation. The latency of response and the section of data that were modeled are also indicated.

is a systematic increase in ankle dorsiflexion beginning at approx. 20% and lasting for the duration of the gait cycle.

2) Consistent latency: Also of importance is a consistent latency across subjects from the onset of the perturbation to when increased TA activity, and change of ankle angle, is seen. The normalized TA EMG as a function of percent gait cycle (mean and standard deviation), with an indication of the delayed response, for two subjects is shown in Fig. 5.

As seen in the figure, the amplitude of muscle activity of all profiles is very similar from the beginning until approx. 18% of the gait cycle where they begin to diverge. The timing of the divergence was consistent across all subjects: mean 18.28%, standard deviation (SD) 0.72% gait cycle. The low stiffness perturbation began approximately $130\,ms$ after heel strike of the left leg which corresponds to 8.0% [SD 0.8%] of the gait cycle. The latency was calculated from the beginning of the perturbation until the mean of the $10\,N/m$ stiffness profile was greater than 1 standard deviation of the EMG activity for walking on a rigid surface. The latency averaged across all subjects resulted in a mean of $171\,ms$ [SD $31ms$].

B. Mathematical Model

While many studies have investigated the kinematic and neuromuscular effect of sensory perturbations on human gait, the authors are not aware of the existence of any model that relates the sensory input (i.e. perturbation) to the noticed effect. Such a model would be desirable for predicting the effects of sensory stimuli. Here, we created a single-input single-output linear model that relates the magnitude of the stiffness perturbation to the evoked TA activity.

The output (i.e. evoked TA activity) was found by subtracting the mean EMG activity of all unperturbed cycles from each of the EMG cycles for each stiffness level. The data were then reduced to only focus on the section from the end of the delay (approx. 18% of the gait cycle) to the local minimum between the two local maximums of TA activity (approx. 41% of the gait cycle), as indicated in Fig. 5. This was done because we are most interested in the TA increase during swing phase, the section of the gait cycle where a decreased dorsiflexion motion, termed drop-foot [24], is usually present in impaired gait. The data for all gait cycles were then low-pass filtered because we are interested in modeling the general trend of evoked muscle activity, as opposed to matching a high frequency EMG signal. The input (i.e. the magnitude of the stiffness perturbation) was a constant step input with its magnitude proportional to the maximum evoked muscle activity.

System identification techniques were used to relate the measured EMG activity (model output) to the stiffness perturbation magnitude (input). A variety of model structures and orders were investigated as options to create a black box model. Eighty percent (80%) of all data-cycles were randomly selected for fitting the models. The resulted models were then tested against the remaining 20% of the data as validation. Expectation maximization (EM) algorithms

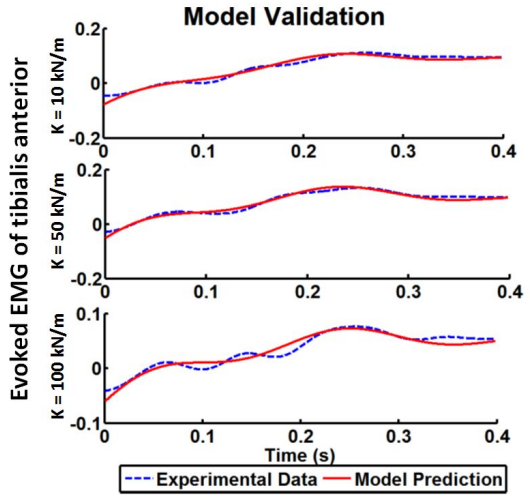

Fig. 6: Predicted evoked TA muscle activity vs experimental data for three perturbed stiffness values (shown from top to bottom) of 10, 50, and 100 kN/m, respectively.

were used for fitting the model, implemented in the System Identification Toolbox in MATLAB. Based on the normalized root mean square error between the model prediction and validation data, and the complexity of the model, a second order linear model was selected. The comparison of validation data and model prediction is shown in Fig. 6 for a representative subject for each level of stiffness perturbation.

As can be seen, the model accurately matches the trend of the validation data for each level of perturbed stiffness. This successfully achieves our goal of creating a mathematical relationship between the input of a stiffness level and the evoked TA activity. The model, expressed in transfer function form, for a representative subject is shown below:

$$\frac{Y(z)}{U(z)} = \frac{0.006483 - 0.006483z^{-1}}{1 - 1.999z^{-1} + 0.9989z^{-2}} \quad (1)$$

where $Y(z)$ is evoked TA activity and $U(z)$ is the perturbed stiffness magnitude. The model was consistent across subjects as indicated by the poles and zeros of the transfer function for all subjects that participated.

IV. Discussion

A. Muscle Activation

The results of this paper confirm our hypothesis that unilateral stiffness perturbations can evoke contralateral muscle activity when balance mechanisms are not involved. Therefore, this suggests the existence of an inter-leg coordination mechanism that is separate from the mechanisms related to balance and posture.

Moreover, increased TA activity in the unperturbed leg is seen predominately in swing phase. This is an exciting result from a functional point of view since this can provide significant solutions to the problem of drop-foot that most impaired walkers suffer from, and it is the leading cause of after-stroke falls [24]. The tibialis anterior evoked activation can play a significant role in avoiding drop-foot in swing phase because it is the primary muscle creating dorsiflexion (toe-up motion). The fact that the induced perturbations can evoke the necessary ankle dorsiflexion to counteract drop-foot indicates that surface stiffness may play a significant role in robotic gait rehabilitation.

Moreover, the repeatability (consistency across subjects) and scalability (systematic increase in EMG activity with decreasing stiffness) suggest that walking surface stiffness is a significant stimulus in gait. As mentioned previously, stiffness control provides a unique way to differentiate force and kinematic feedback because the force exerted by the walker's foot remains the same so a change in stiffness will cause only a kinematic perturbation. While several studies have investigated the effect of length and load feedback on gait, the surface stiffness that is utilized in this study, is a unique way to ensure isolation of one sensory modality.

In addition to the above, the evoked responses were significantly delayed from the onset of the perturbation (delay $> 150\,ms$), which supports the idea of supraspinal pathways regulating inter-leg coordination. A delay of this duration corresponds to a transcortical reflex mechanism [25]. Therefore, our results suggest that supraspinal circuitry is involved in the response to sudden low stiffness perturbations.

From a clinical prospective, the results of this study can be disruptive since they suggest that muscular activity in the contralateral TA can be evoked via the cerebral cortex by altering the surface stiffness below the unilateral leg. Stroke results from lesions in the brain and can cause functional impairment in a variety of motor tasks, including gait. A main deficiency in stroke survivors is insufficient TA activity in the swing phase which results in a decreased dorsiflexion. The results presented above suggest that by manipulating the non-paretic leg in stroke patients, the muscle activation in the TA of the paretic leg can be evoked through supra-spinal inter-leg coordination mechanisms. While other studies have stimulated the impaired TA via functional electric stimulation to improve functional outcome [26], [27], this technique bypasses the brain which is the location of the root cause of the gait impairment created by stoke. On the other hand, our results suggest that by exploiting existing supra-spinal neural circuits, desired TA activity can be evoked by regulating the stiffness of the walking surface. We have shown in this paper that the evoked TA activity can decrease drop-foot and perhaps facilitate neural plasticity in the brain for eventual recovery of normal gait.

B. Mathematical Model

The creation of a mathematical model relating sensory input to muscle activation is the foundation for creating model-based rehabilitation protocols. The second order linear model presented in this paper is used to relate the magnitude of the stiffness perturbation to the evoked TA activity in the unperturbed leg during swing phase. The model is able to match the trend of evoked muscle activity and could be used for prediction purposes. In other words, if a certain TA activation is desired, the model can be utilized to indicate

the level of stiffness perturbation to be used to evoke the desired muscle activity.

In addition, the model could be used as a metric of normal gait patterns because it was proven to be consistent across healthy subjects. This provides a quantifiable level of impairment which can be utilized in rehabilitation and as a means for assessing improvement.

V. Conclusions

This paper presents preliminary but solid results of evoking leg muscle activation using contralateral stiffness perturbations. The presented study provides the first evidence that desired muscle activation can be achieved through mechanisms of inter-leg coordination by manipulating the sensory input to the opposite leg. More, specifically, our results include repeatable and scalable evoked muscle activity of the contralateral TA through unilateral stiffness perturbations. The most dramatic increase of muscle activity is observed during mid swing phase. In addition, a latency of $171\,ms$ from the beginning of the perturbation to increased muscle activity was observed for all subjects which corresponds to neural transmission times of transcortical neural circuitry. Therefore, our results suggest the existence of supra-spinal mechanisms of inter-leg coordination that are centrally controlled. In addition, this paper presents a second order linear model that accurately describes the relationship between the magnitude of the stiffness perturbation and the evoked muscular change of the TA.

The results presented in this paper set the foundation for a paradigm shift of robotic interventions for gait rehabilitation. We suggest that, in the case of healthy human subjects, manipulating sensory input of one leg can create a desired muscle activation in the other leg through mechanisms of inter-leg coordination. Future work will include testing of this hypothesis with hemi-plegic stroke patients. In addition, the work presented in this paper lays the foundation for model-based rehabilitation protocols.

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