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# Unilateral Floor Stiffness Perturbations Systematically Evoke Contralateral Leg Muscle Responses: a New Approach to Robot-assisted Gait Therapy

Jeffrey Skidmore and Panagiotis Artemiadis*

***Abstract*—A variety of robotic rehabilitation devices have been proposed for gait rehabilitation after stoke, but have only produced moderate results when compared to conventional physiotherapy. We suggest a novel approach to robotic interventions which takes advantage of mechanisms of inter-limb coordination. In order to test the viability of this approach, we apply unilateral floor stiffness perturbations via a unique robotic device, and observe evoked contralateral leg responses in kinematics as well as muscle activations in healthy subjects. The real-time control of floor stiffness is utilized to uniquely differentiate force and kinematic feedback, creating novel sensory perturbations. We present results of repeatable and scalable evoked kinematic and muscular response of the unperturbed leg in healthy subjects. Moreover, we provide insight into the fundamental sensorimotor mechanisms of inter-leg coordination. We also lay the foundation for model-based rehabilitation strategies for impaired walkers by presenting a mathematical model that accurately describes the relationship between the magnitude of the stiffness perturbation and the evoked muscle activity. One of the most significant advantages of this approach over current practices is the safety of the patient, since this does not require any direct manipulation of the impaired leg. The novel methods and results presented in this paper set the foundation for a paradigm shift of robotic interventions for gait rehabilitation.**

***Index Terms*—Gait Rehabilitation, Inter-leg coordination, treadmill therapy**

## I. Introduction

Stroke is a common health problem throughout the world with 795,000 new strokes occurring each year in the United States alone [1], [2]. Nearly 90% of stroke survivors require therapy but the majority of patients only achieve poor functional outcome five years after the onset of stroke [2], [3]. Robot-assisted therapy has been proposed as an alternative approach to conventional physiotherapy because robots can easily facilitate the key behavioral signals that drive neural plasticity which is the basic mechanism underlying improvement in functional outcome after stroke [4].

A variety of robotic rehabilitation devices have been developed in the last several years for gait therapy. These include the Lokomat [5], the Gait Trainer [6], and others (e.g. [7]–[10]). However, there have been conflicting results from recent studies about the effectiveness of these devices. Some studies report that when compared to conventional therapy robotic rehabilitation achieves greater functional outcome (e.g. [11], [12]), while others indicate less improvement (e.g. [13], [14]). Therefore there is no clear evidence that robotic gait training is superior to conventional physiotherapy for either chronic or subacute stoke patients at the present time [15].

We propose an alternative approach to robotic interventions in gait therapy which takes advantage of mechanisms of inter-leg coordination. Considering the cyclic coordination between limbs in human walking, we hypothesize that there is a mechanism of inter-limb coordination that may be utilized to regain functionality of the impaired leg, if this mechanism remains intact after a hemiplegic stroke. This unique idea of utilizing the function of the unimpaired leg to provide therapy to the impaired leg provides several advantages over current rehabilitation protocols. One of the most significant advantages is the safety of the patient, since there is no direct manipulation of the paretic leg. Current robotic rehabilitation devices physically interact and manipulate the paretic leg. Moreover, stimulating a mechanism that is still fully functional, such as we suggest in this paper, may elicit greater functional outcome than in stimulating the impaired mechanism.

However, the sensorimotor control mechanisms of inter-leg coordination are currently not well understood. Various platforms and protocols have been used to investigate bilateral reflex mechanisms during different phases of the gait cycle [16]–[18], with the majority of the experimental protocols focusing on over-ground walking and dropping of the supportive surfaces at distinct gait phases [16], [17]. During posture maintenance, experiments including powerful unilateral displacement of one leg produced bilateral responses both in adults and in healthy human infants [19]–[21]. 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 [18]–[23]. For example, an increase in ipsilateral belt speed with a constant contralateral belt speed in differential split-belt treadmill walking has shown increased ipsilateral gastrocnemius and contralateral tibialis anterior EMG activity [23]. Also, unloading of the plantarflexor muscles by dropping the walking surface during stance phase significantly decreases soleus muscle activity [22].

One significant limitation of the previous studies is that

Jeffrey Skidmore and Panagiotis Artemiadis are with the School for Engineering of Matter, Transport and Energy, Arizona State University, Tempe, AZ 85287 USA.
Email: {jaskidmo, panagiotis.artemiadis}@asu.edu
*Corresponding author

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the perturbations induced by the previous experiments 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 (load force remains the same in steady state), but affects kinematics.

Another limitation of previous works is that 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 lack of knowledge leaves a significant gap in our understanding of sensorimotor control of gait, and consequently from engineering effective rehabilitation protocols.

In this paper, we investigate inter-limb coordination mechanisms by designing and applying unilateral low stiffness perturbations that evoke contralateral leg responses. This uniquely allows for the de-coupling of kinematic and force perturbations to the ipsilateral leg. Moreover, body weight support was given to suppress mechanisms related to balance and posture. We present results of repeatable and scalable evoked muscle activity and kinematic response of the contralateral leg of healthy subjects through unilateral stiffness perturbations. We discuss the potential clinical significance of the evoked activity of the contralateral tibialis anterior muscle during the swing phase of walking. Moreover, we discuss the latency of the evoked 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 activity of the tibialis anterior muscle. 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.

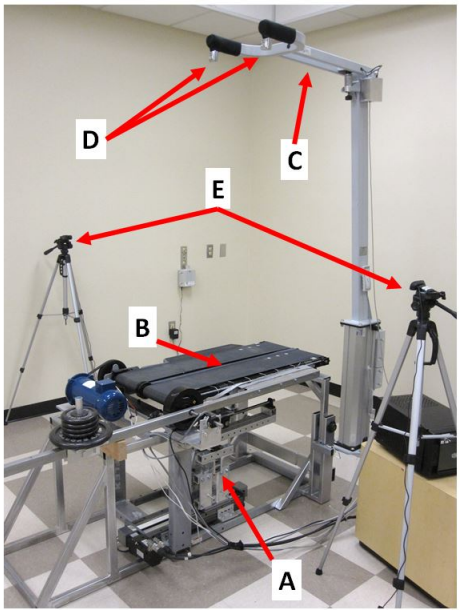


Fig. 1: The VST setup. Subsystems shown include: A) Variable stiffness mechanism, B) Split-belt treadmill, C) Custom-made harness-based body-weight support, D) BWS Loadcells, E) Motion capture system.

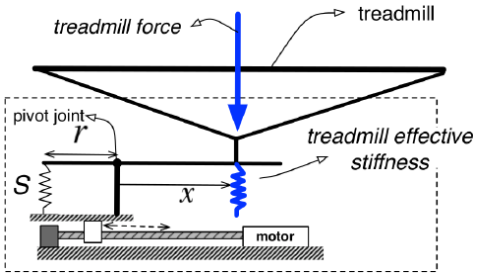


Fig. 2: Conceptual diagram of the variable stiffness mechanism.

## II. Methods

### A. Experimental Setup

The investigation of the effect of unilateral stiffness perturbations on the contralateral leg was performed using the Variable Stiffness Treadmill (VST) system [24], [25] shown in Fig. 1. The VST provides a unique platform for proper investigation of 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. Each component will be discussed 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

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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 [26]. 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, Part Number: 2RE16-150537) which is controlled by a high-precision drive (Kollmorgen, Part Number: 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 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 [25].

*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 at $70\,cm$ above the floor on a frame of steel tubing that permits each belt to independently deflect downward to a maximum of $30^\circ$ from the horizontal position. 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, we can choose to have full or partial body-weight support. 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 are measuring 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.

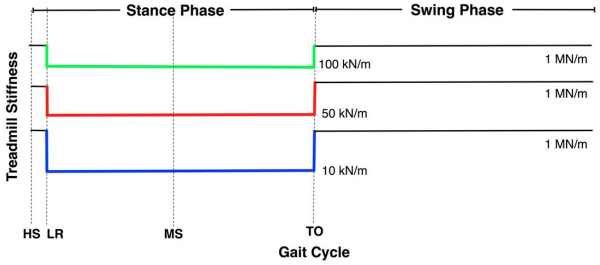


Fig. 3: Timing and magnitude of unilateral stiffness perturbations where HS, LR, MS and TO represent heel strike, loading response, mid-stance and toe-off, respectively.

### *B. Experimental Protocol and Data Analysis*

In order to investigate inter-leg coordination when body balance is not disturbed, we investigated the response of the contralateral (unperturbed) leg to unilateral stiffness perturbations while supplying the subject with approx. 30% body weight support (BWS). A value of 30% BWS was chosen because this level of support has been given in other studies [27], [28].

Five healthy subjects [age 25 $\pm$ 5.4 years, weight 190 $\pm$ 35 lbs] walked on the treadmill at a speed of $0.60\,m/s$ for at least 200 gait cycles. The right treadmill belt was not allowed to deflect for the duration of the experiment thus preventing any direct perturbation of the right leg. The surface underneath the left leg was commanded to maintain a stiffness of $1\,MN/m$, which is considered to be rigid, for 30 gait cycles at the beginning of the experiment. Then, after a random number $n$ of steps, where $n \in [3, 7]$, 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 $n$ number of steps. A graphical representation of the timing and magnitude of the stiffness perturbations is shown in 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 subject 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 soleus (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). The first 30 gait cycles and the

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cycles in between perturbations 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.

## III. Results

### A. Bilateral Response

The kinematic and muscular 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 the hip flexion-extension, knee flexion-extension and dorsi-plantar flexion (mean and standard deviation) for all gait cycles pertaining to each surface stiffness is shown for both the perturbed (left) and unperturbed (right) legs. The data are plotted as a function of the gait cycle percentage, where heel-strike and toe-off of each leg are indicated on the figure as HS and TO, respectively. Colored bars underneath an asterisk are included to indicate when statistically significant changes are observed. Both the muscular and kinematic profiles of walking on a rigid surface resemble that of what would be expected for normal human gait [29], therefore our system and experimental protocol did not alter normal gait patterns.

Although the left leg was directly perturbed through the left treadmill stiffness change, the focus of this work is to understand inter-leg coordination by investigating the response of the unperturbed leg to the stiffness perturbations. Therefore, the analyses for the rest of the paper will be focused on the effects of the perturbation on the contralateral leg response. Moreover, our previous work has shown that the ipsilateral leg kinematics are significantly affected by the stiffness perturbation ([24], [25], [30], [31]), thus a comparison between the perturbed and unperturbed gait cycle is redundant and beyond the scope of the present study. In addition, the kinematic and muscular responses recorded during this study are consistent with what would be expected from the previous studies ([24], [25], [30], [31]).

The experimental data of the response of the unperturbed leg for all subjects reveal three important trends. These are: 1) a systematic change in joint angles, 2) a systematic increase in muscle activity and 3) a delayed response that is consistent across subjects. The results demonstrating each of these trends will be presented below.

*1) Altered contralateral kinematics:* As can been seen in Fig. 4 (bottom right-hand side), low-stiffness perturbations to the left leg evoked systematic changes in the kinematics of the right leg. The change in joint angles is proportional to the magnitude of the stiffness perturbation. These changes are seen beginning at approx. 20% and then converge back to the normal walking pattern later in the gait cycle. The change in kinematics was statistically significant at the 95% confidence level for a two sample unpaired t-test when comparing the data for each perturbation level to the rigid (i.e. normal) condition. Colored bars indicating when significant changes are observed are included in Fig. 4. As can be seen, the significance of the response is dependent on the magnitude of the stiffness

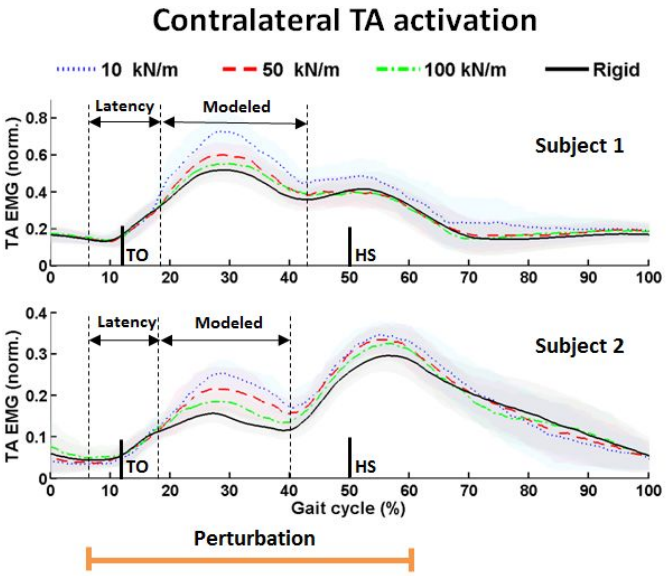


Fig. 5: Averaged TA muscle activity for gait cycles at each of the four surface stiffness levels for two subjects. Mean (darker lines) and standard deviation (lightly shaded areas) values are shown along with an indication of the timing of the perturbation. The latency of response and the section of data that were modeled are also indicated. The timing of toe-off and heel strike of the right leg within the gait cycle are represented by TO and HS, respectively.

perturbation. While only data for a representative subject is shown in Fig. 4, the statistical significance was consistent across subjects.

*2) Evoked contralateral muscle activity:* As can been seen in Fig. 4 (top right-hand side), 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 between toe-off and heel-strike (i.e. swing phase), and after heel-strike (i.e. 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. Similar to the kinematic response, the change in muscle activation was statistically significant at the 95% confidence level. Colored bars indicating when significant changes are observed in muscle activity are included in Fig. 4.

*3) Consistent latency:* Also of importance is a consistent latency across subjects from the onset of the perturbation to when increased TA activity, and altered kinematics, is seen. The normalized TA EMG (mean and standard deviation) as a function of percent gait cycle, 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. 17% of the gait cycle where they begin to diverge. The timing of the divergence was consistent across all subjects: mean 17.03%, standard deviation (SD) 0.63% 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

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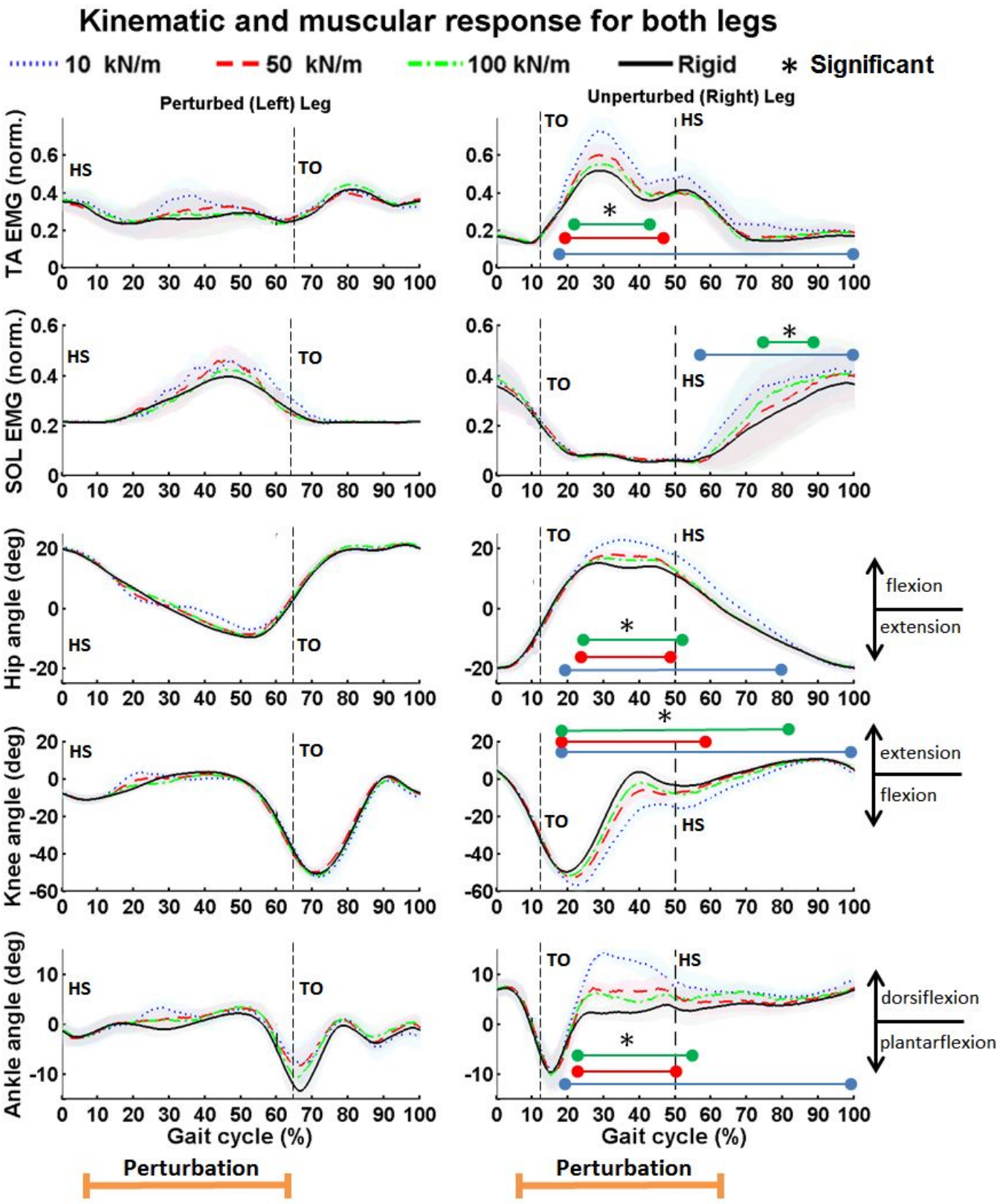


Fig. 4: Averaged muscle activity and joint kinematics of the perturbed (left) and unpertubed (right) legs for a representative subject. Plotted from top to bottom is the normalized TA EMG, normalized SOL EMG, hip flexion (+) - extension (-), knee flexion (-) - extension (+) and ankle dorsi (+) - plantar (-) flexion for gait cycles at each of four surface stiffness levels. Mean (darker lines) and standard deviations (lightly shaded areas) values are shown along with an indication of the timing of the perturbation. Statistically significant changes are indicated by colored bars (corresponding to each stiffness level) that are placed beneath a black asterisk. Heel-strike and toe-off for each leg are indicated by HS and TO, respectively. The duration of the gait cycle is approximately $1.8\,s$.

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cycle. The latency was calculated from the beginning of the perturbation until the EMG activity for a $10\,N/m$ stiffness profile is significantly greater than the EMG activity for a rigid surface. The significance is calculated at the 95% confidence level using a two sample unpaired t-test. The latency averaged across all subjects resulted in a mean of $150\,ms$ [SD $27\,ms$].

### *B. Mathematical Model*

While many studies have investigated and described the kinematic and neuromuscular effect of sensory perturbations on human gait, the authors are not aware of the existence of any model, or mathematical equation that relates the sensory input (i.e. perturbation) to the altered kinematic or muscular response. Such a model would be desirable for predicting the effects of sensory stimuli. For example, if a certain TA activation is desirable in gait therapy, the model can be utilized to indicate the level of stiffness perturbation necessary to evoke the desired muscle activity. In order to achieve this desired predictive capability 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 section of data was chosen 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 [32], 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 used (i.e. the magnitude of the stiffness perturbation) was a constant step input with its magnitude proportional to the treadmill compliance. Compliance, instead of stiffness, was chosen because increased muscle activity is observed with decreased stiffness (increased compliance).

System identification techniques were used to relate the measured EMG activity (model output) to the stiffness perturbation magnitude (input). A variety of model orders and types (both linear and nonlinear) were investigated as options to create a black box model. Eighty percent (80%) of all data-cycles corresponding to the combined 10 and 100 $kN/m$ data sets were randomly selected for fitting the models. The resulted models were then tested against the remaining 20% of the data from that combined set, and all of the 50 $kN/m$ data sets as validation. This range of stiffness for training the models was selected because the authors do not expect the clinical implementation of any stiffness level below or above 10 or 100 $kN/m$, respectively. The model was validated with a stiffness level of 50 $kN/m$ to verify the generalization of the model. Expectation maximization (EM) algorithms 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 prediction of the model was compared to the validation data and is shown in Fig. 6 for all subjects for each level of stiffness perturbation. The data corresponding to the median correlation coefficient of each data set is shown along with the correlation coefficient (mean and standard deviation) for each data set.

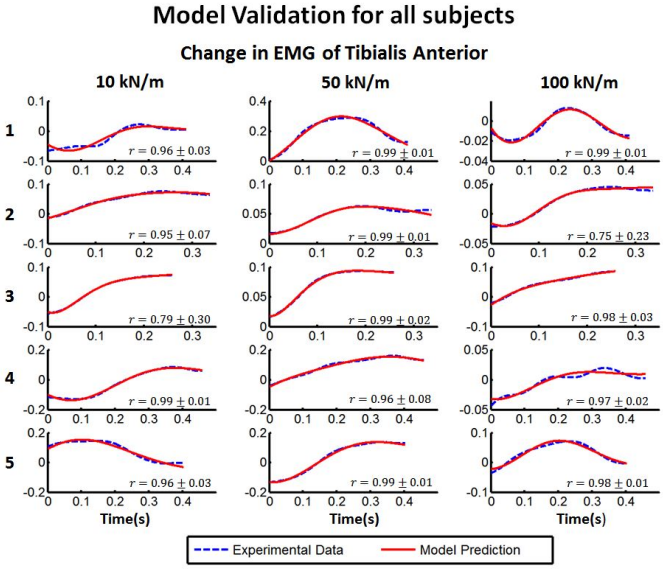


Fig. 6: Predicted evoked TA muscle activity vs experimental data for all subjects (rows from top to bottom) for each of the three perturbed stiffness values (columns from left to right) of 10, 50, and 100 $kN/m$, respectively. The data corresponding to the median correlation coefficient of each data set is plotted. The correlation coefficient (mean and standard deviation) for each data set is also shown.

As can be seen, the model accurately matches the trend of the validation data for each level of perturbed stiffness. The correlation coefficient between the model prediction and experimental data averaged across all subjects was $0.93\pm0.08$, $0.98\pm0.01$, and $0.93\pm0.10$ for the perturbation stiffness levels of low, mid, and high, respectively. This successfully achieves our goal of creating a mathematical relationship between the input of the compliance of the walking surface and the evoked TA activity. The model, expressed in transfer function form, for a representative subject is shown below:

$$\frac{Y(z)}{U(z)} = \frac{-0.1061+0.1061z^{-1}}{1-1.9970z^{-1}+0.9973z^{-2}} \tag{1}$$

where $Y(z)$ is evoked TA activity (model output) and $U(z)$ is proportional to the treadmill compliance (model input). The model was consistent across subjects as shown by the poles and zeros of the transfer function for all subjects that participated in the study, as listed in Table I.

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TABLE I: Poles and Zeros of Fitted Models

| Subject | Poles | Zero |
|---|---|---|
| 1 | $0.9996\angle\pm0.15°$ | 1.0001 |
| 2 | $0.9987\angle\pm0.25°$ | 1.0000 |
| 3 | $0.9979\angle\pm0.18°$ | 1.0000 |
| 4 | $0.9994\angle\pm0.13°$ | 1.0000 |
| 5 | $0.9995\angle\pm0.15°$ | 1.0000 |

## IV. Discussion

### A. Contralateral Response

The results of this paper confirm our hypothesis that unilateral stiffness (i.e. kinematic) perturbations can evoke a response of the contralateral leg when balance mechanisms are not involved. This suggests the existence of a mechanism of inter-leg coordination that is separate from the mechanisms related to balance and posture.

Moreover, the repeatability (consistency across subjects) and scalability (systematic increase in EMG activity and kinematic response 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 on the treadmill remains the same so a change in stiffness will cause a displacement of the foot, without altering load feedback. While there are transient dynamics in the stiffness control, previous work has shown that the system has fast dynamics and is highly damped [25]. 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.

The novel application of stiffness perturbations in this paper resulted in a systematic increase in muscle activity of both the TA and SOL muscles. Moreover, increased TA activity in the unperturbed leg is seen most profoundly 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 is the leading cause of after-stroke falls [32]. 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 (as observed in the kinematic response seen in Fig. 4) to counteract drop-foot indicates that surface stiffness may play a significant role in robotic gait rehabilitation.

In addition to all of the above, the evoked responses were significantly delayed from the onset of the perturbation (delay approx. $150\,ms$), which supports the idea of supraspinal pathways regulating inter-leg coordination. A delay of this duration corresponds to a transcortical reflex mechanism [33]. Therefore, our results suggest that supraspinal circuitry is involved in the response to sudden low stiffness perturbations.

Moreover, the timing of evoked muscle activity with the gait cycle indicates that the brain may be involved in adjusting the gain of system. As can be seen in Fig. 7, the systematic increase of muscle activity only occurs when the muscle is normally active. More specifically, even though the perturbation occurs from 8% to 60% of the gait cycle, evoked muscle activity of the SOL is not seen until the end of the perturbation when the stance phase begins which is when the SOL is active in normal walking. This pattern of activation is also seen in the TA during the swing phase and beginning of the stance phase. This suggests that the brain modifies the amplitude of neuromuscular response to sensory stimuli but does not initiate activation of the muscles in gait. This is consistent with the theory that central pattern generators located in the spinal cord are responsible for generating basic gait motor patterns through cyclical flexion and extension of the joints [34], while the brain modulates the basic gait patterns with descending inputs [35].

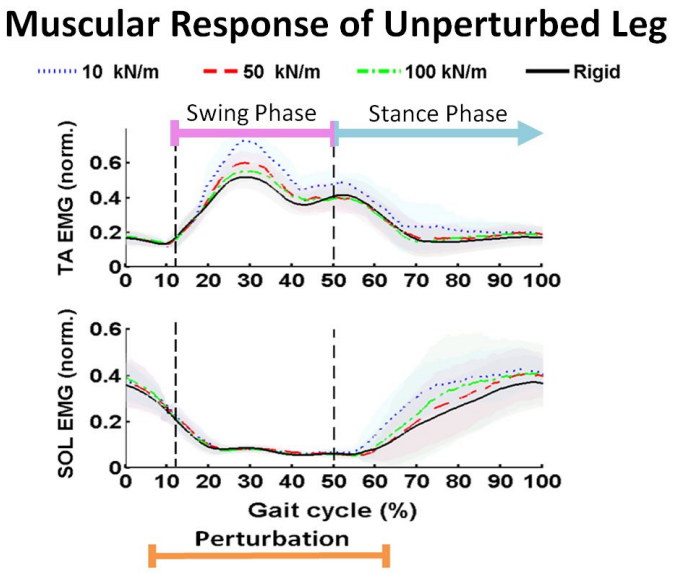


Fig. 7: Averaged TA and SOL muscle activity for gait cycles at each of the four surface stiffness levels for a representative subject. Mean (darker lines) and standard deviation (lightly shaded areas) values are shown. An indication of the timing of the perturbation and the swing and stance phases are also shown.

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 ipsilateral 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 for healthy subjects provide foundational indications suggesting the feasibility of a novel approach that by manipulating the non-paretic leg in stroke patients, the muscle activation in the TA of the paretic leg may 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 [36], [37], this technique by-passes the brain which is the location of the root cause of the gait impairment created

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by stroke. On the other hand, our results suggest the feasibility of an alternative approach to create desired TA activity by exploiting existing supra-spinal neural circuits via regulation of the stiffness of the walking surface. While the results presented in this paper are with healthy subjects, we provide indications that the evoked TA activity can decrease drop-foot and perhaps facilitate neural plasticity in the brain for eventual recovery of normal gait in stroke patients.

### 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 desirable in gait therapy, the model can be utilized to indicate the level of stiffness perturbation necessary 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 results of evoking kinematic and muscular changes in the unperturbed leg of healthy subjects using stiffness perturbations to the opposite leg. The presented study provides the first evidence of unilateral stiffness perturbations having a repeatable and controllable effect on the contralateral leg. This is shown by systematic changes in joint angles and velocities, as well as the activation of the TA and SOL muscles. In addition, a latency of $150\,ms$ from the beginning of the perturbation to the evoked response 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 mathematical model that accurately describes the relationship between the magnitude of a sensory perturbation and the evoked muscular change. The second order linear model presented in this paper can accurately predict the expected evoked TA activity based on a commanded surface stiffness value and lays the foundation for model-based rehabilitation protocols.

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.

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