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Muscle Mechanics and Dynamics of Ocular Motion*

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Abstract

A model of the neuromuscular mechanics of horizontal eye motion is developed. The model of the oculomotor system that is presented incorporates known physiological dynamics and geometry of the musculotendon complex. Muscle force development is a described by a two-component version of Hill's model and consists of a passive and active contractile component. The active component includes the force-velocity and force-length characteristics of the muscle. The passive component accounts for elastic and viscous effects. Activation dynamics couple the neural controls that are appropriate for saccadic movements to the muscle mechanics. Numerical simulations illustrate that the model successfully simulates saccadic movements and accurately depicts eye position and velocity and muscle tension.

1 Introduction

The modeling of the human ocular system and its dynamic properties have been extensively studied by neurologists, physiologists, and engineers. One of the first models of eye movement was developed by Descartes [5] in 1630 based on the principal of reciprocal innervation, a notion of paired muscular activity in which a contraction of one muscle is associated with the relaxation of the other. In 1954 Westheimer [12] developed a linear second order approximation of eye dynamics during a saccade in which the input to the model was assumed to be a step of muscle force. The model

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worked well for 10° saccades but not for larger such movements. In addition, the model predicted the unphysical results that the time of saccade duration would be independent of saccade magnitude and that the peak velocity would be directly proportional to saccade magnitude. A more realistic representation of eye movement was advanced by Robinson [11]. His linear fourth-order model could simulate saccades between 5° and 40° but the velocity profiles predicted by this model were not physically realistic. It was recognized by Westheimer and Robinson that the eye movement mechanism was inherently nonlinear, issues not addressed by their work. Roughly speaking, the nonlinear features of the system can be attributed to the geometry of the system as well as the nonlinear physiological behavior of certain components that describe the extraocular muscle. Martin and Lu [9] developed a model of the eye system that assumed a linear model of muscle behavior but accounted for the nonlinear effects that occur when the recti muscles act in a nontangential fashion on the eyeball. They were able to construct a control law that enabled the eye to track a target through a range of both large and small displacements. The muscle model that was utilized omitted some physiological features of muscle and did not distinguish the effects of passive and active muscle behavior, a notion that will be elaborated upon later. Another group of investigators have concentrated upon ocular models that emphasize the effects of muscular physiology upon system performance. Along these lines, a sixth-order nonlinear model proposed by Cook and Stark [3] and subsequently modified in [1] produced realistic position, velocity and acceleration profiles. This Cook-Clark-Stark model addressed the nonlinear relationship between force and velocity but ignored the force-length characteristics of muscle. This assumption was tantamount to assuming that the medial and lateral rectus muscles operate near the primary position that corresponds to looking straight ahead. Their model incorporates a force-velocity dependence into the active muscle by a velocity dependent viscosity that is experimentally determined by fitting experimental data to Hill's equation. The model does not include any passive viscosity and moreover, the passive elasticity is lumped together with the nonmuscular suspensory passive tissue.

The model of the oculomotor system that is presented incorporates known physiological dynamics and geometry of the musculotendon complex. In particular the model for muscle force development is a two-component version of Hill's model and consists of a passive and active contractile component. The development allows for the inclusion of very general force-velocity and force-length characteristics in the active component. Unlike the model of [1], the muscle model that is utilized here includes passive elastic and viscous effects. It is pointed out in [15, 8] that for rapid eye movements, the passive parallel elasticity is important. In

this paper, attention is focused upon saccadic eye movements which are among the fastest voluntary muscle movements the human body is capable of producing. The eye model includes activation dynamics that couple neural controls which are appropriate for saccadic movements to the muscle mechanics. It should be noted that the model which is investigated here does not account for the geometric nonlinearities addressed in [9]. A justification for this assumption is that for saccadic movements, when motion is typically less than 30°, the nontangential forces associated with the recti muscles do not occur. Before proceeding to the development of the model, a a brief review of the relevant physiology and mechanics of muscle is presented.

2 Musculotendon Dynamics

A commonly used mechanical representation of a skeletal muscle is shown in Figure 1.

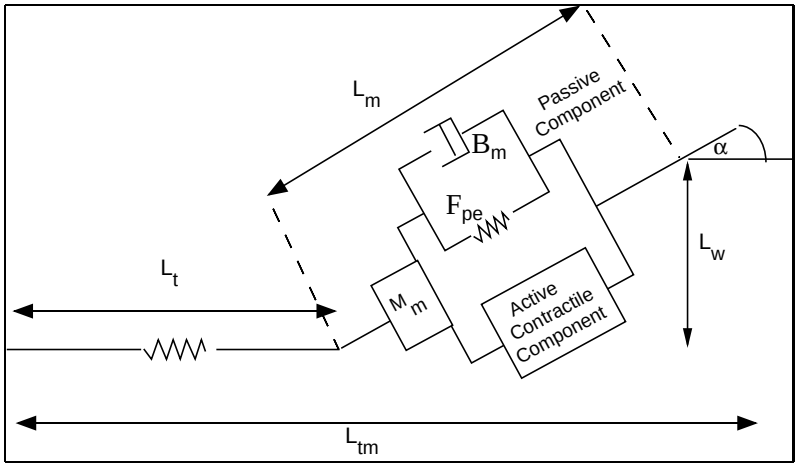

Figure 1. A mechanical representation of muscle

The muscle mass is M_m and the model includes a spring like tendon through which the muscle force is exerted. The muscle, in series and off-axis by the pennation angle α with the tendon, is assumed to consist of two components: an active force generator and a parallel passive component. The passive component is assumed to include a parallel elastic element (F_{pe}) that describes the passive muscle elasticity and a damping component which corresponds to the passive muscle viscosity (B_m). The model for the active contractile component is based on the generally accepted notion that the active muscle force is the product of three factors: (1) a length-tension

relation, (2) a velocity-tension relation, and (3) the activation level. We will denote the active muscle force in the normalized form

$$F_{act} = F_0 F_l(\bar{L}_m) F_v(\dot{\bar{L}}_m) a(t). \tag{1}$$

Neural excitation, as represented by a sequence of motor unit discharges, is coupled to the active contractile component by the variable $a(t)$, referred to as muscle activation. Activation dynamics describe the relation between the neural input to the muscle and its mechanical activation. The corresponding physiological process within the muscle is the release, diffusion, and uptake of the calcium ions that control the production of sliding forces between the actin and myosin filaments. F_0 denotes the maximal isometric force when the muscle is fully active ($a = 1$) and $\bar{L}_m = L_m/L_0$ where L_0 is the length at which the maximal force is achieved. The inclusion of the pennate angle is not necessary to describe the recti muscles that are responsible for the horizontal motion of the eye. However, it is included in the subsequent development and the implications of its inclusion as a possibility for describing the so-called rectus pulley effect [4, 10] will be elaborated upon later.

Based on the mechanical structure of a muscle as shown in Figure 1, Levine, Zajac et.al. [7, 14], have developed a dynamical model of the musculotendon actuator. Here we briefly indicate the relevant results that are utilized in the eye plant model. We begin by noting that the total muscle-tendon length L_{tm}, the muscle length L_m, and the tendon length L_t satisfy

$$L_{tm} = L_t + L_m \cos\alpha. \tag{2}$$

Since muscle maintains a constant volume when shortened or stretched, for the two dimensional model it is required that the muscle width L_w is constant, or

$$L_m \sin\alpha = L_w = \text{constant}.$$

The total force of a muscle is the sum of the passive elastic force F_{pe}, the active force F_{act} and the viscosity $B_m L_m$,

$$F_m = F_{pe} + F_{act} + B_m L_m.$$

The tendon elasticity is characterized by the differential equation

$$\dot{F}_t = K_t(F_t)\dot{L}_t$$

where $K_t = K_t(F_t)$ describes the force length relationship of the tendon. The muscle force is related to the tendon force by

$$F_t = F_m \cos\alpha.$$

By differentiating (2) and using the assumption of constant volume, it follows that

$$\dot{L}_t = \dot{L}_{tm} - \dot{L}_m / \cos\alpha$$

and so the tendon dynamics are expressed as

$$\dot{F}_t = K_t(F_t)(\dot{L}_{tm} - \dot{L}_m / \cos\alpha). \tag{3}$$

For the muscle dynamics a force balance gives

$$M_m \frac{d^2(L_m \cos\alpha)}{dt^2} = F_t - [F_{act} + F_{pe} + B_m \dot{L}_m] \cos\alpha.$$

By again using the assumption of constant muscle volume it can be shown that

$$\frac{d^2(L_m \cos\alpha)}{dt^2} = \frac{L_m}{\cos\alpha} - \frac{\dot{L}_m^2 \tan^2\alpha}{L_m \cos\alpha}.$$

Therefore, for the muscle dynamics we have

$$M_m \ddot{L}_m = F_t \cos\alpha - \cos^2\alpha [F_{act} + F_{pe} + B_m \dot{L}_m] + \frac{M_m \dot{L}_m^2 \tan^2\alpha}{L_m}. \tag{4}$$

The passive muscle (when activation is zero) behaves like a spring whose force-length characteristics are represented by F_{pe}. Recall that F_{act} denotes the purely active force and is given in (1). The effects of neural stimulation upon muscle force are brought to bear upon muscle dynamics through activation dynamics, a notion to be described in the next sectin. One could regard activation as the exogeneous inputs into the muscle-tendon complex and these inputs are manifested through the active component of the muscle model when multiplied by the force-length and force-velocity terms. Specific forms of force-length and force-velocity as well as K_t and F_{pe} will be introduced for the purpose of numerical simulation. The role of the musculotendon actuator as described by equations (3) and (4) upon the oculomotor system will now be addressed.

3 The Plant Model of the Eye

Figure 2 shows a top view of the left eyeball. Of the six extraocular muscles, the lateral and medical recti are used primarily for rotations about the z-axis, the superior and inferior recti for rotations about the y axis and the superior and inferior oblique for rotations about the x axis. Only the medial and lateral recti are shown since the investigation here is restricted to horizontal movements that correspond to rotations about the z axis.

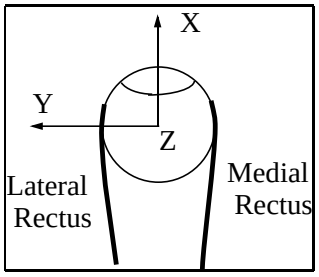

Figure 2. Top view of globe with medial and lateral recti muscles responsible for horizontal rotations about z axis.

For displacements from straight ahead that are less than 30° to 40°, the moment arms of the lateral and medial rectus are approximately equal to the radius of the eyeball. The mechanics is considerably simplified under these assumptions and the derivations presented here are restricted to these range of motions since the primary intent of this work is to model a saccadic movement.

The differential equations that describe eye movement include the musculotendon dynamics in addition to the equation of motion for the globe and the activation dynamics. One must distinguish the equations that describe the lateral rectus, (the agonist) and the medial rectus (the antagonist). All lengths, forces and parameters that pertain to the agonist will be denoted as $F_{t1}, L_{tm1}, B_{m1}, M_{m1}$, etc, while the corresponding quantities for the agonist are indicated by $F_{t2}, L_{tm2}, B_{m2}, M_{m2}$, etc. Figure 3 shows the basic arrangement of the elements of the plant, that is, the eyeball or globe and extraocular muscle. For the sake of convenience, the muscles are shown unwrapped from the globe as though they were translating it. In Figure 3, J_g is the moment of inertia of the eyeball which is acted upon by three forces: the tendon force F_t of the two muscles which constitute the agonist-antagonist pair and the force of the nonmuscular passive tissues.

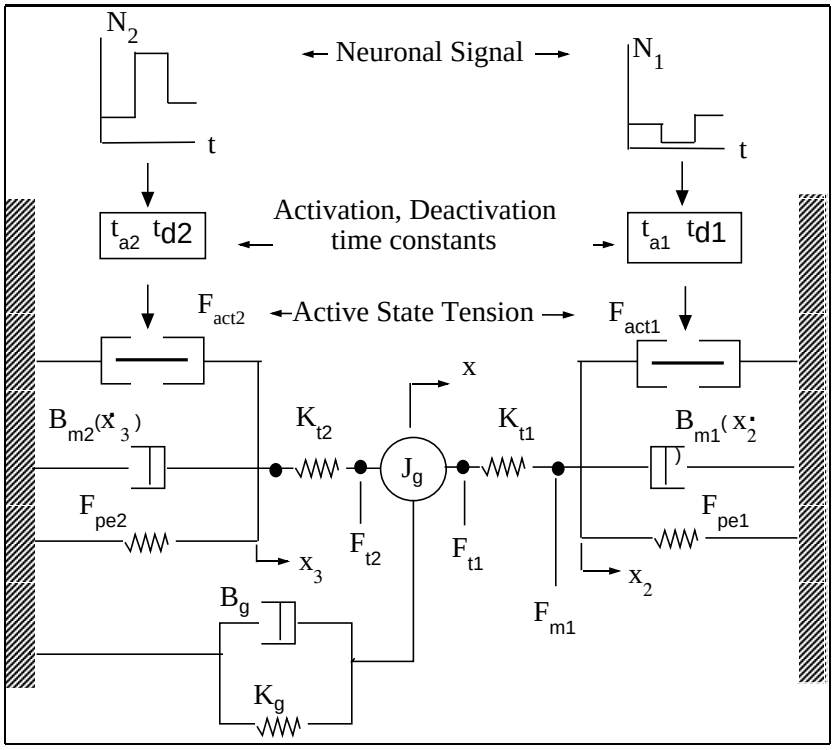

Figure 3. The organization of mechanical elements of the eye model. The agonist and antagonist elements are denoted by subscripts 1,2 respectively.

Let L_{tmp}, L_{tp} and L_{mp} denote the length of the various elements when the muscle is in the primary position that corresponds to looking straight ahead. We assume the primary lengths of the agonist and antagonist are equal. It should also be noted that the primary position does not correspond to the equilibrium lengths of these elements [6]. Introduce the change of variables

$$\begin{aligned} x_1 &= L_{tm1} - L_{tmp} \\ x_2 &= L_{m1} - L_{mp} \\ x_3 &= L_{m2} - L_{mp} \\ x_4 &= L_{tm2} - L_{tmp} \ . \end{aligned}$$

These variables have the interpretation of the change from the primary position of the tendon and active muscle nodes in the agonist and antagonist (see Figure 4). Observe that the angular displacement of the globe (in degrees) is given by

$$\theta = 57.296x/r$$

where r equals the radius of the eyeball with a value of approximately 11mm.

By Newton's second law and a balance of forces on the globe we have

$$J_g\ddot{\theta} + B_g\dot{\theta} + K_g\theta = F_{t1} - F_{t2}.$$

Since

$$r\theta = 57.296(L_{tm1} - L_{tmp}) = 57.296\ x,$$

the equation of motion can be written as

$$J\ddot{x} + B\dot{x} + Kx = F_{t1} - F_{t2} \tag{5}$$

where

$$J = 57.296J_g/r^2,\ B = 57.296B_g/r^2, K = 57.296K_g/r^2.$$

With the above change of variables and because the agonist and antagonist act in opposite directions on the globe, when pennation effects are ignored, the musculotendon dynamics may be written as

$$\dot{F}_{t1} = K_{t1}(\dot{x} - \dot{x}_2) \tag{6}$$

$$\dot{F}_{t2} = K_{t2}(\dot{x} - \dot{x}_3) \tag{7}$$

$$M_{m1}\ddot{x}_2 = K_{t1}(x_2 - x) - F_{act1} + F_{pe1} + B_{m1}\dot{x}_2 \tag{8}$$

$$M_{m2}\ddot{x}_3 = K_{t2}(x - x_3) - (F_{act2} + F_{pe2} + B_{m2}\dot{x}_3). \tag{9}$$

The inputs into the model are the neural control signals N_1, N_2. These signals are coupled with the purely active muscle force by what are known as activation dynamics. Here we adopt the approach of [1] and assume that these processes can be depicted as a low pass filter with activation-deactivation time constants $t_{1a}, t_{1d}, t_{2a}, t_{2d}$. (For a more complete discussion and more sophisticated models of activation dynamics see [14].) In particular we take

$$\tau_1\dot{a_1} - a_1 = N_1 \tag{10}$$

$$\tau_2\dot{a_2} - a_2 = N_2 \tag{11}$$

where

$$\tau_1 = t_{1a}[u(t) - u(t - T_1)] + t_{1d}u(t - T_1)$$

$$\tau_2 = t_{2a}[u(t) - u(t - T_2)] + t_{2d}u(t - T_2).$$

Here $u(t)$ is the Heaviside function and T_1, T_2 correspond to the duration of the agonist and antagonist pulse.

The state variables that will be utilized are the three positions x, x_2, x_3 that correspond to the rotation of the eye, the length of the active muscle, the eye velocity $\dot{x}$, the two tendon forces F_{t1}, F_{t2} and the activation variables $a_1(t), a_2(t)$. Define

$$z_1 = x = \text{ position of eye}$$
$$z_2 = \dot{x} = \text{eye velocity}$$
$$z_3 = x_2 = \text{ position of agonist muscle node}$$
$$z_4 = \dot{x}_2 = \text{ velocity of agonist muscle contaction}$$
$$z_5 = x_3 = \text{ position of antagonist muscle node}$$
$$z_6 = \dot{x}_3 = \text{ velocity of antagonist muscle contaction}$$
$$z_7 = F_{t1} = \text{ agonist tendon tension}$$
$$z_8 = F_{t2} = \text{ antagonist tendon tension}$$
$$z_9 = a_1 = \text{ agonist activation}$$
$$z_{10} = a_2 = \text{ antagonist activation .}$$

By using equations (6)-(11) one obtains the state equations,

$$\dot{z_1} = z_2$$
$$\dot{z_2} = (-(z_7 + z_8) - Bz_2 - Kz_1)/J$$
$$\dot{z_3} = z_4$$
$$\dot{z_4} = (-z_7 - z_9 F_{l1}(z_3) F_{v1}(z_4) + F_{pe1}(z_3) + B_{m1} z_4)/M_{m1}$$
$$\dot{z_5} = z_6$$
$$\dot{z_6} = (z_8 - z_{10} F_{l2}(z_5) F_{l2}(z_6) + F_{pe2}(z_5) + B_{m2} z_6)/M_{m2}$$
$$\dot{z_7} = K_t(z_7)(z_1 - z_4)$$
$$\dot{z_8} = K_t(z_8)(z_1 - z_6)$$
$$\dot{z_9} = (N_1 - z_9)/\tau_1.$$
$$\dot{z_{10}} = (N_2 - z_{10})/\tau_2$$

It should be noted that the effects of nervous activity, that is the controls, N_1, N_2, are modulated by the activation dynamics (the last two differential equations). These effects contribute to active muscle force by multiplication with the force-length and force-velocity terms in the differential equations for z_4 and z_6.

There are a variety of models that have been suggested to account for the tendon stiffness K_t, the passive muscle F_{pe}, the tension-length relation

F_l,and the tension velocity relation F_v. All studies of the eye have assumed that the series elasticity parameter K_t to be a constant. Levine, Zajac et al. [7, 14], have utilized a force length relation of the form

$$\dot{F}_t = K_t(F_t) = \begin{cases} (k_{te}F_t + k_{tl})\dot{L}_t & 0 < F_t < F_{tc} \\ k_t\dot{L}_t & F_t \geq F_{tc} \end{cases}$$

where k_{te}, k_{tl} and k_t are experimentally determined elasticity parameters and F_{tc} denotes the transition point from an exponential to linear spring. In this same work the passive muscle relation may be expressed in terms of our current variables (for say, the agonist) as

$$F_{pe}(z_3) = \begin{cases} \frac{k_{ml}}{k_{me}}(e^{k_{me}z_3} - 1) & z_3 < l_{mc} \\ k_m z_3 + f_{mc} & z_3 > l_{mc} \end{cases}$$

where k_{ml}, k_{me} and k_m are spring constants and f_{mc} and l_{mc} are respectively the force and length at which the passive muscle elasticity changes from exponential to linear. Results of [2] suggest that

$$F_{pei} = az + \sqrt{bz^2 + c}, \quad i = 1, 2$$

where $z = z_2$ or z_3. Values are provided for the constants a, b, c and so this representation of the passive elasticity is employed in the simulations. The force velocity relation that is employed in the subsequent simulations is simply Hill's equation. In particular we utilize the results of [1] and adopt the force-velocity relation $F_v(\dot{L}_m) = F_0 - B\dot{L}_m$ where for the agonist

$$B = \frac{1.25}{1000 + z_4}$$

and for the antagonist $B = 3/1000$. For the force length curve F_l is taken from [2] and amounts to assuming that F_l depends linearly on the displacement from its optimal length L_0. Due to the lack of data for the extraocular muscles, the simulations that are subsequently presented assume a constant tendon elasticity. However, certain qualitative insights into system performance can be gained from the consideration of the more general models of tendon and passive elasticity and force-length and force-velocity relations. Results of these investigations will be presented in future work.

4 Simulations

There are a variety of sources that provide experimental values for the parameters that appear in the model that has been presented in this paper. The data used here is taken from [3, 6]. The parameter values are $K_g =$ .5 gm/°, $J_g = 4.3(10^{-5})$ gm/s^2, $B_g =$ .06 gm/°. There is not universal

agreement on the relationship between motoneuronal firing and saccade duration and amplitude. The following expressions used to represent the nervous activity are obtained from electromyographic studies and have been converted into units of force [6]: $N_{1-pulse} = 55 + 11\theta$ gm, $N_{1-step} = 20.6 + 2.35\theta$ gm, $N_{2-pulse} = .5$ gm, $N_{2-step} = 20.6 - .74\theta$ gm, $t_{1a} = 11.7 - .2\theta$ ms, $t_{1d} = .2$ ms, $t_{2a} = 2.4$ ms, $t_{2d} = 1.9$ ms, $T_1 = 10 + \theta$ ms, $T_2 = T_1 + 6$ ms. In the above expression for the pulse amplitude and duration, θ denotes the magnitude of the saccade.

The system of 10 coupled nonlinear differential equations for the state variables $z_1, \cdots, z_{10}$ is solved numerically when the control input corresponds to agonist activity that begins at time $t = 1$. All of the initial conditions are zero except those that reflect the fact that the recti muscles are not at rest in the primary position. This condition is described by

$$z_3(0) = -z_5(0) = 5.6^\circ$$
$$z_9(0) = z_{10}(0) = 20.6\text{gm}.$$

Simulations of 10° are presented in Figure 5 which illustrates the phenomena of glissadic undershoot and overshoot. If the pulse is too long in relation to the size of the step, an overshoot with glissadic return is seen. If the pulse width if too small, undershoot occurs. The numerical calculations supported the physiological notion that eye motion is quite sensitive to pulse duration as opposed to the magnitude of the signal. When the values for pulse and step suggested in the literature were utilized, the model produced good agreement with the 10° saccade.

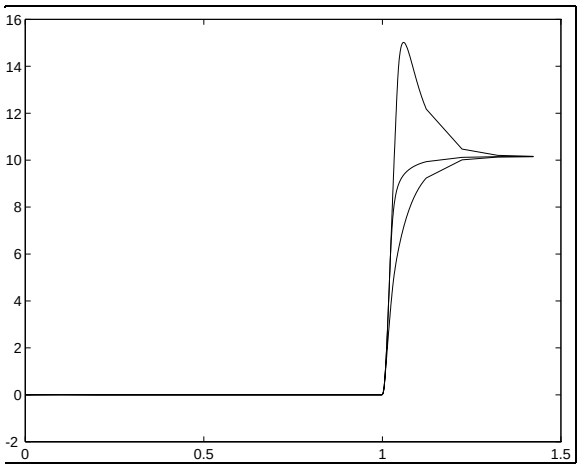

Figure 5. An illustration of glissadic undershoot and overshoot.

The phase portraits for 10° and 30° saccades are illustrated in Figure 6. When compared with the experimental results of [11], the velocities predicted by the model provide good agreement.

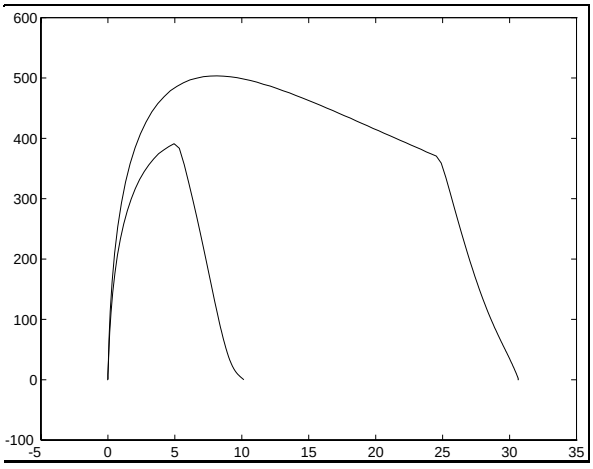

Figure 6. Phase portraits for 10° and 30° saccades.

Figure 7 illustrates a possible consequence of omitting the passive elasticity in the muscle model. For instance in [1] and several other studies, the parallel elasticity is lumped with the passive effects of nonmuscular connective or suspensory tissue. This has tends to overestimate the damping in the system. When the damping in the globe is reduced and yet not accounted for in the passive component of the muscle, oscillations as illustrated in Figure 7 may occur.

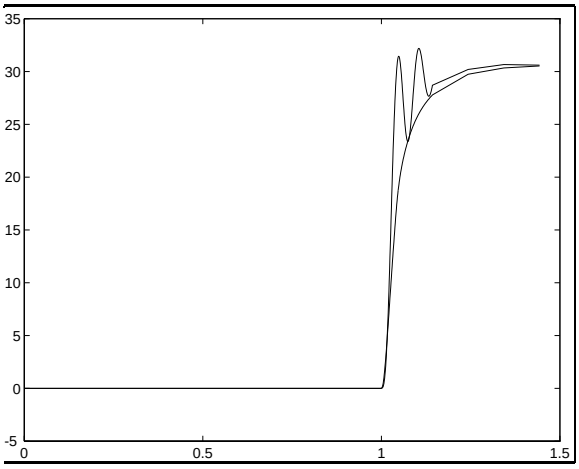

Figure 7. Oscillations may result if passive effects are not retained in the muscle model.

Several issues could be addressed to improve upon the model that has been presented here. Data suggests that the tendon elasticity is parametrically modulated by the active state tension. Consequently, the tendon stiffness K_t should be a function of nervous activity and active force in addition to the tension in the tendon. The use of a linear approximation to the passive elasticity and the force-length curve undoubtedly introduces some error. The role of feedback and the stretch reflex needs to be explored within the context of ocular control and movement.

References

[1] M. R. Clark and L. Stark. Control of human eye movements I. Modeling of extraocular muscles; II. A model for the extraocular plant mechanism; III. Dynamic characteristics of the eye tracking mechanism, *Math. Biosc.*, **20** (1974), 191-265.

[2] C. Collins. Orbital mechanics, in *The Control of Eye Movements*, (P. Bach-y-Rita and C. Collins, eds.). New York: Academic Press, 1971, 283.

[3] G. Cook and L. Stark. The human eye movement mechanism: Experiments, modeling and model testing, *Arch. Opthalmol.* , **79** (1968), 428-436.

[4] J. Demer and J. M. Miller. Surgical implications of the rectus extraocular muscle pulleys, *J. Pediatr. Ophthalmol Strabismus*, **33** (1996), 208-218.

[5] R. Descartes. *Treatise of Man*, (translation by T.S. Hall), Cambridge, MA: Harvard Univ. Press, 1972.

[6] J. D. Enderle. The fast eye movement control system. In *Biomedical Engineering Handbook*, (J.D. Bronsino, ed.), 2473-2493. CRC Press, 1995.

[7] J. H. He, W.S. Levine, G. E. Loeb. Feedback gains for correcting small perturbations to standing posture, *IEEE Transaction on Automatic Control*, **36** (1991), 322-332.

[8] S. L. Lehman. Simulation of linear and nonlinear eye movement models: sensitivity analysis and enumeration studies of time optimal control, *Cybernet. Info. Sci.*, **4** (1980), 21-43.

[9] S. Y. Lu and C. F. Martin. Dynamics of ocular motion, *Computation and Control IV*, Progress in Systems and Control, **20**, 229-245, (K Bowers, J. Lund, eds.). Boston: Birkhauser,1995.

[10] J. M. Miller and J. Demer. Effect of transposition surgery on rectus muscle paths by magnetic resonance imaging, *Opthamology*, **100** (1993), 475-457.

[11] D. A. Robinson. The mechanics of human saccadic eye movement, *J. of Physiol.*, **174** (1964), 245-264.

[12] G. Westheimer. Mechanisms of saccadic eye movements, *Amer. Med. Assoc. Arch. Opthamol.*, **52** (1954), 710-724.

[13] L. R. Young and L. Stark. Variable feedback experiments testing a sampled data model for eye tracking movements, *IEEE Trans. on Human Factors in Electronics*, **4** (1963), 38-51.

[14] F. Zajac. Muscle and tendon: Properties, models, scaling and application to biomechanics and motor control, *Critical reviews in Biomedical Engineering*, **17** (1989), 359-441.

[15] F. E. Zajac and R.G. Wicke. Dependence of jumping performance on muscle properties when humans use only calf muscles for propulsion, *J. Biomechancs*, **17** (1984), 513-523.

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