2014 5th IEEE RAS & EMBS International Conference on
Biomedical Robotics and Biomechatronics (BioRob)
August 12-15, 2014. São Paulo, Brazil

Dynamic simulation of hip strategy of diabetic neuropathic individuals during gait

Aline A. Gomes, Arturo Forner-Cordero, Marko Ackermann, Isabel C. N. Sacco

Abstract—**Patients suffering from diabetic neuropathy present disturbed kinetic, kinematic and electromyographic gait patterns. These disturbances have been experimentally related with plantar ulcerations. However, experimental data are limited because it is not possible to record certain muscle groups (e.g, illiopsoas). In this respect, computational simulations complement the experiments. Our aim is to simulate how the neuromusculoskeletal system of diabetic neuropathic individuals deals with a reduced distal muscle function during level gait. It was hypothesized that proximal muscle compensates the reduced distal muscle function. We used a seven segment planar musculoskeletal model of the body with 8 muscles in each leg. Normal gait muscle excitation patterns were used as reference input in forward dynamics simulations. In order to simulate the neuropathic gait condition, those reference excitations were modified according to functional changes found in diabetic gait. The *tibialis anterior* (3,75%) and *gastrocnemius* (15%) excitation reduction along with *iliopsoas* (11,25%) and *hamstrings* (7,5%) excitation increase during push-off, guaranteed larger pre-swing hip flexion and smaller hip extension during stance. This motion pattern was not observed when hamstrings excitation remained unchanged. Ankle plantar-flexion during push-off and ankle flexion during swing decreased as the gastrocnemius and tibialis were functionally reduced. The musculoskeletal model was able to represent the hip strategy possibly adopted by the diabetic neuropathic patients during gait as an adaptation to loss of function in distal muscles. The increase in hamstrings function is crucial to improve the model dynamic stability opening new approaches to therapeutic handling of these patients.**

Keywords: biomechanics, gait simulation, diabetic neuropathies

I. Introduction

The literature has shown advances in the study of diabetic neuropathic gait, describing kinetic, kinematic and electromyographic patterns and investigating their relationship with plantar ulceration [1].

Smaller ankle range of motion (ROM) [2-3], alterations in spatial–temporal patterns of walking (velocity, step length, stride length, time of double support) [1,4-6], differences in kinetic patterns with modified ground reaction forces [7-8] and in ankle and knee joint moments [4, 9-10], and changes in the lower limbs muscle activation [7, 11-14] are some of the biomechanical adaptations observed during gait in diabetic neuropathic individuals.

Some studies [4, 9, 10] show controversial results for lower limb joint moments of diabetic neuropathic patients during gait. For instance, [4] reports reduced ankle extensor moment and increased hip flexor moment at push-off for diabetic neuropathic individuals. On the contrary, in [9] it is reported a reduced hip flexor moment at the end of the stance phase.

The strength reduction of the ankle extensor, typical in the diabetic neuropathy, compromises the ability to generate adequate propulsion during final stance. It is reasonable to hypothesize that a strategy to compensate for this loss of distal function is to increase hip flexion moment to bring the leg forward in the push-off phase.

This strategy has been suggested in the literature [4] as a gait pattern that diabetic neuropathic individuals might adopt, even though the underlying neuromuscular mechanisms have not been proven yet. Based on this hypothesis, some authors have proposed therapeutic interventions in order to improve the gait quality of these patients, focusing in strengthen hip flexors [15]. However, there is no conclusive study showing that the hip flexors contribute to compensate the loss of distal muscle strength in the push-off phase.

In order to confirm this hypothesis experimentally it would be required to use invasive measurement procedures, such as needle EMG during gait, that is not feasible. As an alternative, this study proposes the investigation of this compensation mechanics using a musculoskeletal model of the lower limb and exploratory forward simulations of gait. The use of dynamic simulations of gait using computational models can contribute to the understanding of gait in diabetic neuropathic patients by providing estimations of muscle activation of several muscles which are difficult or impossible to measure experimentally.

The purpose of this study is to investigate the muscle activation patterns as a result of a bilateral reduction in distal muscles function (gastrocnemius and tibialis anterior) and the probable compensations in the unaffected proximal muscles (iliopsoas and hamstrings). This was performed following a similar approach to the one presented in [16] for

A. A. Gomes is with Physical Education and Physiotherapy Faculty, Federal University of Amazonas, Amazonas, Brazil and Movement Biomechanics and Human Posture Laboratory. Physical Therapy, Speech and Occupational Therapy Department. Medicine Faculty, University of Sao Paulo, Sao Paulo, Brazil. (phone: 55-11-3091-8426; e-mail: aline.arcanjo@usp.br). Supported by FAPEAM.

I. C. N. Sacco is with the Laboratory of Biomechanics of Movement and Human Posture at the Physical Therapy, Speech and Occupational Therapy Department. School of Medicine, University of Sao Paulo, Sao Paulo, Brazil. (phone: 55-11-3091-8426; e-mail: icnsacco@usp.br).

M. Ackermann is with the Department of Mechanical Engineering, The University Center of FEI, Sao Paulo, Brazil. (e-mail: mackermann@fei.edu.br).

A. Forner-Cordero is with the Biomechatronics Laboratory, Mechatronics and Mechanical Systems Department. Escola Politécnica, UniversityofSao Paulo, Sao Paulo, Brazil. (e-mail: aforner@usp.br).

the analysis of recovery strategies to perturbations during gait. Our hypothesis was that the decreased function of the distal muscles could be compensated by a larger activation of the iliopsoas, leading to increased hip flexion moment.

II. Material and Methods

This section introduces the details of the musculoskeletal model used to simulate the diabetic neuropathic gait.

A. Musculoskeletal Model

A planar musculoskeletal model of the body [17] consisting of seven rigid body segments (trunk, thighs, shanks and feet) with $f = 9$ degrees of freedom was used. The equations of motion read as

$$\boldsymbol{M}(\boldsymbol{y})\ddot{\boldsymbol{y}} + \boldsymbol{k}(\boldsymbol{y}, \dot{\boldsymbol{y}}, \boldsymbol{t}) = \boldsymbol{q}(\boldsymbol{y}, \dot{\boldsymbol{y}}, \boldsymbol{t}) \quad (1)$$

where $\boldsymbol{y}(\boldsymbol{t})$ is the vector (dimension f) of generalized coordinates, **M** is the mass matrix, $\boldsymbol{k}$ is the vector of Coriolis and gyroscopic forces and $\boldsymbol{q}$ is the vector of generalized applied forces and includes the muscle forces. Eight muscle groups are included in each lower limb: iliopsoas, glutei, hamstrings, rectus femoris, vasti, gastrocnemius, soleus and tibialis anterior. Each muscle is represented by a three-element Hill-type muscle model and includes the first order activation dynamics and the first order contraction dynamics of each muscle [18] with muscle properties extracted from [17]. The complete musculoskeletal model has a total of 50 states in $\boldsymbol{x}$: 9 generalized coordinates, 9 generalized velocities, 16 muscle contractile element lengths (lce), and 16 muscle activations. The dynamics of the musculoskeletal system is represented by

$$\dot{\boldsymbol{x}}(\boldsymbol{t}) = \boldsymbol{f}(\boldsymbol{x}, \boldsymbol{u}) \quad (2)$$

where $\boldsymbol{u}$ are neural excitations to the muscles. The interaction between feet and ground is modeled by means of 10 nonlinear spring-damper elements uniformly distributed along each foot sole [19]. All model parameters are documented in [17-19].

B. Reference Gait

The reference normal gait patterns for the model, which reproduce normative gait patterns fairly well, where obtained by solving an optimal neuromuscular control problem, see [19] for details. This problem consists of searching for time histories of controls $\boldsymbol{u}(\boldsymbol{t})$ and states $\boldsymbol{x}(\boldsymbol{t})$ that minimize a cost function J, and satisfy the musculoskeletal dynamics Eq. (2) and constraints that guarantee periodicity of gait and physiological muscle forces ($0<\boldsymbol{u}<1$). The cost function was composed by two terms, one penalizing muscle activation squared and the other quantifying the deviation of model kinematics and ground contact forces from experimental data available in [20] as

$$\boldsymbol{J} = \sum_{i=1}^{8} \frac{1}{T} \int_0^T a_i^2 \, dt + \frac{1}{5} \sum_{j=1}^{5} \frac{1}{T} \int_0^T \left(\frac{Z_j - \breve{Z}_j}{\sigma_j} \right)^2 dt \quad (3)$$

where a_i is the activation of the i[th] muscle, the Z_j's are the horizontal GRF, vertical GRF, hip angle, knee angle and ankle angle, and $\breve{Z}_j$ and σ_j are, respectively, the corresponding mean values and the standard deviations reported in [20].The average walking speed was prescribed as 1.1 m/s [16]. The resulting optimal control problem was transformed into a large-scale Nonlinear Programming problem using direct collocation [17, 21] and solved using the SNOPT package, a large-scale, sequential quadratic programming optimization code for Matlab (Tomlab Optimization Inc., Pullman, WA).

C. Simulation of Gait Hip Strategy

Prior to the gait hip strategy simulation, it was necessary to reduce the number of muscle combinations in the strategies proposed in the model. In other words, to reduce the number of degrees of freedom. It was assumed that human walking results from the combination of locomotor muscle synergies [22, 23]. Therefore, we analyzed the neural excitation matrix of the planar musculoskeletal model with a non-negative matrix factorization (NNMF) algorithm. The NNMF allowed the identification of the muscle combinations that played a major role in the generation of the model gait pattern. NNMF decomposition technique assumes that the set of measured data is composed of linear combinations of a smaller number of underlying elements. The components are independent, so no component can be defined as a linear combination of the other components [24]. We considered four components of the NNMF algorithm to interpret the combinations of muscle excitations in the model gait pattern (table 1). The choice of four components resulted from the compromise between the order reduction and the error in the root mean squared residual of the reconstructed data, which was below 0.16.

TABLE I. COEFFICIENTS OF NON NEGATIVE MATRIX FACTORIZATION OF NEURAL MUSCULAR EXCITATION MATRIX OF THE PLANAR MUSCULOSKELETAL MODEL

Muscles	Coeff.1	Coeff. 2	Coeff. 3	Coeff. 4
R_*Iliopsoas*	0,2119	0,0635	**0,6571**$^{\#}$	0,0000
R_*Glutei*	0,0042	0,0404	0,0024	0,0383
R_*Hamstrings*	**0,4383**$^{\&}$	0,1225	0,0021	0,0000
R_*Rfemoris*	0,0000	0,0000	0,0535	**0,6382**$^{\$}$
R_*Vasti*	0,0000	0,0479	0,0000	0,2081
R_*Gastrocnemius*	**0,8015***	0,0000	0,0000	0,0000
R_*Soleus*	0,1604	0,0000	0,0103	0,0220
R_*Tibialis anterior*	0,0000	**0,2676**$^{+}$	**0,3201**$^{+}$	0,1059
L_*Iliopsoas*	0,0627	0,2141	0,0000	**0,6559**$^{\#}$
L_*Glutei*	0,0401	0,0042	0,0384	0,0026
L_*Hamstrings*	0,1215	**0,4362**$^{\&}$	0,0000	0,0028
L_*Rfemoris*	0,0000	0,0000	**0,6375**$^{\$}$	0,0537
L_*Vasti*	0,0474	0,0000	0,2081	0,0000
L_*Gastrocnemius*	0,0000	**0,8023***	0,0000	0,0000
L_*Soleus*	0,0000	0,1603	0,0220	0,0103
L_*Tibialis anterior*	**0,2691**$^{+}$	0,0000	0,1054	**0,3209**$^{+}$

*,+,&,#,$ indicate muscles with NNMF coefficient higher than 0,25.

R means right and L means left-

The NNMF analysis suggests that the muscles were grouped according to their function during gait, showing the muscles that work together in the gait phases, and also showed a lateral symmetry. For instance, coefficient 1 has larger weights for right hamstrings (knee flexor) and gastrocnemius (both knee flexor and ankle plantar-flexor), while coefficient 2 had similar weights of the left hamstrings and gastrocnemius. By visual inspection of the table, it can be observed that a cut-off value of 0.25 is appropriate to discriminate muscles (in bold) associated to each of the four coefficients.

Afterwards we modified the activation functions of the identified muscles while maintaining proportionality in the weighting functions. Therefore, a reduction in the gastrocnemius due to neuropathy would result in a compensatory increase in the hamstrings in order to maintain the synergy. Although the rectus femoris is also identified in the NNMF table as important based on the cut-off coefficient criteria adopted, it was not altered because it has a function similar to the iliopsoas. The changes were bilateral and proportional during the stance and the swing phases, in three gait sub phases: heel strike to foot-landing (100 - 300 ms); toe-off and early swing (300 - 600 ms); and mid and late swing (600 - 800 ms). These three gait sub phases were adopted because the activation of the chosen muscles are essential in these particular phases of the gait cycle.

The angular displacements of the hip, knee and ankle joints were calculated by forward simulation using the modified neural muscular excitations reported in table 2. Based on previous EMG studies in diabetic population, we chose to reduce in 15% the neural excitation of the gastrocnemius muscle (100% change) and the tibialis anterior, iliopsoas and hamstrings excitations were altered as a percentage of the gastrocnemius alteration, according to the weights of the NNMF coefficients (25% reduction of tibialis anterior, 50% increase of hamstrings and 75% increase of iliopsoas).

TABLE II. PERCENTAGE OF NEURAL MUSCULAR EXCITATION REDUCTION IN TIBIALIS ANTERIOR (TA) AND GASTROCNEMIUS (GA) AND INCREASE IN ILIOPSOAS (IL) AND HAMSTRINGS (HT) MUSCLES

Conditions	**Muscles**				
	Time Interval (ms) of Gait Cycle	*TA*	*GA*	*HT*	*IL*
A	100 – 300	- 3,75%	---	---	---
	300 – 600	- 3,75%	- 15%	+ 7,5%	+ 11,25%
	600 – 800	- 3,75%	---	---	---
B	100 – 300	- 3,75%	---	---	---
	300 – 600	- 3,75%	- 15%	---	+ 11,25%
	600 – 800	- 3,75%	---	---	---
C	100 – 300	- 3,75%	---	---	---
	300 – 600	- 3,75%	- 15%	+ 7,5%	+ 15%
	600 – 800	- 3,75%	---	---	---
D	100 – 300	- 3,75%	---	---	---
	300 – 600	- 3,75%	- 15%	---	+ 15%
	600 – 800	- 3,75%	---	---	---

III. RESULTS

Joint kinematics was affected by the changes in neural muscular excitation in the model. The reduction in the tibialis anterior and in gastrocnemius excitation, together with an increase in the iliopsoas and in the hamstrings excitation during push-off, resulted in a greater hip flexion in the swing phase and a smaller hip extension in the stance phase (figure 1 – dotted and dashed lines). However, when only the function of iliopsoas was increased, without increasing the hamstrings, the gait pattern turned unstable with loss of periodicity. The iliopsoas is not able alone to remove the foot from the ground at late stance and propel the lower limb forward.

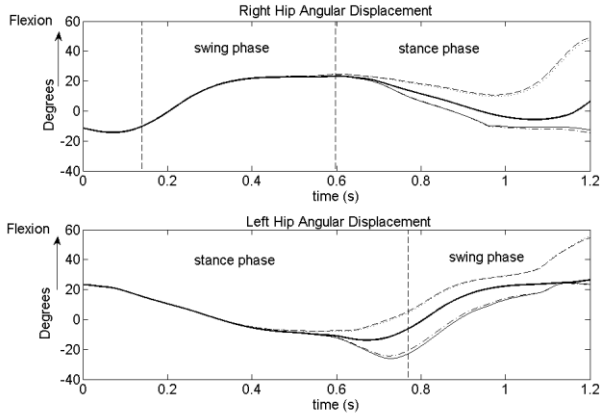

Figure 1. Angular displacement (degrees) of the right and left hip joints during the gait simulations. The lines represent neural muscular excitations (dotted - A, solid line- B, dashed line - C, dashdot line – D, bold solid line – reference neural excitation pattern without changes).

The dotted and dashed lines in Fig. 2, corresponding to conditions A and C show a reduction in the knee extension before heel strike, an increase in the knee flexion peak in the swing phase, and a knee flexion anticipation at the late stance phase (figure 2 – dotted and dashed lines).

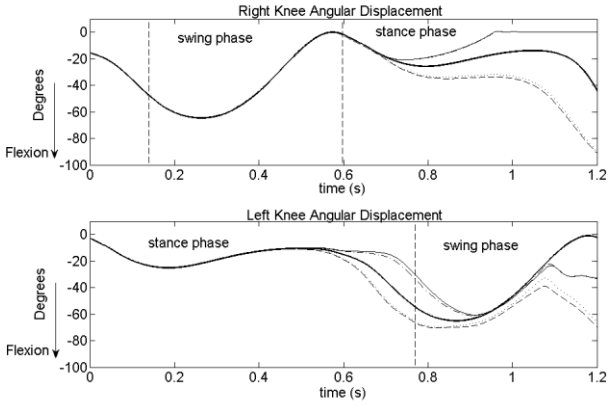

Figure 2. Angular displacement (degrees) of the right and left knee joints during the gait simulations. The lines represents neural muscular excitation (dotted - A, solid line - B, dashed line - C, dashdot line – D, bold solid line - neural excitation matrix without changes)

The ankle flexion increased during the stance phase and decreased in the swing phase as the gastrocnemius and tibialis anterior function was reduced (figure 3 – dotted and dashed lines).

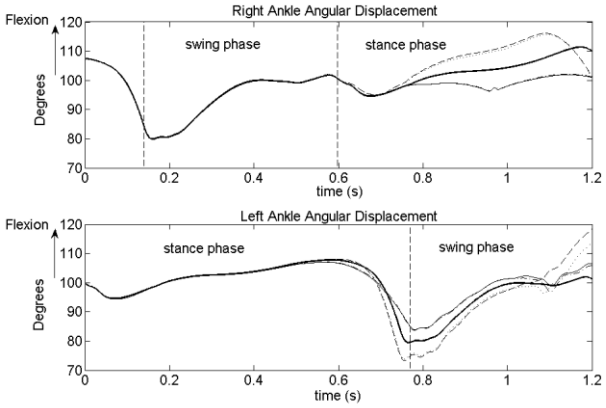

Figure 3. Angular displacement (degrees) of the right and left ankle joints during the gait simulations. The lines representes neural muscular excitation (dotted - A, solid line- B, dashed line - C, dashdot line – D, bold solid line - neural excitation matrix without changes).

IV. Discussion

Our aim was to investigate the muscle coordination adaptations as a result of bilateral reduced distal muscle function (gastrocnemius and tibialis anterior) during level gait, particularly, in terms of the proximal muscles role (iliopsoas and hamstrings). Our hypothesis that the decreased function of distal muscles would be compensated by a larger activation of the iliopsoas was partially confirmed, because it was necessary to associate the increased iliopsoas function to an increased hamstrings function to achieve a stable and periodical gait in this condition.

The literature reports that diabetic neuropathic individuals presented delayed activation of tibialis anterior [7,11] and delayed gastrocnemius medialis peak activation in the stance phase [14,15]. The reduced tibialis anterior function could hinder the appropriate excentric control during the flat-foot phase of the gait. The gastrocnemius medialis delayed activation, in turn, may compromise propulsion with a reduction of the maximum ankle plantar flexion moment in diabetic neuropathic patients [3].

The activation of the hip flexors in the late stance, when the body is in a closed kinematic chain, drives the lower limb forward providing hip and knee flexion in order to compensate for the altered gastrocnemius function [4]. Our musculoskeletal model was able to show this pattern. However, the reduced gastrocnemius and tibialis anterior functions along with the increase in iliopsoas function resulted in a trip (foot scuffing) if the hamstrings excitation was not increased simultaneously.

The hamstrings activation appears to be important to remove the foot from the ground in the absence of normal gastrocnemius activation and could be critical for gait stability in diabetic neuropathic subjects. In fact, [12] shows an anticipation of the medial hamstrings activation in the initial swing and a delay in the hamstrings deactivation during stance phase in neuropathic diabetic patients compared to non-diabetic individuals in gait.

It must be noted that the model adopted in this study presents some limitations. First of all, it can be argued that it is a planar model. However, while most of the motion during gait occurs in the sagittal plane, there could be additional effects in the frontal and coronal planes that might have some influence on the adaptation strategies. Also, the muscle excitation patterns adopted as reference in this work were based on those obtained from simulations. Nevertheless, some of the muscles involved cannot be measured directly in human gait experiments, thus justifying this approach.

Finally, the study is based on gross manipulation of reference neural excitation patterns and forward simulations and it can be noted that the simulation results from altered neural excitation patterns do not result in a stable periodic motion. Predicting muscle coordination adaptations while guaranteeing a periodic motion would require re-solving the optimal control problem, a task which is the focus of ongoing efforts.

V. Conclusion

The musculoskeletal planar model adopted in this study was able to represent the hip strategy possibly adopted by the diabetic neuropathic population during gait as a response to reduced function of distal muscles, particularly, the tibialis anterior and the gastrocnemius. An increased iliopsoas function without concomitant increased hamstrings function was show to result in model instability during gait. The increased hamstring activation appears to be important to help removing the feet from the ground in late stance and initial swing in the presence of weakened ankle plantar flexors. This indicates the key role of the hamstrings to compensate for the functional loss of distal muscles typically found in diabetic neuropathy.

Acknowledgment

A.A.G. thanks Amazonas Research Foundation (FAPEAM) for doctorate fellowship (Process: 197/2013).

A.F.C. thanks the National Research Council of Brazil (CNPq) for grants (311659/2012-8 and 478863/2010-1).

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