

Customizing to User Functional Electrical Stimulation of Walking: Optimal Control

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An improved planar biomechanical model of a human leg has been developed to investigate automatic control for Functional Electrical Stimulation (FES). The model comprises three body segments (trunk, thigh and shank-foot complex) and two joints (hip and knee). The actuators of the system were assumed as two antagonistic muscles acting at each of the joint. Each equivalent muscle (flexor and extensor) is modeled by a three component multiplicative model (driving force), a two component multiplicative passive visco-elastic model (opposing force) with exponential limiters representing the ligaments and other tissues at boundary positions. The complexity of model was driven by the needs that the model parameters must be estimated for an eventual individual with disability. The model was simulated in the MatLab environment. The simulation used dynamic programming to resolve the problem of redundancy. This model is suitable for off-line testing of the FES system.

Keywords: Functional Electrical Stimulation (FES), human leg, modeling, walking, optimal control.

1. INTRODUCTION

Functional Electrical Stimulation (FES) can assist individuals with disability to stand and walk [1]. The functional electrical stimulation needs to activate paralyzed muscles of the legs in synchrony with non-paralyzed muscles and results with healthy like standing and walking [2]. There are many muscles that are used for normal standing and walking; yet only a fraction of those can be effectively activated by means of electrical stimulation. Fortunately, some muscles that can be easily activated are the most important ones for the standing and walking (e.g., *Quadriceps m.* extends the knee joint and partly contributes to the hip flexion, *Gastrocnemius m.* controls the ankle extension, *Tibialis Anterior m.* controls the dorsi flexion of in the ankle joint, etc.). This work aims to contribute to better control with paralyzed muscles. Better control in this work was defined as the one that leads to better tracking of the desired trajectories and minimized activation of muscles.

The optimization that we adopted needs sufficiently complex, yet, practical biomechanical model of the body when walking. Most biomechanical models used for simulation of the walking are far too complex and comprise too many parameters that can not be identified to describe individual characteristics of an individual with disabilities. These models are appropriate for studying of the walking of able-bodied individuals. The

walking can often be reduced into a dominant component in the sagittal plane. The reduction in

complexity is usually achieved by both confining the model to a plane and using a small number of links [3]. The ways of managing complexity may be summarized as follows: reducing the number of degrees of freedom analytically by finding approximations and constraints, and by designing systems with the minimum number of joints; e.g., 8 degrees of freedom are usually used to describe a seven segment body model [3,4]; and decomposing a complex problem into several simpler ones by (e.g., separating the control of quantities that do not interact significantly).

A seven link planar model was developed by Onyshko and Winter [4]. Hatze [5,6] used the Lagrangian approach to model the human musculoskeletal system. This model comprised a linked mechanical and musculo-mechanical and it is developed as a set of ordinary first-order differential equations which described the dynamics of the segment model and muscle model respectively. Marshall et al. [7] used a general Newtonian approach to simulate an N-segment open chain model of the human body. The model simulated planar movement using data for joint torques and initial absolute angular displacements and velocities for each body segment. Zajac et al. [8,9] developed a planar computer model to investigate paraplegic standing induced by Functional Electrical Stimulation (FES). Yamaguchi and Zajac [3] reported about a minimal set of muscles that when activated lead to the walking alike healthy individual. They found that the trajectory is much more sensitive to the timing compared to the intensity of muscle activations. Abbas and Riener [10] summarized how the modeling needs to be addressed in designing controllers for electrical stimulation. Riener et al. developed further the complete

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model that is suitable for the development of controllers [11].

Here, we propose a simplified biomechanical model based on earlier developed identification procedures [12]. The model is based on physiological and anatomical considerations. The model structure is defined using information available in the literature, and data collected during experiments on individuals with disability. The overall effect of the stimulation on the muscles of the legs was considered, although the factors that influence the moment produced (i.e. muscle length, moment arm, distance between electrodes and motor points) were not modeled individually.

2. MODEL

Planar model of the leg (Fig. 1). We adopted a mechanical structure representing the leg with two synovial joints (hip and knee). This reduces the model to a moving double pendulum in the sagittal (x-y) plane. The upper segment is the thigh connected to the trunk by a hinge joint (hip - H). The lower segment is the shank-foot complex connected to the thigh via a hinge joint (knee - K). We selected to use the often used notation of introduced in Yoshikawa [13]: ϕ_K - knee angle; ϕ_H - hip angle; ϕ_S and ϕ_T - the angles of the shank and thigh with respect to the positive x axis direction; m_S and m_T - masses of the shank and thigh; J_{CS} and J_{CT} - moments of inertia of the shank and thigh for the axis perpendicular to the sagittal plane (parallel to z axis) for the center of mass; L_S and L_T - lengths of shank and thigh; d_S and d_T - distances between the proximal joints and the centers of masses of the shank and thigh. M_K and M_H are net joint torques at the hip and knee joints parallel to the z axis. It was also assumed that gravitational force (mg) acts in the opposite direction of y axis.

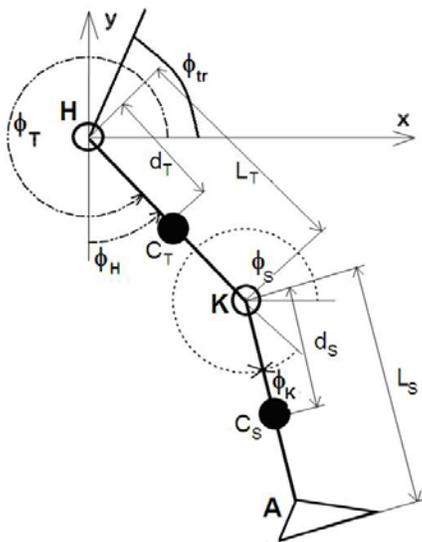


Figure 1: The biomechanical model of the leg used for simulation of walking assisted with Functional Electrical Stimulation

The equations of motion are:

$$\begin{aligned} M_H &= M_{11} \ddot{\phi}_H - M_{12} \ddot{\phi}_K - 2h_{112} \dot{\phi}_H \dot{\phi}_K + H_{122} \dot{\phi}_K^2 + g_1 \\ M_K &= M_{21} \ddot{\phi}_H - M_{22} \ddot{\phi}_K + h_{211} \dot{\phi}_H^2 + g_2, \end{aligned} \quad (1)$$

where the coefficients are:

$$\begin{aligned} M_{11} &= m_T d_T^2 + J_{CT} + \\ &\quad + m_S (L_T^2 + d_S^2 + 2L_T d_S \cos \phi_K) + J_{CS}, \\ M_{12} &= M_{21} = m_S (d_S^2 + L_T d_S \cos \phi_K) + J_{CS}, \\ M_{22} &= m_S d_S^2 + J_{CS}, \\ h_{122} &= h_{112} = -h_{211} = m_S L_T d_S \sin \phi_K, \\ g_1 &= m_T g d_T \sin \phi_H + m_S g [L_T \sin \phi_H + d_S \sin (\phi_H - \phi_K)], \\ g_2 &= m_S g d_S \sin (\phi_H - \phi_K). \end{aligned}$$

The net torques at the knee and hip (M_K, M_H) are the differences of the active and passive torques of the flexor and extensors muscles that acts at the knee and hip respectively. In this study we reduced the dimensionality of the system by introducing the equivalent flexor and extensor muscles, being equal to the net sum of all muscles contributing to the hip and knee rotations. We intentionally considered only the monoarticular muscles; yet the model could be expanded to include biarticular muscles.

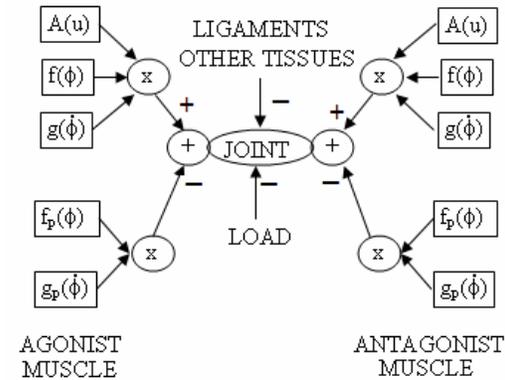


Figure 2: The model of the joint with agonist and antagonistic muscles. The multiplicative model was used for both active and passive component of the muscle force. See text for details.

The torques M_K and M_H comprise three terms each. The active flexor torque drives the joint to flexion (positive direction), while the active extensor drives the joint in extension. The other components acting are passive and they are the consequences of elastic and viscous properties of muscles and tendons exposed to external stretching, as well as the behavior of the ligaments and the surrounding tissues. With the definition of joint angles the equations for the net joint torques are:

$$\begin{aligned} M_H &= \tau_{H,f} - \tau_{H,e} - \tau_{H,p} , \\ M_K &= -(\tau_{K,f} - \tau_{K,e} - \tau_{K,p}) . \end{aligned} \quad (2)$$

Active torque model (Fig. 2). The nonlinear model of muscle dynamics used for joint angle control in this simulation software is a modified version of the Hill model [14-17]. Active muscle force depends on three factors: neural activation called recruitment, muscle length and velocity of shortening or lengthening [18]. The model is formulated as a function of joint angle and angular velocity, rather than muscle length and velocity. The three factor model is given by:

$$\tau_a = A(u) f(\phi) g(\dot{\phi}) , \quad (3)$$

where τ_a is the active torque generated by the muscle contraction; $A(u)$ is the dependence of torque on the level of evoked muscle activity u (which in turn depends on the stimulus amplitude, pulse width and stimulus frequency); $f(\phi)$ is the dependence on the angle ϕ and $g(\dot{\phi})$ is the dependence on the angular velocity $\dot{\phi}$. The parameter u is normalized to the range $0 < u < 1$ and the function g has the value of 1 under isometric conditions defined by $\dot{\phi} = 0$. The muscle model [18] described by Equation 1 can predict the muscle torque with 85-90% accuracy during simultaneous, independent, pseudo-random variations of recruitment, angle and angular velocity.

Activation dependence $A(u)$. The muscle response to electrical stimulation was approximated by a second order, critically damped, low pass filter with a delay [18-21]. The activation dynamics was expressed by

$$\frac{A(j\omega)}{U(j\omega)} = \frac{\omega_p^2}{\omega^2 + 2j\omega\omega_p + \omega_p^2} e^{-j\omega t_d} , \quad (4)$$

where $A(j\omega)$ is the Fourier transform of the muscle's contractile activity, $U(j\omega)$ is the Fourier transform of the muscle's electrical activity, ω_p is muscle's natural (pole) frequency (typically in the range of 1-3 Hz), t_d is the excitation-contraction and other delays in the muscle (typically 20-50 ms). The nonlinear model of muscle dynamics used is a modified, discrete time version of Hill's model.

Dependence of muscle torque on joint angle and speed. The nonlinear function relating torque and joint angle is simulated by a quadratic curve, $F = a_0 + a_1\phi + a_2\phi^2$. Coefficients a_0 , a_1 and a_2 define the shape of the torque-angle curve. Maximum torque is generated at an angle $\phi_{max} = a_1/2a_2$, and the torque is then $F_{max} = a_0 + 0.5 a_1^2/2a_2$. The angle and angular speed are given in rad and rad/s respectively. The nonlinear function $F = 0$, for angles

$$f(\phi) = \begin{cases} F & \text{if } F \geq 0 , \\ 0 & \text{if } F < 0 . \end{cases} \quad (5)$$

The nonlinear curve relating torque and angular velocity of the joint is simulated using a piece-wise linear approximation. Let $G = 1 + b_1\dot{\phi}$:

Passive torque model. The passive torque based on [14] can be presented with:

$$\begin{aligned} \tau_{i,p} &= c_{1,i} \exp(c_{2,i} \phi_i) - c_{3,i} \exp(c_{4,i} \phi_i) + \\ &+ c_{5,i} + c_{6,i} \phi_i + K_i \dot{\phi}_i , \end{aligned} \quad (6)$$

where the $c_{i,j}$ and K_i are constants. Equation 6 has five terms with seven parameters that need to be determined individually for every user. The first two terms are exponential limiters representing the ligaments and tendons in anatomically boundary flexion and extension. The other three terms represent the contribution of antagonist muscles when not being stimulated (passive force caused by stretch).

The model that was developed is redundant since there are two actuators per each of the joints. The second difficulty arises when both legs are on ground (double support phase) causing the kinematic chain formed by legs to be closed; thus, undetermined. The closed kinematic chain was resolved by using the experimental data for distribution between ground reaction forces on legs. In order to resolve the problem of the redundancy we used the optimization. The optimization used dynamic programming developed in our earlier works [22-26]. The dynamic programming required first the transformation of the second order differential equation to the Cauchy form:

$$\dot{X} = f(X, U) .$$

The vector of state variables is

$$X(x_1 = \phi_K, x_2 = \dot{\phi}_K, x_3 = \phi_H, x_4 = \dot{\phi}_H) .$$

The vector of controls is

$$U(u_1, u_2, u_3, u_4) .$$

The control variables were the levels of stimulation u_i ($i=1, 2, 3$, and 4) of each of the equivalent muscles, being constrained between zero (no activation) and one (maximum).

The model in state space has the following form:

$$\begin{aligned} \dot{x}_1 &= x_2 & \dot{x}_2 &= P_2 + \sum_{j=1}^8 G_{2,j} u_j , \\ \dot{x}_3 &= x_4 & \dot{x}_4 &= P_4 + \sum_{j=1}^8 G_{4,j} u_j . \end{aligned} \quad (7)$$

The terms G_{ij} and P_i ($i=2, 4$; $j=1, 2, 3$, and 4) were obtained after a series of transformations of second order differential equations of movement and they comprise customized measures of geometry and inertial of the potential user.

The optimal control algorithm is an extension of the method developed for simulation of planar model [27-29]. The cost function used for optimization is given with Equation 8:

$$R(u) = \int_{t_0}^{t_0+T} \left\{ \lambda_1 \left[\frac{(\phi_H^*(t) - \phi_H(t))}{\phi_{H,max}} \right]^2 + \left[\frac{(\phi_K^*(t) - \phi_K(t))}{\phi_{K,max}} \right]^2 + \lambda_2 \left[u_1^2(t) + u_2^2(t) + u_3^2(t) + u_4^2(t) \right] \right\} dt. \quad (8)$$

The index "max" relates to the maximum value of the joint angle. The coefficients λ_1 and λ_2 are included to allow the variation of the importance of the muscle activation (intensity) and the tracking errors to the final results. In this study we used the values 0.5 (equal importance of the minimization of the muscle efforts and the tracking errors). The first two terms in the cost function are the squares of the normalized tracking errors, while the third term is the sum of the squares of the muscle activation levels. The algorithm was implemented using SIMULINK 2.0 (Mathworks, Natick, MA, U.S.A.).

The data for analysis were the following: 1) the desired hip and knee joint angles (ϕ_K and ϕ_H); 2) the trunk angle ϕ_{Tr} ; 3) the ground reaction forces X_G and Y_G ; and 4) the acceleration of the hip \ddot{x}_H , \ddot{y}_H . This data was acquired in the professional gait laboratory at Aalborg University instrumented with the force platforms OR6-5 (Advanced Mechanical Technology Inc., Massachusetts, U.S.A.) and ProReflex Motion Capture Unit (Qualisys, Sweden). The data were collected at a PC computer through the ACB-530 serial interface board with RS-422 serial communication (Sealevel, SC, U.S.A.), and the PCI-DAS1602/16 acquisition board (ComputerBoards, Inc., Massachusetts, U.S.A.). The kinematic and dynamic data were acquired synchronously at 240 samples per second (QTrac Capture & View software, Qualisys, Sweden). ProReflex system was calibrated before the beginning of the experiment; hence, all data were obtained in the absolute reference system. Off-line analysis used low-pass filtering at 5 Hz using (4th order Butterworth filters). The ground reaction forces and position of the center of pressure were calculated using the recorded force plate data and the manufacturer software that was delivered with the hardware at 240 samples per second.

3. RESULTS

The input in simulation is presented in Fig. 3.

Here we limit the presentation to two set of parameters that define the model: 1) data estimated for an able-bodied individual, and 2) data estimated for an individual with incomplete paraplegia at T10 level. The paraplegic subject could generate smaller joint torques for 50 percent compared with the able-bodied individual. In both cases we used the same trajectory.

Fig. 4 shows the simulation for the able-bodied individual. It can be seen that the tracking of desired trajectories is excellent, and that the levels of muscle activation are small.

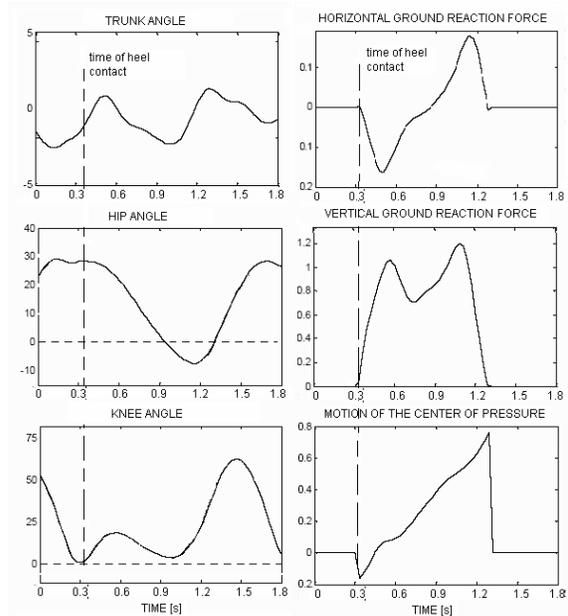


Figure 3: The input into simulation of the walking assisted with Functional Electrical Stimulation. See text for details

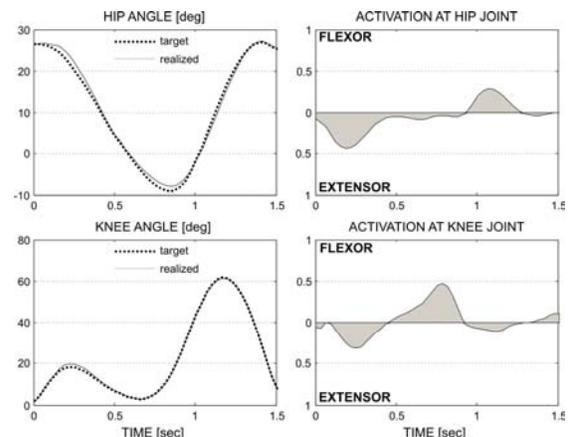


Figure 4: The results of simulation when the model parameters are estimated for an able-bodied individual. The activations of joint torques were normalized to the maximum. The flexors and extensors are presented in opposite direction in order to suggest their opposing functioning.

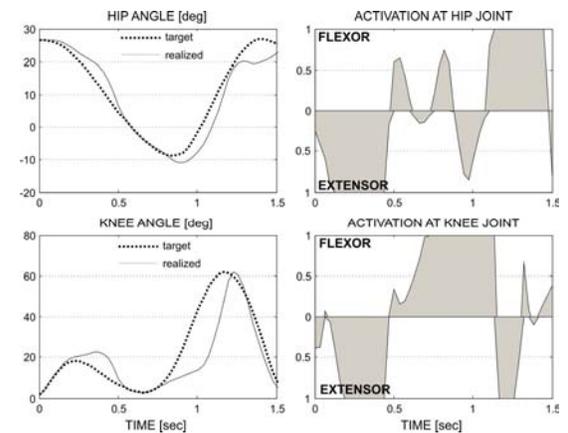


Figure 5: The simulation results for the input shown in Fig. 3 and model parameters characteristic for an individual with paraplegia. The tracking error is big, and the joint torques go into saturation ($u=1$).

Fig. 5 shows the simulation results for the individual with paraplegia. It can be seen from this figure that the tracking error was substantial, and that the level of activation reached 1, that is, maximum level of joint torques.

This result suggests that the desired trajectory is not feasible for the individual with paraplegia.

4. DISCUSSION

This study starts from a model of the body determined with user-specific parameters, individualized with respect to the lengths, masses, inertia, muscle and joint properties. The trajectory used for simulation was recorded from an able-bodied subject while walking with ankles fixed using ankle-foot orthoses. The aim of the simulation was to study plausible trajectories keeping in mind the limitations imposed by the spinal cord injury (SCI) such as spasticity, decreased range of movements in some joints, limited strength of paralyzed, externally activated muscles. If the muscles were capable of generating the movements required and the trajectory was achieved, then the simulation provided two kinds of information: 1) timing of the onset and offset of muscle activations with respect to the various gait events; and 2) patterns of activation with respect to the maximum activation.

A weakness of this model is that it reduces locomotion to a planar system, and includes only four actuators per leg. The complexity of the optimization algorithm was the main reason to select this reduced model. The limitation of the analysis to only four muscle groups is still suitable for the design of an FES system which commonly only has four to six channels of stimulation for walking with ankle-foot orthoses.

The use of a multiplicative model of joint actuators is advantageous, compared to other biomechanical models that include muscles and their tendinous geometry, because the actual characteristics of the actuators can be determined experimentally. Models dealing with muscle forces require a complete knowledge about the actuators such as: muscle force vs. length, muscle force vs. velocity of shortening, geometry of insertion points, elasticity of tendons, etc. The muscle forces or tendon properties can not be measured directly and estimation of all the anatomical data for a given subject (e.g., point of tendon insertion, position of the center of rotation, etc.) is almost impossible. As a result these detailed models are generally not anatomized for individual subjects.

For the individual with disability the mean absolute tracking errors were several times larger than those calculated for the simulation of walking for the able-bodied subject. This finding was expected, because the muscle forces from the subject with disability are smaller than the forces that an able-bodied person can develop.

Even though the walking was rather slow, relatively large joint torques were still needed, compared to the data in the literature for able-bodied humans walking. This increase results from the use of ankle-foot orthoses and the limitation to monoarticular muscles. Biarticular muscles (e.g. rectus femoris m.) are well suited for

simultaneous flexion and extension of neighboring joints which occurs during several phases of the gait cycle, so integration of these muscles into the model should decrease the levels of activation. The inclusion of biarticular muscles would introduce further redundancy. This increased redundancy could be resolved by using a cost function that separates the effects of biarticular and monoarticular muscles, or by simple algebraic constraints between the torques generated by the monoarticular and biarticular muscles. However, determining appropriate constraints will require further study, and would require data that may be very difficult to measure in subjects with SCI.

The cost function used for the simulation minimizes the overlap of agonist and antagonist activity, but not the total activity of the two muscle pairs. Reducing cocontraction will reduce muscle fatigue by providing rest periods for the muscle during each step. Fatigue is often the limiting factor in application of electrical stimulation to subjects. However, the absence of cocontraction may not be completely beneficial, because in many activities, including gait, cocontraction increases stiffness and the resistance to perturbations. Including cocontraction would require a modification of the cost function.

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**ПРИЛАГОЂЕЊЕ ОБЛИКА ФУНКЦИОНАЛНЕ
ЕЛЕКТРИЧНЕ СТИМУЛАЦИЈЕ ЗА ХОД:
ОПТИМАЛНО УПРАВЉАЊЕ**

**Александар Вег, Дејан Поповић,
Страхиња Дошен**

Усавршени, равански, биомеханички модел људске ноге развијен је са циљем унапређења примене Функционалне Електричне Стимулације (ФЕС). Модел укључује три телесна члана (торзо, бутина, подколеница) са две кинематске везе (кук и колена). Актуатори у систему су два пара антагонистичних мишића са дејством у везним тачкама. Сваки од еквивалентних мишића (флексор и екстензор) је моделиран трокомпонентним мултипликативним моделом који одговара активној сили, и двокомпонентним мултипликативним моделом који одговара пасивним, виско-еластичним елементима, отпорна сила. Укључена су експоненцијална ограничења која представљају лигаменте и тетиве, као и друга везивна ткива у граничним положајима. Комплексност модела је проистекла из потребе да се управљање прилагоди индивидуалним особинама (параметри модела) за појединог пацијента. Модел је развијен у MatLab 7.2 окружењу. Коришћено је динамичко програмирање да би се решио проблем динамичке неодређености система. Модел се показао погодним за иницијална испитивања ФЕС опреме и почиње се са тестирањем на пацијентима.