A comprehensive analysis of sensorimotor mechanisms of inter-leg coordination in gait using the Variable Stiffness Treadmill: Physiological insights for improved robot-assisted gait therapy

Jeffrey Skidmore and Panagiotis Artemiadis*

Abstract— **Rehabilitation robotics is an emerging field in which gait training has been largely automated allowing more intensive, repetitive motions which are important for facilitating recovery. However, there is no clear evidence that robot-assisted gait training is superior to conventional therapy. A limitation of current approaches to gait therapy is that they do not consider mechanisms of inter-leg coordination and how the sensory feedback from one leg affects the motion of the other leg. Instead they impose motion on the impaired limb. Recent research suggests that utilizing the coupling between limbs in stroke rehabilitation therapies could lead to improved functional outcome. Therefore, a fundamental understanding of underlying sensorimotor mechanisms of inter-leg coordination may facilitate improved interventions in gait therapy. This paper systematically explores and analyzes a sensorimotor mechanism of inter-leg coordination that is stimulated through sudden unilateral low-stiffness perturbations to the walking surface. The potential contribution of each sensory modality to the perception and response of the perturbation will be investigated. Additionally, the neural pathway that relays the sensory signal into the motor output will be described in order to fully characterize this sensorimotor mechanism of inter-leg coordination. This work provides physiological understanding of inter-leg coordination that will benefit robot-assisted gait therapies.**

I. Introduction

Rehabilitation robotics is an emerging field in which gait training has largely been automated [1], [2]. A benefit of robot-assisted gait therapy is that robots can perform many repetitions with high accuracy, thus replacing the physical effort required of a therapist and allowing more intensive, repetitive motions which are important for facilitating recovery [3]. A variety of robotic rehabilitation devices have been developed in the last several years for gait therapy (e.g. [4]–[7]). However, there is no clear evidence that robot-assisted gait training is superior to conventional therapy [2], [8], [9]. With an aging population and expected shortages of health care personnel, there is a need for better solutions for providing gait therapy [10].

A limitation of the robotic devices for gait therapy is that they do not consider mechanisms of inter-leg coordination and how the sensory feedback from one leg affects the motion of the other leg [11]. Rather, the state-of-the-art devices impose motion on the impaired limb. A recent review suggests that utilizing inter-limb coupling in stroke rehabilitation therapies will lead to improved functional outcome [12]. Therefore, a fundamental understanding of underlying sensorimotor mechanisms of inter-leg coordination may facilitate improved robotic interventions in gait therapy [11].

Our previous work of investigating mechanisms of inter-leg coordination with healthy subjects has shown a systematic and scalable contralateral response to unilateral stiffness perturbations [13] that is dependent on the phase of the gait cycle [11]. This response has been repeatedly observed across healthy subjects [11], [13], [14] and was recently observed in hemi-paretic patients [15]. Additionally, electroencephalography (EEG) experiments with healthy subjects have shown that these responses may pass through the sensorimotor cortex [14]. However, the underlying mechanism of this repeatably observed contralateral response is not well understood.

Therefore, the object of the present research is to analyze in a comprehensive way the sensorimotor mechanisms of inter-leg coordination underlying the observed contralateral response. We hypothesize that proprioceptive and vestibular sensory feedback are primarily responsible for initiating the observed sensorimotor mechanism. Our hypothesis is tested by systematically exploring and analyzing the potential contribution of each sensory modality to the perception and response of the perturbation. Additionally, the neural pathway that relays the sensory signal into the motor output will be described in order to fully characterize this sensorimotor mechanism of inter-leg coordination.

II. Methods

A. Experimental Setup

All of these investigations utilize unilateral perturbations to the walking surface stiffness induced using the Variable Stiffness Treadmill (VST) system. The VST provides a unique platform for investigating mechanisms of inter-leg coordination through stiffness perturbations. The system has been detailed in previous work [16], [17] and will not be described in this paper for brevity.

B. Experimental Protocol

While there are slight variations in experimental conditions depending on the sensory modality under investigation, the general protocol for all experiments is as follows:

The subject walked on the treadmill at a constant speed of $0.60\,m/s$ for the duration of the experiment while being

Jeffrey Skidmore is with the Department of Biomedical Engineering, The Ohio State University, Columbus, OH, 43210 USA.

Panagiotis Artemiadis is with the School for Engineering of Matter, Transport and Energy, Arizona State University, Tempe, AZ 85287 USA
Email: skidmore.104@osu.edu, panagiotis.artemiadis@asu.edu
*Corresponding author

supported with approximately 30% BWS. The right treadmill belt was not allowed to deflect for the duration of the experiment thus preventing any direct perturbation of the right leg. Low-stiffness perturbations were imposed on the left leg by commanding the surface underneath the left leg to maintain a stiffness of $1\,MN/m$, which is considered to be rigid, as the default stiffness level. Then, the stiffness would be suddenly changed to a smaller magnitude, thus inducing an unexpected perturbation. The low stiffness perturbation began shortly after heel strike (approximately $130\,ms$) 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 several steps until another perturbation was commanded.

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).

C. Data Acquisition and Processing

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. Muscle activity was obtained using surface electromyography (EMG) via a wireless surface EMG system (Delsys, Trigno Wireless EMG) and recorded at 2000 Hz. 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 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 gait cycles at rigid stiffness (except for two cycles following a perturbation to eliminate any residual effects from the perturbation) are included in the unperturbed data set. This results in kinematic data and normalized EMG signals as a function of percent gait cycle, where 0% corresponds to the heel strike of the left leg.

D. Biomechanics Model

The body does not detect changes in joint angles directly, but rather through muscle length and velocity sensed by muscle spindles. Therefore, ipsilateral kinematics will not be discussed directly in this work, but they will be used in biomechanical simulations to understand the proprioceptive feedback. The OpenSim software package [18] was used to perform these analyses with the experimental data recorded in the experiment described in [13]. A bipedal, 10 DOF model with 18 muscles (9 for each leg) was chosen in order to calculate ipsilateral changes in muscle length. First of all, the model parameters were scaled with anatomical measurements (i.e. length of limb segments and total weight) of the subject. The bilateral kinematic data and ground reaction forces recorded during the experiment were then loaded in the software. The Inverse Dynamics Tool calculated the joint torques required to achieve the loaded motion. The Static Optimization Tool was then run to resolve the net joint torques into individual muscle activations by minimizing the total activation energy required to achieve the required joint torques. Finally, the Muscle Analysis tool provided detailed information regarding the muscle lengths and velocities.

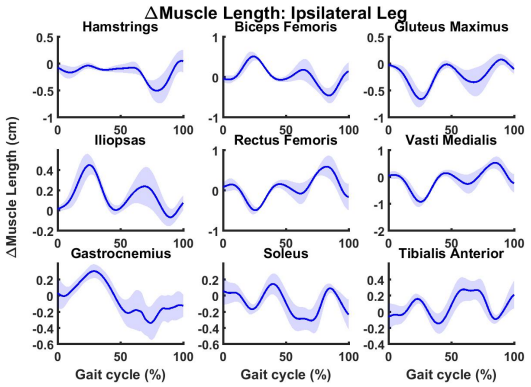

Fig. 1. Average (mean and standard deviation) change in ipsilateral muscle lengths due to unilateral stiffness perturbations.

III. Results

A. Muscle Afferents

Muscle afferents from muscle spindles and Golgi Tendon Organs (GTOs) are a part of the proprioceptive sense. Proprioception is the sense of relative positioning of parts of the body and is important in gait by regulating the amplitude of muscle output in various phases of the gait cycle and by facilitating the switch between phases [19]. Muscle spindles are situated in parallel with the muscle and provide feedback to the central nervous system by encoding the length of the muscle as well as its change over time (i.e. muscle velocity). GTOs are in series with the tendons of the muscles and sense the tension (i.e. muscle force) in the muscle.

As shown in previous work [13], [17] there are distinct changes in ipsilateral kinematics due to the unilateral stiffness perturbations. In order to understand the difference in feedback information being relayed to the central nervous system by the muscle afferents, the average change in muscle length was calculated when comparing gait cycles with perturbations and gait cycles without perturbations. In order to do this, the average muscle length (provided by the biomechanics model) across all normal cycles preceding a perturbation cycle (i.e. up to the gait cycle immediately after the previous perturbation) were subtracted from the muscle length during that perturbation cycle. This difference was then averaged across all perturbations of the same type (i.e. magnitude and timing). The change in muscle length (mean and standard deviation) of all nine modeled muscles on the ipsilateral leg for one type of stiffness perturbation (20 kN/m during the loading response) is shown in Fig. 1.

Due to changes in all three ipsilateral joint angles (hip, knee, ankle) there are changes in all nine of the muscles. Moreover, antagonistic muscles have reciprocal changes in muscle lengths. This can be seen clearly in the soleus and tibialis anterior, as well as the gluteus maximus and iliopsas.

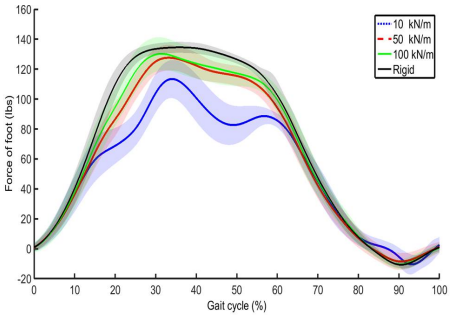

Fig. 2. Foot force (mean and standard deviation) over one gait cycle at four levels of walking surface stiffness.

Therefore, the simulation data matches the experimental data from [13]. The main conclusion from this analysis is that proprioception is definitely affected by the stiffness perturbations, through unilateral changes in muscle length, and must be accounted for in understanding the observed sensorimotor mechanism.

B. Skin Afferents

Cutaneous feedback from skin afferents is utilized for correcting the placement of the foot during gait and provides information with regards to foot contact and interaction with the walking surface [20]. Stiffness perturbations were chosen for this research because they allow the subject to maintain contact with the walking surface, thus preventing a total loss of force feedback and high impact forces upon re-establishing contact with the walking surface.

In a previous experiment [13], subjects experienced several perturbations to the walking surface stiffness in which the stiffness was dropped from 1 MN/m, which is considered to be rigid, 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 stance phase. The average force over the gait cycle for four levels of walking surface stiffness for a representative subject from this experiment is shown in Fig. 2.

As can be seen, there are only slight deviations from the normal force profile during the high and medium stiffness perturbations of 100 and 50 kN/m, respectively. The force during the low stiffness perturbation of 10 kN/m is significantly less than the normal profile during the single support phase of the left leg. This difference is explained by the compliance in the BWS that was implemented by design. A spring is mounted on the body-weight system which allows the subject's body to lower with the treadmill deflection. This allows the foot to continue to apply a similar force to the treadmill for most levels of stiffness, as shown by the 100 and 50 kN/m profiles. However, with larger deflections of the treadmill, such as those seen with low stiffness perturbations (i.e. 10 kN/m), the spring on the BWS is completely compressed by the weight of the subject, and acts like a hard stop. Therefore, the harness will carry a greater load as it prevents the subjects from falling. Nevertheless, there is continuous contact with the walking surface during stiffness perturbations, thus the body can always detect that a surface is present (as evidenced by the nonzero force profile). Therefore, any changes in cutaneous feedback due to the stiffness perturbations are assumed to be negligible during the stiffness perturbations.

C. Audition

Sound can be incorporated in locomotion by utilizing rhythmic sounds to organize movement and mark time. The use of rhythmic acoustic stimuli, such as metronomes or music, has gained popularity in gait rehabilitation of various movement disorders, including stroke and Parkinson's disease [21], [22]. While there is no auditory indication of an upcoming perturbation during an experiment, a sound is created when the linear actuator moves to create a change in walking surface stiffness. A couple of investigations were performed in order to investigate the contribution of auditory feedback in the observed contralateral response.

First, an investigation was performed in which a subject walked on the VST with and without noise canceling headphones and experienced the same stiffness perturbations. The perturbations for this investigation were applied during the loading response with a magnitude of 60 kN/m, in the same manner as utilized in previous experiments [13]. Moreover, the subject walked at a speed of 0.6 m/s and was provided with 30% BWS, to be consistent with previous experiments. The subject experienced 20 perturbations for each experimental condition (i.e. with and without headphones).

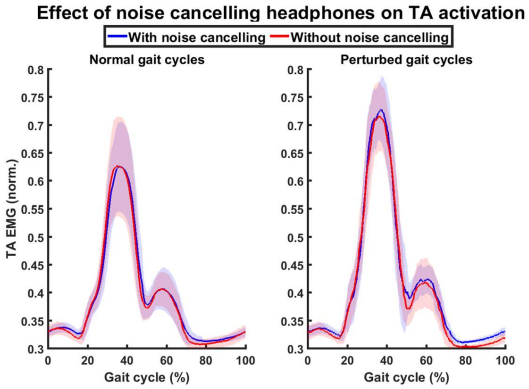

Fig. 3. Normalized TA EMG with and without noise canceling headphones for both normal (left) and perturbed (right) gait cycles.

The average tibialis anterior (TA) activation while wearing and not wearing the noise canceling headphones are plotted on top of each other for comparison in Fig. 3 for both normal and perturbed gait cycles. The plot on the left of the TA activation with and without noise canceling headphones serves as a validation that the same muscle activation is seen during both trials. The plot on the right shows that there is no change in evoked contralateral TA activation due to the noise canceling headphones. Therefore, it is concluded that the noise of the linear actuator during a perturbation does not contribute to the evoked contralateral response. However, comparing the level of TA activation from the perturbed gait

cycles (right plot) with the normal gait cycles (left plot) confirms the previously presented result that unilateral low stiffness perturbations applied during the loading response result in increased contralateral TA activation [13].

Second, an intense auditory stimulus can generate a startle response in humans that is partially seen in legs [19], [23]. Subjects report that they do not feel startled when experiencing a perturbation with our platform, but in order to objectively verify that a startle response is not occurring, EMG electrodes were placed bilaterally on the the sternocleidomastoid (SCM) muscles while subjects experienced low stiffness perturbations. Three subjects participated in this investigation and each subject experienced 50 perturbations that were applied during the loading response with a magnitude of 20 kN/m, in the same manner as was described in previous work [13]. Each subject walked at 0.6 m/s and was provided with 30% BWS to ensure consistency with previous experiments.

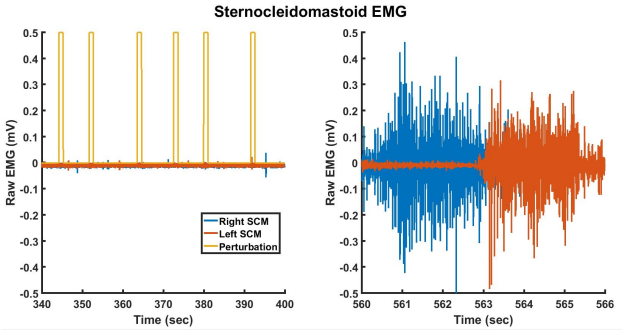

Fig. 4. Raw sternocleidomastoid EMG during treadmill walking with random stiffness perturbations (left) and when voluntarily contracting muscles after walking by looking left and then right (right).

There was no significant difference in EMG for either the left or right SCM. For comparison with voluntarily activated SCMs, the raw EMG from the experiment is plotted in Fig. 4, where the data is plotted for 1 minute of experiment time followed by the subject looking left and then right for 6 seconds after the walking portion of the experiment. An indication of when stiffness perturbations occurred is also shown. This comparison reveals that there is no substantial activation of the SCMs during the experiment. Since startle responses are characterized by rapid bilateral sternocleidomastoid muscle activation within 80ms of stimulus onset [23], it is concluded that a startle response is not elicited by low stiffness perturbations. Therefore, it is concluded that the audition has no effect on the contralateral response and is not a part of the related sensorimotor mechanism.

D. Vestibular

The vestibular system provides information that is used for orientation, balance, and posture [19] and patients with vestibular deficiency show abnormal gait patterns [24], [25]. The vestibular system is located inside each ear and is composed of the saccule and utricle that detect gravity and linear accelerations and three nearly orthogonal semicircular canals that detect rotational accelerations. The use of 30% BWS for

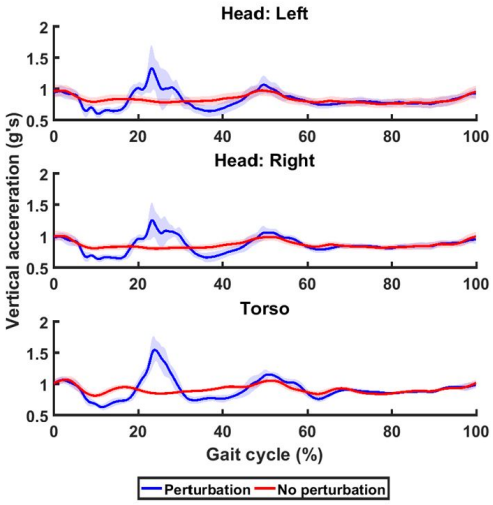

Fig. 5. The average vertical acceleration (mean and standard deviation) of the body over the gait cycle from accelerometers placed on the left of the head (top), right of the head (middle), and torso (bottom), for a representative subject.

the majority of this research was chosen to partially support the patient, thereby providing postural stability. This was intended to eliminate vestibular responses due to maintaining balance. However, inherent in a stiffness perturbation is the deflection of the treadmill under the load of the subject. Therefore, some amount of vertical linear acceleration would be anticipated.

In order to experimentally verify the change in vestibular feedback during stiffness perturbations, an investigation was performed in which 3-axis accelerometers (Delsys Trigno) were placed bilaterally on the mandible (so as to roughly approximate the location and function of the saccule and utricle) and on the upper sternum. Two subjects walked on the treadmill at 0.6 m/s with 30% BWS for at least 250 gait cycles. The perturbations for this investigation were applied during the loading response with a magnitude of 20 kN/m, in the same manner as was described in previous work [13]. Each subject experienced the described perturbations 17 times.

The vertical acceleration of the body as recorded from the accelerometers for a representative subject is shown in Fig. 5. As is expected for normal gait on a flat walking surface, there are small fluctuations in vertical acceleration during gait cycles without a perturbation (shown in red) as the center of mass of the subject rises and falls in a cyclic manner [26]. Additionally, there are significant deviations from the normal profile during the stiffness perturbations which verifies that the vestibular feedback is perturbed during stiffness perturbations. An intense change in vestibular input could also elicit a startle response [27], but the startle response has already been eliminated as a contributing factor as described above.

E. Vision

The visual system is used to choose a direction and avoid obstacles during locomotion [19] and is also important in postural stability [28]. In the experiments with stiffness perturbations, there are no indications preceding a perturbation that would alert the subject to an upcoming perturbation. The first visual indication of a perturbation is when the lever arm attached to the treadmill platform deflects upward due to the rotation of the treadmill platform about its pivot point under the load of the subject. This is also coupled with a small vertical deflection of the treadmill (<5 cm). However, this is a very minor change in the optical flow field and assumed not to be involved in the perception of the perturbation. While there is support for the existence of a short-latency visuomotor pathway for the leg [29], this pathway was evoked with visual perturbations of 21 cm changes in target foot placement, which are significantly larger visual perturbations than in our experiments. Additionally, the use of 30% BWS for the majority of this research was chosen to partially support the patient, thereby providing postural stability, and reducing the need for visual feedback. Therefore, for the purpose of this study, vision is concluded to not be influential in the sensorimotor mechanism under investigation.

IV. Discussion

A sensorimotor mechanism consists of 3 components: 1) sensory perception, 2) motor output, and 3) connecting neural pathway. The systematic investigation of the role of several sensory modalities (muscle afferents, skin afferents, vision, audition, vestibular) in Section II has shown that the body detects unilateral changes in walking surface compliance created by the VST through proprioceptive and vestibular feedback. The motor output and neural pathway have been investigated in our previous work [11], [13]–[15] and will be summarized here briefly for completeness of presentation of the sensorimotor mechanism of inter-leg coordination.

A. Motor Output

An early study of applying varying magnitudes of unilateral low stiffness perturbations with the VST to healthy subjects revealed an increasing level of evoked contralateral response - both kinematic and muscular - with increasing perturbation magnitude [13]. This systematic response was repeatable and predictable for perturbations beginning at early stance phase. A later study demonstrated the varying contralateral response to perturbations beginning at varying phases of the gait cycle [11]. Recently, comparable responses were observed in hemi-paretic patients when experiencing the same low-stiffness perturbations that healthy subjects had experienced. [15].

B. Neural Pathway

Our previous work has provided results indicating that this sensorimotor mechanism is mediated through the brain, and is not simply a spinal reflex [11], [14]. We hypothesize that the neural pathway of this sensorimotor mechanism passes through the motor cortex and may be considered as what has been defined as a "transcortical reflex loop" [30]. Support for this hypothesis is provided in 3 ways: 1) the latency of the contralateral response, 2) the timing of the evoked muscle activation during the gait cycle, and 3) a study with EEG recordings.

1) Latency of Response: The latency between the onset of the unilateral stiffness perturbation and the evoked contralateral muscle activation averaged across subjects and experiments in a previous study was a mean of 202 $\pm$ 60 ms [11]. A delay of this duration is longer than the 50-80 ms latency that would be seen for spinal reflexes [31], [32]. Rather delays greater than 125 ms correspond to transcortical circuitry [30], [33], [34], suggesting that supra-spinal regions are stimulated through the low stiffness perturbations

2) Timing of Response: In addition, the timing of the evoked muscle activity within the gait cycle suggests that supraspinal structures modify the amplitude of the neuromuscular response to sensory stimuli created by sudden changes in surface stiffness but does not initiate activation of the muscles in gait. This is consistent with the theory that supraspinal structures are not responsible for generating basic gait motor patterns through cyclical flexion and extension of the joints, but rather in modulating these basic gait patterns with descending inputs [19]. In our previous studies [11], [14], [15] the majority of the evoked muscle activity occurs only when the muscle is normally active. Specifically, evoked EMG in the TA is seen during the swing phase and beginning of the stance phase, with the greatest change in EMG occurring at the same time (approximately 30% of the gait cycle) as the peak EMG during normal walking. This result also agrees with research that has shown motor evoked potentials (MEPs) in plantar- and dorsi-flexors evoked by transcranial magnetic stimulation (TMS) are only evident during phases of the gait cycle when a muscle is active [35]. For example, MEPs in the soleus are present during stance and absent during swing which corresponds to the activation of the soleus during normal walking. [26].

3) EEG Recordings: Our previous study in which healthy subjects experienced unilateral low stiffness perturbations while wearing EEG sensors showed changes in brain activation that appeared between the onset of the unilateral perturbation and the evoked contralateral muscle activation [14]. Moreover, the main concentration of significant changes in brain activity was seen in the medial side of the left brain near the mid-coronal plane which is the approximate location of the medial section of the primary motor cortex which is associated with motor output in the lower limb of the right leg. This result agrees with other studies that have shown the existence of a long-loop transcortical reflex that can modulate a stretch reflex [30], [34]. One study showed suppression of the tibialis anterior stretch reflex in early stance phase by repetitive TMS over the motor cortex, which indicates that this reflex is modulated through the motor cortex [34].

V. Conclusions

This paper presents a systematic investigation of the role of several sensory modalities (muscle afferents, skin afferents,

audition, vestibular, vision) which reveals that the body detects unilateral changes in surface compliance created by the Variable Stiffness Treadmill setup through proprioceptive and vestibular feedback. We also show that the evoked contralateral response to the unilateral stiffness perturbations is not elicited by a startle response. We conclude that the repeatable and predictable contralateral response seen in previous studies is a transcortical reflex loop that may pass through the motor cortex which has potential to improve gait rehabiliation outcomes after stroke. The physiological insights gained through this study could be beneficial for individualized, model-based gait therapy. Once a model of this sensorimotor mechanism is developed, sensory stimuli can be tuned for the specific patient in order to evoke a desired and predictable response.

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