Proceedings of the 28th Conference
on Decision and Control
Tampa, Florida • December 1989

FEEDBACK GAINS FOR CORRECTING SMALL PERTURBATIONS TO STANDING POSTURE

Jiping He[1], William S. Levine[2] and Gerald E. Loeb[3]

[1]Center for Biological Information Processing, MIT
[2]Electrical Engineering Department, University of Maryland, College Park, MD 20742
[3]Bio-medical Engineering Unit, Abramsky Hall. Queen's University, Kingston, Ontario K7L 3NG, Canada

ABSTRACT

A dynamical model of the neuro-musculo-skeletal mechanics of a cat hindlimb has been developed to investigate the feedback regulation of standing posture under small perturbations. The model is a three-joint limb, moving only in the sagittal plane, driven by 10 musculotendon actuators, each having response dynamics dependent on activation kinetics and muscle kinematics. Under small perturbations the nonlinear postural regulation mechanism is approximately linear. Sensors exist which could provide state feedback. Thus, the linear quadratic regulator is proposed as a model for the structure of the feedback controller for regulation of small perturbations. System states are chosen to correspond to the known outputs of physiological sensors: muscle forces (sensed by tendon organs), a combination of muscle lengths and velocities (sensed by spindle organs), joint angles and velocities (sensed by joint receptors), and motoneuron activities (sensed by Renshaw cells). Thus the feedback gain matrices computed can be related to the spinal neural circuits. Several proposals for control strategy have been tested under this formulation. It is shown that a strategy of regulating all the states leads to controllers that best mimic the externally measured behavior of real cats.

1. INTRODUCTION

The use of control theory to provide insight into neurophysiology has a long history. As early as the beginning of the century, Sherrington and his colleagues described stretch reflexes [31], a phenomenon caused by the feedback action of muscle spindles, sensors of a combination of muscle length and rate of change of muscle length.

Most of the previous work on muscle regulation has been based on the theory of single-input, single-output (SISO) servo-mechanisms. An example of particular relevance to our research is Merton's [25] proposal that the stretch reflex was an experimental observation of a motor control strategy, namely servo-control of individual muscle length by the spindles. Houk et al. [12] later proposed the regulation of individual muscle stiffness (by sensors of both length and force) as the motor control strategy.

It has recently become possible to trace the sensory feedback pathways through the spinal cord. It is now known that sensory feedback exhibits considerable cross coupling among the actuators [7,13,34]. In other words, the neuromuscular control system is certainly multiple input multiple output (MIMO).

Because of the enormous number of neurons and reflex pathways involved in the control of locomotion, a comprehensive identification of the structure of the feedback control circuitry is a formidable experimental task. A theoretical approach based on a carefully developed model could shed light on its likely general structure, thereby providing guidance to experimenters. Such a model must consider the mechanical plant consisting of limb segments and musculotendon actuators and the feedback controller, consisting of the reflex pathways in the spinal cord circuitry.

The regulation of static posture, and the closely related but more difficult, dynamic stabilization of locomotion, are rich subjects for theoretical and experimental study. It is quite clear that the highly coupled mechanical structure of the articulated leg [20,32] and complex dynamics of the musculotendon actuators [35] constitute a complicated nonlinear system.

However, under small perturbations this highly nonlinear but smooth [12] system could be well-approximated by a linear system. The fact that physiological sensors exist for all of the state variables provides plausibility for a linear state feedback controller. Thus, a linear quadratic regulator (LQR) is proposed as a model for the neuromuscular regulation of posture. By choosing system states to correspond to physiological sensors, the resulting controller predicts feedback projection patterns of sensory feedback upon spinal interneurons, thus generating some testable hypotheses about interneuronal connections for experimental neuroscientists to investigate.

The feedback gains of the LQR depend on the weighting matrices in the performance criteria. Several possibilities were investigated, including those which correspond to optimal MIMO implementations of both length and stiffness control strategies.

We would be remiss if we did not mention the pioneering paper by Chow and Jacobson [3] which was the first to utilize an LQ model to study human locomotion. Our paper is able to improve substantially the relevance to neurophysiology by studying a simpler problem, the regulation of posture, and by utilizing a greatly improved model.

In the next section, we deal with the dynamics of the neuromuscular skeletal control system (NMSCS) and the formulation of the optimization problem. In section 3, we explain the solution of the optimization problem and its relation to the proposed control strategies. In section 4, we make some connections between the model predictions and experimental data. Finally, we point out some directions for future research.

2. THE MODEL

The NMSCS of a cat hindlimb is a highly efficient locomotion machine. It has been used widely in studies of biomechanics and neurophysiology to understand kinematics and sensorimotor control [14,15,21,32].

In this study, a cat hindlimb is modeled in the sagittal plane by a three joint linkage system driven by musculotendon actuators. More than 30 muscles on the limb are grouped into 10 according to their attachment geometries and actions in the sagittal plane. The model preserves the inter-segmental coupling and multi-articular muscle structure, but has manageable complexity. The complete model includes four major components: limb mechanics, musculotendon actuators, activation kinetics, and sensors.

* This work was supported in part by NIH Contracts #NO1-NS-3-2348 and #NO1-NS-6-2300.

CH2642-7/89/0000-0518$1.00 © 1989 IEEE

2.1 The Dynamics of the Musculoskeletal Mechanics

Mechanical structures similar to the articulated leg in our model have been studied extensively in both robotics research and biomechanics. Therefore the derivation of the dynamics is omitted. The major differences are in the choice of coordinate systems and the generation of joint torques. We use intersegment joint angles ($(\varphi_h, \varphi_k, \varphi_a)' = \varphi$) as the generalized coordinates. The joint torques are generated by a set of 10 musculotendon actuators. This can be seen from figure 1 where the mechanical structure of the system is represented.

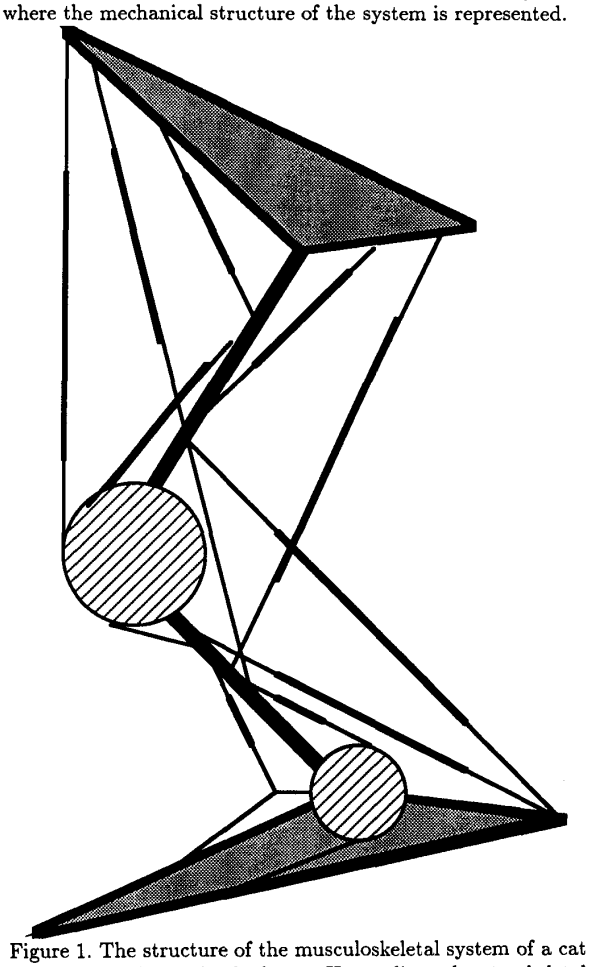

Figure 1. The structure of the musculoskeletal system of a cat hindlimb in the sagittal plane. Heavy lines denote skeletal segments. Light lines denote tendons. Intermediate lines denote muscles. Muscles are grouped according to attachment geometry. The circle at the knee indicates that the patella is modelled as a pulley. The circle at the ankle denotes a pulley that models the ankle crossing of several dorsi-flexors of the toes.

The dynamical equations of the system for a standing posture are of the form:

$$\ddot{\varphi} = J^{-1}(\varphi)\left[M(\varphi,\dot{\varphi}) + N(\varphi)g + \left(\frac{\partial L_p}{\partial \varphi}\right)' F_t + \beta F_b\right] \quad (1)$$

where

$J(\cdot)$: the moment of inertia matrix;

$M(\cdot)$: the vector of centrifugal and Coriolis forces;

$N(\cdot)$: the coefficients for the gravitational force;

g: the gravitational constant;

$(\frac{\partial L_p}{\partial \varphi})'$: the matrix of moment-arms for musculotendon actuators. The musculotendon lengths $L_p = (L_{p1}, L_{p2}, \cdots, L_{p10})'$ are determined from the limb configuration and the attachment geometry. See He [10] for details;

$F_t = (F_{t1}, F_{t2}, \cdots, F_{t10})'$: the force outputs of the musculotendon actuators;

$F_b = (F_x, F_y)'$: the action of the body on the leg at the hip joint.

2.2 The Dynamics of Musculotendon Actuator

There are several approximations involved in this model. It is clear that cats use their toes to help maintain their balance. However, the forces produced by the muscles that control the toes are relatively small. Modelling the effect of these muscles is very complex because of the many independent articulations involved. Experimental data about toe muscle forces is very difficult to obtain. Thus, the toes have been replaced by a single point ground contact.

Another major approximation is the assumption that the effect of the other three legs and the rest of the cat's body can be replaced by a force vector at the hip. While this would be unrealistic for many motions it is reasonable for some small perturbations. In particular, it is a reasonable model for experiments in which the contact point of one leg is displaced.

As the actuator, muscle in series with tendon (musculotendon) has always been one of the focal points of motor control research [27,29]. Surveys of current knowledge about muscle physiology can be found in Gans [6] and McMahon [24]. There are many unsettled questions about muscle properties and dynamics, some of which were investigated recently by Rindos [29]. We assumed that all muscles in the model have the same dynamical characteristics and differ only in specific parameters. Hence, parameters in this section are all scalars referring to a general musculotendon actuator.

In a recent review paper, Zajac presented an excellent summary on modeling the musculotendon actuator [35]. Many forms of dynamical model have been developed to suit different research interests. Most of them are based on the structure shown in Fig. 2. In modeling muscle, it is critically important to define carefully the conditions under which muscles operate. At a standing posture, some muscles are excited to various degrees, while others are completely silent and generate only passive forces when stretched. An applied perturbation of limb position will stretch some muscles while shortening others. This requires a muscle model that can deal simultaneously with both active and passive muscles under all dynamic conditions, that possesses the most relevant properties of musculotendon tissues, and that is simple to use. None of the muscle models found in the literature fully satisfy the requirements because they either need some unobtainable parameters (Hatze [8]) or lose controllability for passive muscles (Zajac [36]). We modified Zajac's model to deal with the controllability problem while retaining the elegance of the original model. matic drawing of muscle-tendon structure and force generation components.

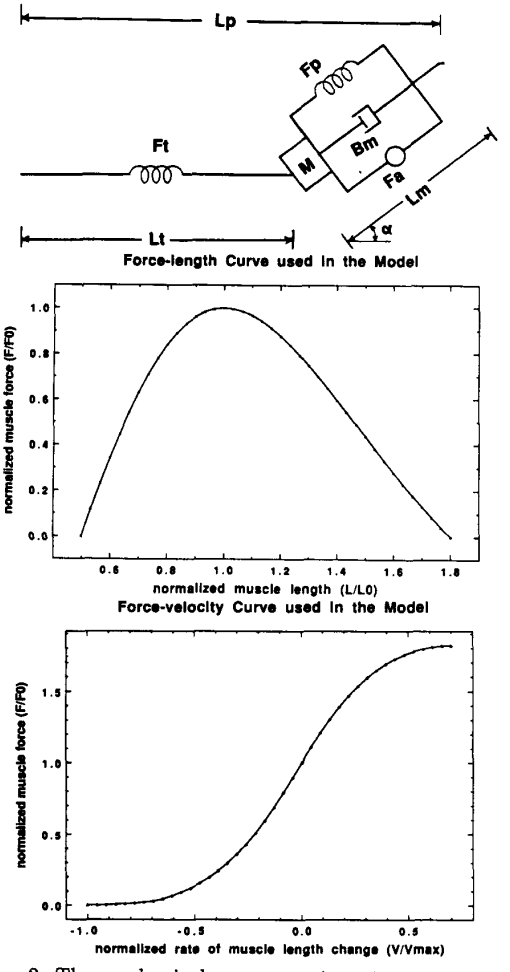

Figure 2. The mechanical representation of the musculotendon actuator and major muscle characteristics. A. is the schematic drawing of muscle-tendon structure and force generation components. B is a plot of the force-length relation. C is a plot of the force-velocity relation.

In solving for the neuromuscular controls to achieve maximum height jumps, Zajac et al. assumed a minimum activation ($a(t) \geq a_{\min} > 0$) for all muscles [36]. They assumed that individual muscle mass can be ignored compared to the limb mass being driven and that muscle internal viscosity is negligible. They solved $\dot{L}_m$ from the relation

$$F_t - F_p \cos\alpha = F_z f_L(L_m) f_V(\dot{L}_m) a \tag{2}$$

to get

$$\dot{L}_m = f_V^{-1}\left(\frac{F_t - F_p \cos\alpha}{F_z f_L(L_m) a}\right) \tag{3}$$

where

$F_p(F_t, \varphi)$ is the passive force of a muscle due to stretch, and is determined by the musculotendon force F_t and joint angle φ;

F_z is the maximum isometric muscle force the musculotendon actuator can achieve under activation (a constant for each muscle);

$f_L(L_m)$ is the force-length relation of muscle due to myofilament overlap, given in figure 2;

$f_V(\dot{L}_m)$ is the force-velocity relation of muscle, an intrinsic property of the cross-bridges between the myofilaments that generate the active force as shown in figure 2;

$L_m(F_t, \varphi)$ is the relative length of muscle fibers (and hence sarcomeres) which is determined once F_t and φ are given;

$\alpha(F_t, \varphi)$ is the pinnation angle of muscle w.r.t. the action line of F_t, determined by F_t and φ;

$a(t)$ is the mechanical activation level of musculotendon actuator (after motoneuron commands have been transformed by the activation kinetics of the muscle fibers; see Eqn. 10 below).

The dependence on F_t and φ of F_p, L_m, and α is given by equations:

$$F_p = \frac{k_{ml}}{k_{me}}(e^{k_{me}(L_m - L_{ms})} - 1) \tag{4}$$

$$L_m = \sqrt{L_w^2 + (L_p - L_t)^2} \tag{5}$$

$$L_t = L_{ts} + \ln(k_{te} F_t / k_{tl} + 1)/k_{te} \tag{6}$$

$$\cos\alpha = (L_p - L_t)/L_m \tag{7}$$

where k_i's are spring constants and L_p is the musculotendon length defined earlier.

Substituting $\dot{L}_m$ into the tendon force-length relation gives the dynamics for the musculotendon actuator:

$$\dot{F}_t = K_t(F_t)\left[\dot{L}_p - \frac{\dot{L}_m}{\cos\alpha}\right] \tag{8}$$

where $K_t(F_t)$ is the stiffness function of tendon tissues.

This is a model which is computationally tractable yet includes most known properties of muscle. In reality, however, muscles are often completely relaxed, particularly during slow walking and standing. When $a(t) \to 0$, the above model is not defined. We have shown elsewhere that it is not always justifiable to ignore muscle mass (He [9]). Hence, an obvious modification to the model is to include the muscle mass. The corresponding dynamics are given directly by using Newton's laws for the musculotendon actuator shown in Figure 2:

$$\ddot{L}_m = \frac{1}{M_m}\left[F_t \cos\alpha - \left(F_z f_L(L_m) f_V(\dot{L}_m) a + F_p + B_m \dot{L}_m\right) \cos^2\alpha \right] + \frac{\dot{L}_m^2 \tan^2\alpha}{L_m} \tag{9}$$

This generates a two-state representation of the musculotendon actuator. This model can be considered as a general version of many currently available, first-order models. For example, the first-order model given earlier can be derived from the model in Eqn. (9) by applying the technique of singular perturbations, assuming muscle mass to be a small parameter. For detailed derivation and discussion, see He [9] and Zajac [35].

2.3 Activation Dynamics

The activation dynamics describes the relation between the neural input to the musculotendon actuator and its mechanical activation. The most important characteristics to be included in the activation dynamics are the different time constants for activation and deactivation, the low-pass filter property, and the saturation of activation. The independence of activation dynamics from muscle contraction dynamics is also assumed (Zajac [35]), although this remains controversial (Rack [28]).

A first-order, nonlinear differential equation is used to describe the dynamics of activation of the musculotendon actuator:

$$\dot{a}(t) = (u(t) - a(t))(c_1 u(t) + c_2) \tag{10}$$

where u is the neural input (excitation), taken as the rectified smoothed electromyogram (EMG), $c_1 + c_2$ is the activation rate constant (when $u = 1$), and c_2 is the deactivation rate constant (when $u = 0$).

This activation dynamics has the following features:

1. Mechanical activation ($a(t)$) follows excitation ($u(t)$) asymptotically, and is bounded within $[0, 1]$.
2. It has a larger rate constant for activation than deactivation, conforming with the experimental evidence that muscle force rises much faster than it decays.
3. It is an analytical function suitable for feedback control analysis.

2.4 Physiological Sensors

The limb is equipped with thousands of proprioceptive sense organs whose transduction and encoding properties have been studied intensively. Major physiological sensors considered in our model include muscle receptors and joint receptors. We have considered only the most sensitive and fastest conducting signal sources, presuming these to be most useful for servo-regulation.

Among the muscle receptors we have modeled muscle-spindle organs sensing muscle kinematics (length and rate of change in length), and Golgi tendon organs sensing muscle force. Spindles reside deep inside muscles and are parallel with the main muscle fibers, thereby experiencing the same stretch as the muscle fibers. There are direct control neurons (γ motoneurons) innervating spindles, to modulate their sensitivity according to the anticipated range of motion. It is clear now that the ouputs of spindles go not only to motoneuron pools controlling the same muscle, but also to those controlling other muscles, forming a complex feedback network [21]. The output signals of spindle organs correspond to a nonlinear function of muscle kinematics, i.e. they are sensitive to both muscle lengths and rate of change of length. Furthermore, the nature of this function can be changed dynamically by the γ motoneurons (see Loeb [18] and Matthews [21]). Since our study is restricted to posture control during small perturbations, a linear combination of the two mechanical inputs (length and velocity) is a reasonable approximation of activity recorded from hindlimb spindles in naturally behaving animals [17,19]. The effect of active modulation on spindle organs during standing is simulated by a scaling scheme that limits the range of spindle output within $[0, 1]$:

$$\begin{cases} s_1 \times \delta L_m + s_2 \times \delta \dot{L}_m = & \text{maximum of } (\delta L_m + \delta \dot{L}_m) \\ s_1 \times \delta L_m = & 0.5 \text{ maximum of } (\delta L_m + \delta \dot{L}_m)] \end{cases} \tag{11}$$

The sensitivity of tendon organs is not modulated. Their output signals represent simply tensions, influenced little by rates of change of tension. The discharge rate of tendon organs increases monotonically with tension in muscle, though not exactly linearly [21]. It is quite reasonable to assume a linear function for tendon output during standing posture. As for spindles, tendon organs are known to also have a wide-spread feedback projection through inhibitory interneurons.

Figure 3 shows a schematic diagram for some of the known feedback projections of the muscle receptors. For simplicity, only the connection between neurons of a pair of agonist and antagonist muscles at one joint are drawn, though the real projection pattern is much more diffuse [14,23].

Notice from the figure the connection of a special interneuron—the Renshaw cell (RC). It receives input from a collateral of the axon coming out of the motoneuron and feeds back the signal to both the original motoneuron and to other motoneurons. Neuroscientists have proposed several functions for RC [2]. We believe it is an estimator of muscle activation level, because of the signal it receives and its projection within the spinal cord. Since RC was never considered as a sensor before, no experimental data is available to relate the discharge rate of the neuron to motoneuron activity or muscle activation level.

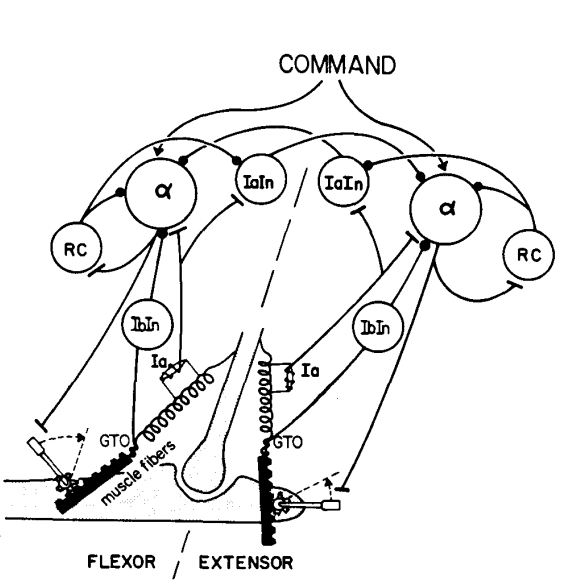

Figure 3. Major afferent pathways in the muscle control loop. For involuntary and small perturbation regulation, only muscle and joint receptors and Renshaw cell recurrent pathways are considered.

The functional role of joint receptors has been controversial (Ferrell [4]). Intuitively, some sensors are needed to provide information about joint angles and angular velocities. Joint receptors are natural candidates. However, many experiments indicate that signals from joint receptors occur predominantly at extreme or unusually loaded joint positions [22], leading to speculation that their role in locomotion is to signal readiness for the step phase transition [33]. Since many of those experiments were conducted on anaesthesized animals and results were obtained from some subset of all receptors on a joint, one can not conclude that joint receptors are not used as joint sensors in a normal sense. We assume in our model the availability of joint angle and angular velocity information from joint receptors. We have also examined the observability of the system without explicit joint receptors. The system is observable [9] so it would be possible to incorporate an estimator for joint angle feedback, based on spindle and tendon organ signals.

In summary, we have incorporated sensors based upon four known receptor modalities: joint receptors ($y_1(\varphi, \dot{\varphi})$) for joint angles and angular velocities; tendon organs ($y_2(F_t)$) for force outputs; spindles ($y_3(L_m, \dot{L}_m)$) for a combination of muscle

lengths and velocities; and Renshaw cells ($y_4(a)$) for activation levels in muscles. These provide for a full-state feedback scheme that can be used to model the feedback projection network residing in the spinal cord for the involuntary control of movement. An alternative to the full state feedback scheme, incorporating an observer to estimate joint angles and angular rates from signals from the other three sensor modalities, has also been developed (see He [7]).

2.5 The Feedback Structure of the NMSCS

In order to relate our results to the physiology, we chose system inputs to be the neural inputs to musculotendon actuators, and the outputs to be the outputs of the physiological sensors identified above. A block diagram for the NMSCS dynamics is given in Figure 4. The feedback pathways have been identified as Ia (muscle spindle primary endings) for muscle length and velocity, Ib (tendon organs) for muscle force, Rc (Renshaw cells) for activation level. Because of the uncertainty of joint receptor action in locomotion control, a switch is shown in the figure to indicate uncertainty whether a joint angle receptor or an estimator is used.

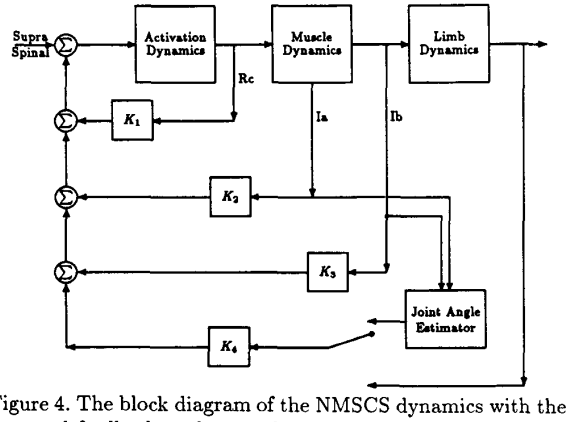

Figure 4. The block diagram of the NMSCS dynamics with the neuronal feedback pathways. Engineering approaches can be used to approximate the feedback structure, $K_i, i = 1, 2, 3, 4$ of the system.

For a standing posture, the system response to a small perturbation will be well represented by a linear system. The behavior of the spinal neural controller for involuntary action should also be close to linear. Under these assumptions, we linearized the system dynamics around a quiet standing posture where the nominal values for all parameters are available from experimental measurement. Let δ denote the variation of a variable from its nominal value. Then the system state vector in the linearized system is given by $x = (\delta\varphi, \delta\dot{\varphi}, \delta F_t, \delta\dot{L}_m, \delta a)'$. The linearized system matrices are

$$A = \begin{pmatrix} 0 & i_{3\times 3} & 0 & 0 & 0 \\ \frac{\partial f_1}{\partial x_1} & \frac{\partial f_1}{\partial x_2} & \frac{\partial f_1}{\partial x_3} & 0 & 0 \\ \frac{\partial f_2}{\partial x_1} & \frac{\partial f_2}{\partial x_2} & \frac{\partial f_2}{\partial x_3} & \frac{\partial f_2}{\partial x_4} & 0 \\ \frac{\partial f_3}{\partial x_1} & 0 & \frac{\partial f_3}{\partial x_3} & \frac{\partial f_3}{\partial x_4} & \frac{\partial f_3}{\partial x_5} \\ 0 & 0 & 0 & 0 & \frac{\partial f_4}{\partial x_5} \end{pmatrix}$$

$$B = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ \frac{\partial f_4}{\partial u} \end{pmatrix}$$

$$C = \begin{pmatrix} \frac{\partial y_1}{\partial x_1} & \frac{\partial y_1}{\partial x_2} & 0 & 0 & 0 \\ 0 & 0 & \frac{\partial y_2}{\partial x_3} & 0 & 0 \\ \frac{\partial y_3}{\partial x_1} & 0 & \frac{\partial y_3}{\partial x_3} & \frac{\partial y_3}{\partial x_4} & 0 \\ 0 & 0 & 0 & 0 & \frac{\partial y_1}{\partial x_1} \end{pmatrix}$$

where f_1 is the skeletal dynamics (Eqn. 1), f_2 is the tendon dynamics (Eqn. 8), f_3 the muscle dynamics (Eqn. 9), and f_4 the activation dynamics (Eqn. 10). Consequently, $A \in R^{36\times 36}, B \in R^{36\times 10}, C \in R^{33\times 36}$.

To find the linear feedback controller, we assumed a linear quadratic regulator. The performance index to be optimized has the form:

$$J(u) = \frac{1}{2}\int_0^\infty (x'Qx + u'Ru)\,dt \tag{12}$$

This formulation provides us with two weighting matrices. In effect, at this point the problem is an inverse optimal control problem. Given that the control is linear state feedback, what are the Q and R for which this feedback is optimal?

As an important principle of neuromuscular organization, each muscle fiber receives innervation from only one motoneuron, and one motoneuron can innervate many fibers of the same muscle [2,24]. We have also assumed that muscle activation is independent of muscle contraction dynamics. Consequently, muscle excitations are independent of each other. Thus, R is assumed to be a diagonal matrix to reflect the independence of excitations for different muscles. Even though the weighting factor in R for each muscle can be different in accordance to its activity at a specific posture, to concentrate on the investigation of the effects of different assumptions about motor control strategy, we used the same weighting for all muscle excitations. Therefore, R is of the form rI.

The choice of Q matrix is determined by the control strategy to be simulated.

1. Under the "joint position servo" control strategy, what is to be minimized is the error in joint positions:

$$x'Qx = \sum_{i=1}^{3} q_i \delta\varphi_i^2 \tag{13}$$

2. Under the "length servo" strategy, the controller should regulate the system in such a way that muscle lengths are maintained at their desired values. Correspondingly,

$$x'Qx = \sum_{i=1}^{10} q_i \delta\ L_{pi}^2 = \sum_{i=1}^{10} q_i \left(\frac{\partial L_{pi}}{\partial \varphi}\delta\varphi\right)^2 \tag{14}$$

3. Under the "muscle stiffness" control strategy, proposed by Houk [12] the controller is to minimize any deviation of muscle stiffness from its nominal value. Since the stiffness of a muscle is defined as the ratio between the change in force and that in length, (F_t/L_m), after linearization, we obtain:

$$x'Qx = \sum_{i=1}^{10} q_i^f \delta F_{ti}^2 + q_i^l \delta L_{mi}^2 \tag{15}$$

4. Under the "full state feedback" control strategy, the controller will modulate, in balance, all the state variables to maintain a good performance:

$$x'Qx = \sum_{i=1}^{10}(q_i^f \delta F_{ti}^2 + q_i^l \delta L_{mi}^2 + q_i^a \delta a_i^2) + \sum_{i=1}^{3}(q_i^\varphi \delta\varphi_i^2 + q_i^{\dot\varphi}\delta\dot\varphi_i^2) \tag{16}$$

3. RESULTS

In the previous section, we set up the model for the NMSCS, linearized it around a nominal standing posture, and formulated the optimization problem. The optimal control for the problem, as is well known, is the solution of the corresponding algebraic Riccati equation (ARE) [1].

$$A'K + KA + Q - KBR^{-1}B'K = 0 \qquad (17)$$

The Schur decomposition method (Laub [16]) is used to find the positive definite solution of the ARE (it is easy to show that the system is controllable and observable). For each Q matrix listed in the previous section, the feedback gain matrix was calculated for several different values of r to see how the feedback pattern changes under different levels of control effort.

The simulation results are compared to experimental measurements to test the various proposals for motor control strategy. The experimental data is from Rushmer et al. [30]. A cat standing with one foot on each of four movable force plates, has its posture perturbed by a sudden movement of one plate. The ground reaction forces were measured.

Under full state feedback and when $r = 1.0$, we obtained a ground reaction force response quite similar to the experimental measurement. The results are shown in Fig. 5. The left column shows the measured ground reaction forces, the middle shows the simulated perturbations and the computed ground reaction forces, and on the right are shown joint angle responses. From the figure it is clear that not only the shape but also the time of the responses of the model match with the data. The response of joint angles and muscle forces (Fig. 6) looks smooth and reasonable.

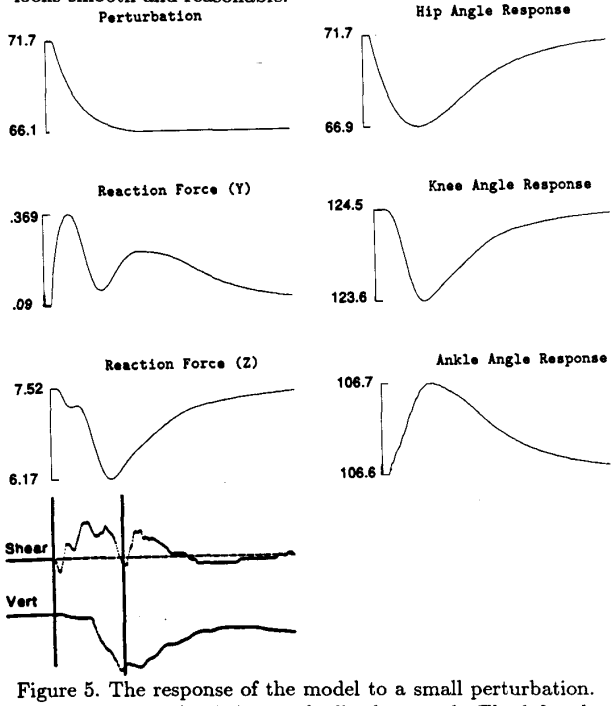

Figure 5. The response of the model to a small perturbation. The model is under full state feedback control. The left column shows the measurement of ground reaction forces from (Rushmer et al 1983).

Under the same control strategy but for different values of r, we obtained similar joint responses but ground reaction forces differed significantly.

Under the other control strategies, however, we either got large force excursions (joint position and length servo control), or large joint angle overshoots (muscle stiffness control). This is evidence that these are not realistic motor control strategies.

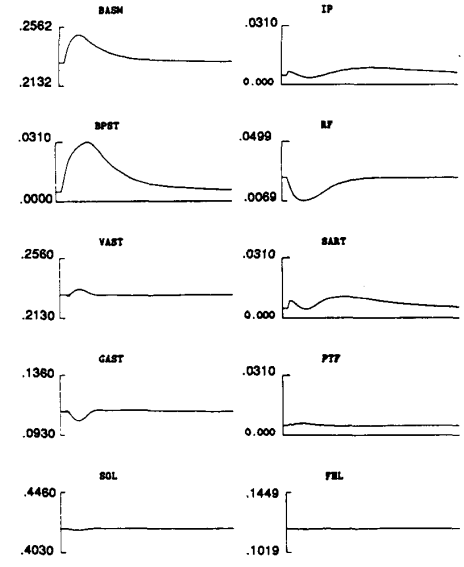

Figure 6. The predicted force responses of the model to a small perturbation. The model is under full state feedback control.

These simulations suggest that full state feedback control might be the motor control strategy used by the NMSCS. The corresponding feedback gain matrix is plotted separately in Fig. 7 for tendon organ feedback projections and in Fig. 8 for spindle organ feedback projections, respectively. Feedback gains for Renshaw cells and joint receptors are not shown here because of the lack of experimental data to give a meaningful discussion.

In Figs. 7 and 8, each small graph represents the feedback gain from one sensor to the control input of one musculotendon actuator as a function of r. The graphs are arranged in columns, according to the neural control input of one musculotendon actuator from all receptors, and in rows, according to the projections of the sensory feedback from one musculotendon actuator to neural control inputs of all actuators.

One pattern that can be immediately recognized from these feedback matrices consists of large excitatory actions (positive feedback) of spindle organs and large inhibitory actions (negative feedback) of tendon organs, on the diagonal elements. These represent the homonymous feedback, of sensors onto the actuators of the same muscles. The signs of these diagonal elements are invariant when r changes, while many heteronymous feedbacks (referring to actions and connections between different muscles) change sign. The pattern from those homonymous feedbacks is consistent with what neurophysiologists have already discovered in experiments. It is this kind of homonymous feedback pattern, and a disregard of the generally smaller heteronymous feedbacks, that prompted proposals for servo control of individual muscles, such as the length servo and muscle stiffness servo. However, there are many heteronymous feedback actions on each muscle, which are not negligible especially when r is large. The simulation suggests that $r = 1.0$ is about the level of control for the NMSCS at standing posture. Around $r = 1.0$, the homonymous actions are still larger than any heteronymous action in all muscles, but the combined

effect of the many heteronymous actions on each muscle may be as large or larger.

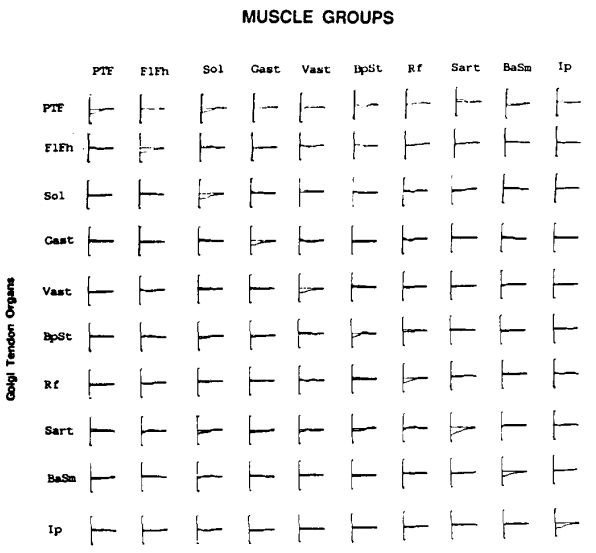

Figure 7. The feedback gain matrix from tendon organs to neural inputs. The model is under full state feedback control. Each graph represents the feedback gain as a function of r. It connects tendon organs from the muscle indicated on the left to the neural input to the muscle indicated on the top of the column.

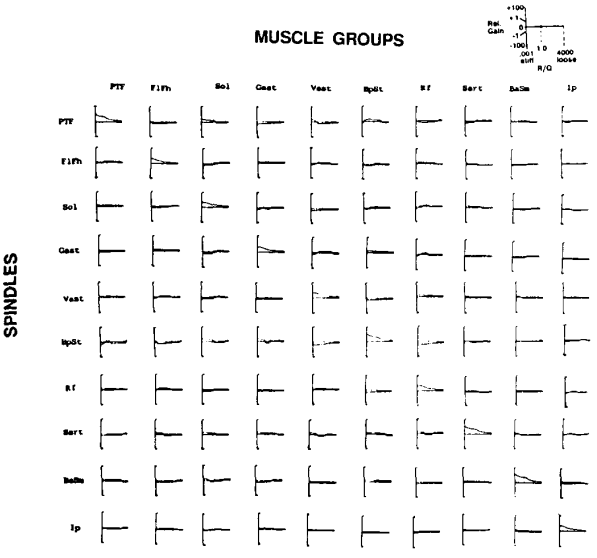

Figure 8. The feedback gain matrix from spindle organs to neural inputs. The model is under full state feedback control. Each graph represents the feedback gain as a function of r. It connects tendon organs from the muscle indicated on the left to the neural input to the muscle indicated on the top of the column.

There are a few exceptions to the homonymous inhibitory action of tendon organs. In Fig. 7 showing tendon organ feedback connections, we can see sign reversals from the homonymous feedback of BPST and PTF. PTF is the major flexor of the ankle joint, while BPST is both a hip extensor and knee flexor. Both muscle groups are silent during quiet standing but the hip flexor RF (which is also a knee extensor) is active. Therefore, the positive force feedback from these two muscle groups might have the effect of increasing the whole limb stiffness in response to the perturbation by temporary co-contraction of both extensors and flexors. These sign reversals of homonymous feedback from BPST and PTF might also have a compensating action for the strong inhibitory feedback of Renshaw cells (not shown) and excitatory action of spindles (Fig. 8) from the same muscles. For MIMO systems, feedback actions are much more complicated, and are no longer as intuitive as they are for SISO systems.

Although heteronymous feedback gains are smaller than homonymous feedback gains, a diffuse feedback projection pattern is generally evident in the feedback matrices. There are feedback connections not only among muscles acting on the same joint, but also acting on different joints. These connections to muscles controlling other joints have a general pattern, The 3 uniarticular extensors in the model attach to the limb on alternate sides: ankle extensors (soleus) on the posterior, knee extensors (vasti) on the anterior, and biceps on the posterior of the limb. Therefore, the feedback interconnections of the 3 musculotendon actuators also take alternate signs: negative on the ankle, positive on the knee, then negative on the hip. The cat hindlimb is a mechanically coupled, multi-linkage system with multiarticular musculotendon actuators. We might expect its controller to have comparably distributed structure.

How much is this feedback pattern dependent on the choice of coordinate system used in the model? Let H be a nonsingular transformation matrix relating the new system states z with x. Then we have

$$z = Hx \quad A_x = H^{-1}A_zH \quad B_x = H^{-1}B_z \quad C_x = C_zH$$

It is easy to show that the relation between the solution of the algebraic Riccati equation under the two coordinate systems is

$$K_x = H'K_zH$$

From the above relations we get

$$G_z = R^{-1}B_z'K_z = R^{-1}B_x'K_xH^{-1} = G_xH^{-1}$$

Hence the choice of the system states in the model is important to the generation of the feedback patterns mentioned above, though the control effect will be the same because the optimal control will be

$$u^* = -G_zz = -G_xH^{-1} = -G_xx$$

The lesson is that a further aspect to the use of the LQR to model the neurophysiological regulation of posture is the need to infer the appropriate choice of state variables. Fortunately, the well-defined modalities of the naturally occurring sensors correspond well to the natural state variables of a Newtonian model. Because the form of our model retains the essential structure and important physiological properties of the NMSCS, we are able to make some reasonable predictions about neural interconnections of sensory feedback pathways.

However, the neural circuits in the spinal cord are probably much more complex than even those predicted by our LQ control model. After all, we are only describing one specific function of a system with many behaviors. There are many parallel interneuronal systems residing in various reflex pathways, with different latencies. There are additional receptor modalities to those considered in the present model. By using techniques such as neuron staining, microelectrode implantation and intracellular recording, neurophysiologists have identified extensive heteronymous interconnections and some interneuronal systems in the spinal cord [13,23]. Because cats are often anaesthesized in such acute experiments, the state of the spinal cord is not known, and there is evidence that the transmission in many such circuits is modulated throughout the different phases of the walking step cycle. Moreover, because of methodological limitations those acute experiments reveal only the short latency reflex pathways involving one or two interneurons. It is the sum of all contributions from the several parallel interneuronal systems that would generate the net projection strengths that are predicted by this model.

The absolute values of feedback gains depend on the metric used in the model, so we have avoided any interpretation of those values. However, the relative magnitudes of the feedback gains in the matrix constitute important tests of the model's predictions regarding neural connections. If the model prediction is valid, wherever the model predicts small gains there may or may not be a connection; even a strong connection is not a contradiction to our model because the connection might exist, and be used, for an entirely different behavior. If the model predicts a large gain, then there needs to be a strong feedback connection with the appropriate sign to verify our model. The largest gains, which are predicted for homonymous feedback, have long been experimentally established as existing connections in the spinal neural network (Fig. 3), but their relative functional importance during natural motor behaviors remains the subject of study and debate.

The model predicts some reversals in the sign of heteronymous feedback gains, as the ratio of Q/r varies over the range from one extreme to the other. Changes of feedback sign have been observed during the various phases of locomotion in animal experiments. (Forssberg [5] and Orlovsky et al [26]). This suggests, in terms of our model, that the control strategy may change as a function of ongoing motor activity perhaps as a result of changes in performance criteria, activation state, posture or external constraints (e.g. foot in the air vs. on the ground).

4. FUTURE RESULTS

Since out model contains only one leg of a quadrupedal cat and the motion is restricted to the saggital plane, it is difficult to make direct comparisons with experimental data other than the ground reaction forces. One obvious improvement is to generalize the model into 3D space and include more legs. Then the model becomes more complex but more realistic simulation can be performed so that much more experimental data can be used to test the model.

The study of the neural control of walking and running will be more challenging because the dynamical effects become more important in the system behavior. How the neural controller modifies feedback gains to facilitate transitions between different phases of locomotion will be an interesting problem that can be addressed by the approach used here.

Even in the current model, there are still many remaining questions to be answered. The role of joint receptors can be investigated by comparing the patterns of feedback with them or with an estimator for these state variables. Muscle synergy is another issue that can be studied. We have changed Q to test assumptions about motor control strategy. R and Q can also be changed so as to emphasize coordination within selected subgroups of muscles, perhaps incorporating some known features of spinal feedback circuits to determine the relative strength of the "missing", unknown parts of the control system needed to achieve stability.

In summary, the model developed here and the formulation used in its analysis provide us with a tool to study the general structure of the neural control network of the NMSCS. Some experimentally testable predictions were generated, and several proposals for motor control strategy were examined. This form of analysis promises to reveal much about the relationship between the neural control circuitry and the musculotendon mechanics.

Please note that we are not claiming that the spinal cord functions as an LQ controller. We are using LQ theory as a tool to study the spinal cord and its relation to the limb. We have used the theory to create a family of models which predict the kinds of feedback that should occur in the spinal cord under precisely stated assumptions.

ACKNOWLEDGEMENT

The authors are grateful to Drs. C.J. Heckman, C.A. Pratt, A.J. Rindos, and J.L.F. Weytjens, for providing valuable data and suggestions.

REFERENCES

[1] M. Athans and P.L. Falb, *Optical Control*, McGraw-Hill, Inc., 1969.

[2] R. Burke and P. Rudomin, Spinal neurons and synapses. In *Handbook of Physiology—The Nervous Systems I*, Williams and Wilkins, MD, 1981.

[3] C.K. Chow and D.H. Jacobson, Studies of human locomotion via optimal programming, Math. Biosci. 10, 239-306, 1971.

[4] W.R. Ferrell, S.C. Gandevia, and D.I. McMloskey, The role of joint receptors in human kinaesthesia when intramuscular receptors cannot contribute, J. Physiol., 386:63-71, 1987.

[5] H. Forssberg, Phasic gating of cutaneous reflexes during locomotion. In A. Taylor and A. Prochazka, editors, *Muscle Receptors and Movement*, Oxford University Press, 1981.

[6] C. Gans, Fiber architecture and muscle architecture, *Exerc. Sport Sci. Rev.*, 10:160-270, 1982.

[7] Z. Hasan and D.G. Stuart, Animal solutions to problems of movement control: The role of proprioceptors, *Ann. Rev. Neurosci.*, pages 199-223, 1988.

[8] H. Hatze, A myocybernetic control model of skeletal muscle, *Biological Cybernetics*, 25:103-119, 1977.

[9] J. He, *A Feedback Control Analysis of the Neuro-musculo-Skeletal Control System of a Cat Hindlimb*, Ph.D. thesis, Department of Electrical Engineering, University of Maryland, College Park, 198.

[10] J. He, Partial inversion of cat locomotion dynamics and estimation of joint torques in the left hind limb of a walking cat. Master's thesis, Department of Electrical Engineering, University of Maryland, College Park, 1984.

[11] N. Hogan, The mechanics of multi-joint posture and movement control, *Biological Cybernetics*, 1985.

[12] J. Houk, Regulation of stiffness by skeletomotor reflexes. *Annu. Review of Physiology*, 41:99-114, 1979.

[13] E. Jankowska, Interneuronal organization in reflex pathways from proprioceptors, in *Frontiers in Physiological Research*, Australia Academic Science, 1984.

[14] E. Jankowska, T. Johannisson, and J. Lipski, Common interneurons in reflex pathways from group Ia and Ib afferents of ankle extensors in the cat, *J. Phyiol.*, pages 381-402, 1981.

[15] G.C. Joyce, P.M.H. Rack, and D.R. Westbury. The mechanical properties of cat soleus muscle during controlled lengthening and shortening movements, *J. Physiology*, 204:461-474, 1969.

[16] A.J. Laud, A Schur method for solving algebraic Riccati equations, *IEEE Trans. Automatic Control*, AC-24:913-921, 1979.

[17] G.E. Loeb, The control and responses of mammalian muscle spindles during normally executed motor tasks, *Exercise and sport sciences reviews*, 12:157-204, 1984.

[18] G.E. Loeb, What the cat's hindlimb tells the cat's spinal cord. In W. Barnes and M. Gladden, editors, *Feedback and motor control in invertebrates and vertebrates*, Croom Helm Ltd., London, 1985.

[19] G.E. Loeb, J.A. Hoffer, and C.A. Pratt, Activity of spindle afferents from cat anterior thigh muscles, I, Identification and patterns during normal locomotion, *Journal of neurophysiology*, 54(3):549-564, 1985.

[20] G.E. Loeb, W. Marks, A. Rindos, J. He, W. Robert, and W.S. Levine, The kinematics and dynamics of a cat hindlimb during locomotion, *Soc. Neuroscience Abstr.*, 1985.

[21] P.B.C. Matthews, Proprioceptors and the regulation of movement. In *Handbook of Behavioral Neurobiology: Motor Coordination*, volume 5, chapter 3. Plenum Press, New York, 1981.

[22] P.B.C. Mathews, Where does Sherrington's "muscular sense" originate? Muscles, joints, corollary discharges? *Ann. Rev. Neurosci.*, pages 189-218, 1982.

[23] D.A. McCrea, Spinal cord circuitry and motor reflexes, *Exercise and Sport Science Review*, 14:105-141, 1986.

[24] T.A. McMahon, *Muscles, Reflexes, and Locomotion*, Princeton University Press, 1984.

[25] P.A. Merton, Speculations on the servo control of movement, in *The Spinal Cord.* Little Brown, Boston, 1953.

[26] G. Orlovsky and G. Pavlova, The effect of different descending systems on flexor and extensor activity during locomotion, *Brain research*, 40:359-371, 1972.

[27] P.M.H. Rack, Limitations of somatosensory feedback in control of posture and movement, in *Handbook of Physiology—The Nervous System II*, chapter 7, Williams and Wilkins, MD, 1981.

[28] P.M.H. Rack and D.R. Westbury, The effects of length and stimulus rate on tension in the isometric cat soleus muscle, *J. Physiology*, 204:443-460, 1969.

[29] A.J. Rindos III, *Determination of Muscle Force Generation by Various Cat Hindlimb Muscles under Dynamic Conditions by Means of A Computer-Automated Experimental Paradigm*, Ph.D. thesis, Department of Electrical Engineering, University of Maryland, College Park, MD, 1988.

[30] D.S. Rushmer, C.J. Russell, J.M. Macpherson, J.O. Phillips, and D.C. Dunbar, Automatic postural responses in the cat:responses to headward and tailward translation, *Exp. Brain Res.*, 50:45-61, 1983.

[31] C.S. Sherrington, Flexion-reflex of the limb, crossed extension reflex, and reflex stepping and standing, *Journal of Physiology*, 40:28-121, 1910.

[32] J.L. Smith and R.F. Zernicke, Predictions for neural control based on limb dynamics, *Trends in Neurosci.*, 10:123-128, 1987.

[33] J.F. Stein, *An Introduction to Neurophysiology*, Blackwell Scientific Publications, 1982.

[34] J.F. Stein, What muscle variable(s) does the nervous system control in limb movements? *The Behavioral and Brain Sciences*, 5:535-577, 1982.

[35] F.E. Zajac, Muscle and tendon: properties, models, scaling, and application to biomechanics and motor control. *CRC Critical Reviews in Biomedical Engineering*, 1988.

[36] F.E. Zajac, W.S. Levine, J. Chapelier, and M.R. Zomlefer, Neuromuscular and musculoskeletal control models for the human leg, *Proc. 1983 ACC*, pages 229-234, June 1983.