Construction of Simple Dynamic Models of the Gamma-loop Mechanism

Dávid Csercsik

Research Report SCL-001/2006

Contents

Construction of Simple Dynamic Models of the Gamma-loop Mechanism

Dávid Csercsik[1]
E-mail: csercsik@scl.sztaki.hu

Research Report SCL-001/2006

[1]Process Control Research Group,
Systems and Control Research Laboratory, Computer and Automation Institute HAS
H-1518 Budapest, P.O. Box 63, Hungary

Human locomotion control research strongly needs the understanding of the various feedback mechanisms, appearing in the human neuromuscular systems. For describing the effect of these feedback mechanisms, the modeling of the system components, including the affected limb or joint is needed. Two models of the gamma-loop mechanism based on different muscle spindle models are proposed in this paper, for the investigation of this nonlinear feedback mechanism in human locomotion. The first intuitive model is based only on the dynamical properties of the muscle spindles, while the second physical model is based on a simple spindle structure composed of a contractile and an elastic element. The developed models are transformed into a standard nonlinear state-space model form by substituting the algebraic equations into the differential ones. This form enables to apply standard control theoretic methods for dynamic analysis of the partially controlled system. The effect of the gamma-loop mechanism is studied on a simple, elbow-like nonlinear limb model, described in [2]. The first model is able to describe the stretch reflex, and both model can describe the smoothing of movement, the appearing muscle tone, and the alpha-gamma coactivation. Both models can be extended easily with a polysinaptic feedback.

1 Introduction

The importance of understanding the way in which sensorimotor feedback influences motor control has increased significantly in the past decades. Several publications studied the role of sensory feedback in motor control [6], [7], [10]. Also several studies deal with the properties of the receptors, which provide the sensory information for this feedback [8], [14], [5]. In fact, the integration of reflexes at the lower levels (spinal chord) of the central nervous system (CNS) to sensorimotor control models is not prevalent in literature, only few studies are published on this field [11], [9].

The aim of this work is to build simple models of the gamma loop mechanism to provide a principle for future studies on human locomotion control. In these future researches we will investigate how the gamma loop mechanism can be involved to control (in the case of a simple nonlinear limb model), which control models involving the gamma loop mechanism provide the best performance (in the case of various tasks) and which models are the closest to the physiology of human motion control. To this aim, we investigate two simple models of the muscle spindle. In both cases we investigate the muscle spindle, only as a part of the gamma reflex loop, and not as a source of information about the state of the limb. In fact, we suppose that the variables, who's derivatives appear in the differential equations of the model (state space variables), are fully available for a feedback controller.

2 A simple nonlinear limb model

Figure 1 shows the simplified limb model:

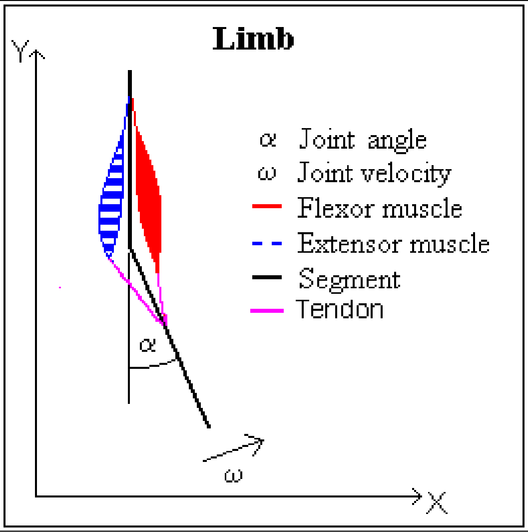

Figure 1: The simple limb model

For the description of the nonlinear dynamic properties of the limb and the working muscles, we use a simplified version of the model described in [2]. In this simplified model we neglect the dynamics of the tendon, and assume constant tendon lengths. This can be done in the case of an elbow-like structure without validity problems. In this case the equations of the model are the following:

$$
\begin{aligned}
\frac{dq_1}{dt} &= -\left(\frac{1}{\tau_{act}}(\beta + [1-\beta]u^f(t))\right) q_1 + \frac{1}{\tau_{act}}u^f(t)\\
\frac{dq_2}{dt} &= -\left(\frac{1}{\tau_{act}}(\beta + [1-\beta]u^e(t))\right) q_2 + \frac{1}{\tau_{act}}u^e(t)\\
\frac{d\alpha}{dt} &= \omega\\
\frac{d\omega}{dt} &= \frac{1}{\Theta + ml_{COM}^2}(M(q_1, q_2, \alpha, \omega) + ml_{COM}cos(\alpha + \xi)g_y)
\end{aligned}
\tag{1}
$$

where $q_1(t)$ denotes the activation state of the flexor muscle, and $q_2(t)$ denotes the activation state of the extensor muscle, τ_{act} [s] is the activation time, showing how quick the muscle reacts on the external activation signal coming from the nerval system. β is a constant, describing the correlation between the decrease of the activation state and the external activation signal. If $\beta = 1$ then the external activation signal does not affect the decrease of the activation state, if $\beta = 0$ then it strongly affects it. u^f and u^e denotes the activation signals of the muscles. α [rad] denote the external joint angle. ξ [rad] is the angle between the global coordinate-system's x axis, and the not-moving upper segment of the limb (in our model ξ is always equal to $-\pi/2$), ω [rad/s] is the angle velocity, Θ $[kgm^2]$ is the moment of inertia defined to the mass-center point of the bone, m [kg] is the mass of the moving limb part, l_{COM} [m] is the distance between the moving limb part's center of mass point and the joint axis. M [Nm] is the resulting joint torque, and $g = [g_x, g_y]$ $[m/s^2]$ is the vector of gravitational acceleration.

With the notation x_i for the state-space variables ($x_1 = q_1$, $x_2 = q_2$, $x_3 = \alpha$, $x_4 = \omega$), the equations are as follows:

$$
\begin{aligned}
\frac{dx_1}{dt} &= -\left(\frac{1}{\tau_{act}}(\beta + [1-\beta]u^f(t))\right) x_1 + \frac{1}{\tau_{act}}u^f(t)\\
\frac{dx_2}{dt} &= -\left(\frac{1}{\tau_{act}}(\beta + [1-\beta]u^e(t))\right) x_2 + \frac{1}{\tau_{act}}u^e(t)\\
\frac{dx_3}{dt} &= x_4\\
\frac{dx_4}{dt} &= \frac{1}{\Theta + ml_{COM}^2}(M(x_1, x_2, x_3, x_4) + ml_{COM}cos(x_3 + \xi)g_y)
\end{aligned}
\tag{2}
$$

The parametrization of the model is the same as in [2] except the maximal active force of the muscles which is in this case 500 N, and the force-length characteristics (the function is the same, but the ideal muscle lengths are now defined as lengths belonging to $\alpha = \pi/6$). These parameters were changed to these more realistic values.

We have to note, that the linearized open-loop model can be instable or at the edge of stability, depending on the steady state-point, the muscle characteristics, and the values of the model parameters (for example, the optimal length of the muscles).

3 The gamma loop

3.1 Muscle spindles

To understand the functioning of the gamma-loop mechanism, we have to know some facts about one of the main components of the system: the muscle spindles.

Muscle spindles are found within the fleshy portions of muscles, embedded in so-called extrafusal muscle fibers, aligned parallel to them (unlike Golgi tendon organs, which are oriented in series). The ends of the fusiform spindles, which vary in length from 5 to 10 mm, attach to the intramuscular connective tissue. The spindles are composed of 3-10 intrafusal muscle fibers (of which there are two types, nuclear bag fibers (8-10 mm long) and nuclear chain fibers) and the axons of sensory neurons. Intrafusal muscle fibers are essentially a collection of miniature skeletal muscle fibers enclosed in a connective tissue capsule.

The muscle spindle has both sensory and motor components. As a sensory receptor, the muscle spindle has an afferent supply of different afferents, over which the action potentials are transmitted to the Central Nervous System (CNS), providing the sensory component of the structure via stretch-sensitive excitatory ion-channels of the axons. There are two subtypes of the nuclear bag fiber, which have different elastic properties, and correspondingly, different functions. The *dynamic bag fiber* is sensitive mainly to changes in muscle length. The other, the *static bag fiber*, signals only a change in muscle length [4].

The efferent input to the intrafusal fibers of the muscle spindle are the endings of gamma (γ) and beta (β) motoneurons. In general, skeletal muscle fibers are innervated by three groups of motoneurons, which can be distinguished by size, and by the type of the muscle that they innervate. The alpha (α) motoneurons are the largest and innervate the extrafusal muscle fibers. Gamma (γ) motoneurons are the smallest, and connect exclusively to intrafusal muscle fibers. Beta (β) motoneurons are intermediate in size, and innervate both type of muscle fibers [3].

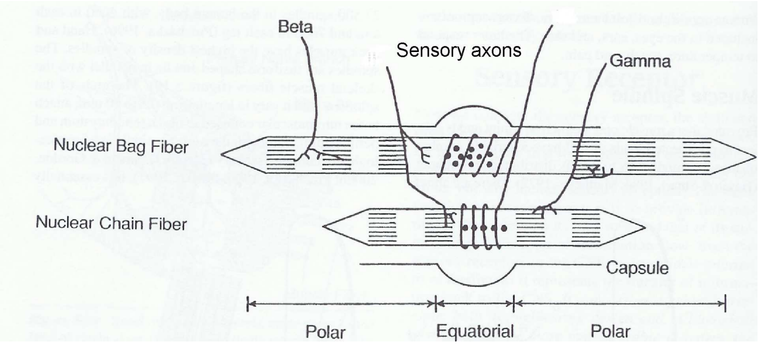

Figure 2: Muscle spindles (figure taken from [3])

The activation of the gamma motoneurons causes the contraction of the equatorial region, and the appearing stretch heightens the sensitivity of the muscle spindles [3].

3.2 The servomechanical gamma-loop

The length of the muscle spindles on the one hand serve as a sensory input for the central nervous system, providing information about the length of the muscle, and so the state of the joints too.

On the other hand they serve as a component for the servomechanical γ-loop. Inside the muscle spindles are muscle fibers, thinner and shorter than the working muscle fibers, which provide the majority of the force, doing most of the muscle work. These muscle fibers inside the muscle spindles are innervated by the γ-nerves originating from the γ-neurons in the spinal marrow. In general case the γ-neurons regulate the length of the muscle spindles to match the actual length and stretch state of the working muscle.

The servomechanical gamma loop effect:

If the γ-neurons (denoted with green in figure 3) are stimulated by the impulses from the descending tracts in the spinal marrow (magenta), the muscle fibers inside the muscle spindle (yellow) contract, providing difference between the stretch state of the working muscle and the muscle spindles, which is instantly detected by the receptors of the muscle spindle, innervated by neurons transferring the signal back to the spinal marrow (denoted with green). Consequently the α-motoneurons in the spinal marrow (red) stimulate the working muscle to contract. Because the impulses from the descending tracts through the γ-neurons first reach the muscle spindle, then return to the spinal marrow, and then reach the working muscle, we call this loop-like path the gamma loop. The consequence is that the central nervous system does not need nerves descending to all of the muscle fibers, furthermore the impulses of the central nervous system can affect the activation of the muscle in a way that the final effect depends on the actual state (length) of the muscle. [12]

Figure 3: The servomechanical γ-loop

Polysynaptic Control:

The gamma-afferents connect not only to the spinal (alpha-efferent) motoneuron innervating the agonist muscle (here: the same muscle, in which the spindle is located), but through an inhibitory synapsis, they connect also to the motoneuron of the antagonist muscle, and block it's functioning [13].

Alpha-gamma coactivation:

Consider the situation in which a muscle is contracting actively against a load. Because a muscle spindle attached parallel to the adjacent extrafusal fibers, one might erroneously infer that its overall length is determined (approximately only) by the length of the surrounding muscle; when the muscle contracts, the spindle shortens, and the activity of the afferent (sensory) neurons decrease. This is not so; if the intrafusal fibers remained passive during extrafusal muscle fiber contraction, the shortening of the muscle would relax the equatorial region of the intrafusal fibers, and the afferent neurons would cease firing. This slack inactive muscle would be use less for reporting muscle dynamics. In reality, the spindle retains its sensitivity, because when the brain signals the α-motoneuron to initiate muscle contraction, it sends parallel impulses to the gamma neurons to cause the intrafusal fibers to contract. Therefore, when the extrafusal muscle fibers shorten, the intrafusal fibers also shorten, because their gamma motor neurons are activated at the same time, and the equatorial regions of the intrafusal fibers remain under nearly constant tension. This phenomenon is called *alpha-gamma coactivation* [4].

4 Modeling the gamma loop

In general case, the signal flow diagram of the system including the gamma loop mechanism can be seen in the following figure (4).

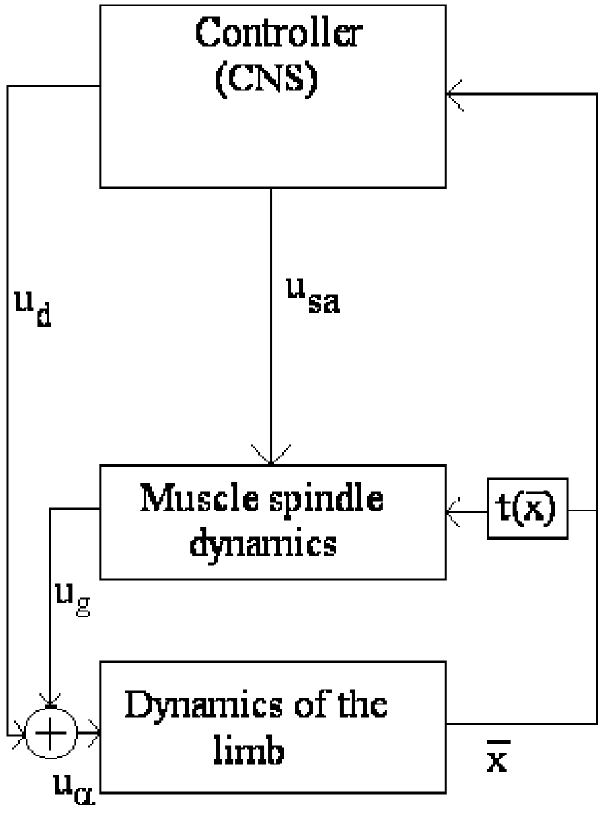

Figure 4: The Signal flow diagram of the gamma loop mechanism and the controller

In figure 4 u_d means the direct activation signal of the extrafusal (working) muscles, originating from the CNS, u_s denotes the activation signal of the intrafusal muscle fibers, u_g denotes the activation signal of the extrafusal muscles, originating from the gamma-loop mechanism, u_α denotes the total (summated) activation signal of the extrafusal muscle fibers, $\overline{x}$ denotes the state-space variables of the simple nonlinear limb model. The state-space variables of the simple nonlinear limb model affect the muscle spindle dynamics via $t(\overline{x})$ (form the state-space variables, the lengths of the (extrafusal) muscles can be computed, which necessary appears in the input of the muscle spindle dynamics).

4.1 The first simplified model of the Gamma-loop

Simplifying assumptions

- In the model, we are interested only in the motor component of the system. In our case the a controller, which will be developed in the future, will be modeling the adequate functions of the CNS.

- The one muscle spindle in the model refers to the summation of the spindles.
- In the case of the spindles, only gamma innervation is modeled.
- We take only the static bag fibers into account: The receptors of the muscle spindles can sense only the length difference between the muscle fibers inside the muscle spindles (l_s), and the surrounding muscle fibers, of which length are commensurable to the working muscle length (l_{CE}). In this case, this is also the simple model of the alpha-gamma coactivation. In our model we suppose the normalized length of the surrounding muscle fibers always equal to the normalized length of the working muscle (l_{CE}). Therefore the length difference detected by the muscle spindles is $l_{CE} - l_s$. However, we have to note, that the first model is sensitive for quick changes of muscle length, as we will see it later.
- The activation signal generated by the spinal neurons (denoted with u_g) is sigmoid in the measured difference. This is shown in figure 5.
- At first we do not model polysynaptic control.

4.1.1 Model equations

In this case, we can extend our model with two more state-space variables: With the normalized lengths of the muscle fibers of the spindles (l_s^f in the case of the flexor muscle, and l_s^e in the case of the extensor muscle).

If we suppose, that the normal length of the muscle spindles (and the surrounding muscle fibers) is always equal to the working muscle, we can describe the behavior of the new variables with the following equations:

$$\begin{aligned} \frac{dl_s^f}{dt} &= c_P(l_{CE}^f - l_s^f) - c_G u_s^f(t) \\ \frac{dl_s^e}{dt} &= c_P(l_{CE}^e - l_s^e) - c_G u_s^e(t) \end{aligned} \tag{3}$$

where c_P [1/s] is a constant showing how quick the length of the muscle spindles (and the muscle fibers inside the muscle spindles) follows the length of the working muscle (l_{CE}^f, l_{CE}^e), c_G is a constant showing how sensitive the muscle spindle is to the external activation signal of the descending tracts, and $u_s(t)$ is the activation signal of the descending tracts acting on the the muscle fibers inside the muscle spindles ($u_s^f(t)$ in the case of flexor, and $u_s^f(e)$ in the case of extensor muscle).

If we make these assumptions, we have to modify (expand) the model described in [2].

The complete activation signal of the muscles in this case can be computed on the one hand from the direct activation signal u_d, on the other hand the activation signal originating from the gamma loop mechanism (u_g). u_g can be computed from the actual difference between the lengths of l_{CE} and l_s (both as normalized value).

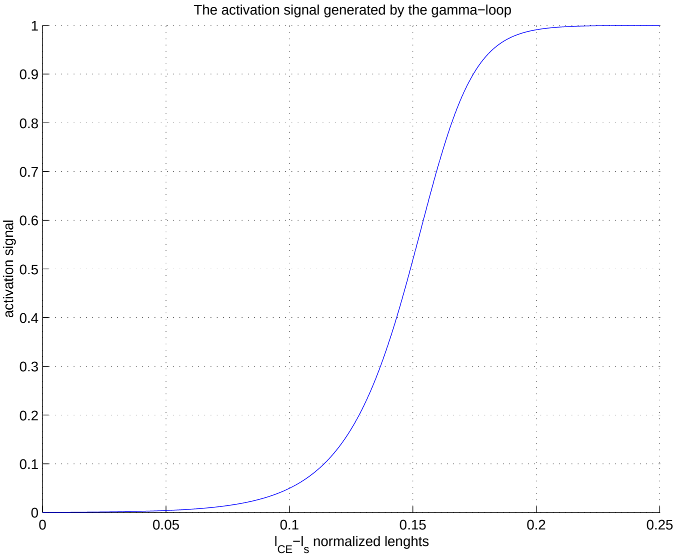

Figure 5: The activation signal generated by the gamma loop mechanism

We can rearrange the first two state-space equations in [2] as follows:

$$\begin{aligned}\frac{dx_1}{dt} &= -\left(\frac{1}{\tau_{act}}(\beta + (1-\beta)u^f(t))\right)x_1 + \frac{1}{\tau_{act}}u^f(t) \\ &= -\frac{1}{\tau_{act}}\beta x_1 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_1 + \frac{1}{\tau_{act}}\right)u^f(t) \qquad (4)\end{aligned}$$

and

$$\begin{aligned}\frac{dx_2}{dt} &= -\left(\frac{1}{\tau_{act}}(\beta + (1-\beta)u^e(t))\right)x_2 + \frac{1}{\tau_{act}}u^e(t) \\ &= -\frac{1}{\tau_{act}}\beta x_2 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_2 + \frac{1}{\tau_{act}}\right)u^e(t) \qquad (5)\end{aligned}$$

where u_1 and u_2 are replaced by u^f and u^e referring to the fact that the total flexor and extensor activation signal is composed on the one hand from the direct activation signal and on the other hand from the effect of the gamma-loop.

With the notation of x_1,x_2,...,x_6 for state space variables the equations are as follows:

$$
\begin{aligned}
&\dot{x_1} = -\tfrac{1}{\tau_{act}}\beta x_1 + \left(-\tfrac{1}{\tau_{act}}(1-\beta)x_1 + \tfrac{1}{\tau_{act}}\right) u^f(t) \\
&\dot{x_2} = -\tfrac{1}{\tau_{act}}\beta x_2 + \left(-\tfrac{1}{\tau_{act}}(1-\beta)x_2 + \tfrac{1}{\tau_{act}}\right) u^e(t) \\
&\dot{x_3} = x_4 \\
&\dot{x_4} = \tfrac{1}{\Theta + ml_{COM}^2}(M(x_1, x_2, x_3, x_4) + ml_{COM}cos(x_3 + \xi)g_y) \\
&\dot{x_5} = c_P(l_{CE}^f(x_3) - x_5) - c_G u_s^f \\
&\dot{x_6} = c_P(l_{CE}^e(x_3) - x_6) - c_G u_s^e
\end{aligned}
\tag{6}
$$

where $u^f(t)$ is the total activation signal of the flexor muscle, $u^e(t)$ is the total activation signal of the extensor muscle, u_s^f is the activation signal of the flexor muscle's spindle and u_s^e is the activation signal of the extensor muscle's spindle.

Let us define the following notation:
Let $u_1 \in [0,1]$ be the direct activation signal of the flexor muscle (u_d^f) (not originating from the gamma loop mechanism), $u_2 \in [0,1]$ the direct activation signal of the extensor muscle (u_d^e), $u_3 \in [0,1]$ the activation signal of the flexor muscle's spindle (u_s^f), $u_4 \in [0,1]$ the activation signal of the extensor muscle's spindle (u_s^e).

With this notations u_g^f can be computed as:

$$u_g^f = f_{gas}^f(x_3, x_5) \doteq (sigm2[(l_{CE}^f(x_3) - x_5) - 0.11])^6 \tag{7}$$

where $sigm2(x)$ stands for the function $\frac{1}{1+e^{-100x}}$. This function is depicted in figure 5.

$$u^f = u_g^f + u_d^f = u_g^f + u_1 \tag{8}$$

and

$$u_g^e = f_{gas}^e(x_3, x_5) \doteq (sigm2[(l_{CE}^e(x_3) - x_6) - 0.11])^6 \tag{9}$$

$$u^e = u_g^e + u_d^e = u_g^e + u_2 \tag{10}$$

($l_{CE}^f(x_3)$ denotes the flexor muscle's length, as a function of the joint angle ($l_{CE}^e(x_3)$ denotes the extensor's length).

We can substitute these expressions in equation (6), to get the following form:

$$
\begin{aligned}
\dot{x_1} &= -\frac{1}{\tau_{act}}\beta x_1 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_1 + \frac{1}{\tau_{act}}\right)(f_{gas}^f(x_3, x_5) + u_1) \\
\dot{x_2} &= -\frac{1}{\tau_{act}}\beta x_2 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_2 + \frac{1}{\tau_{act}}\right)(f_{gas}^e(x_3, x_6) + u_2)
\end{aligned}
$$

$$
\begin{aligned}
\dot{x_3} &= x_4 \\
\dot{x_4} &= \frac{1}{\Theta + ml_{COM}^2}(M(x_1,x_2,x_3,x_4) + ml_{COM}cos(x_3+\xi)g_y) \\
\dot{x_5} &= c_P(l_{CE}^f(x_3) - x_5) - c_G u_3 \\
\dot{x_6} &= c_P(l_{CE}^e(x_3) - x_6) - c_G u_4
\end{aligned} \tag{11}
$$

If we rearrange the equation (11) we can get the following general form of input-affine systems:

$$
\begin{aligned}
\dot{x} &= f(x) + \textstyle\sum_{i=1}^m g_i(x)u_i(t) \\
y &= h(x)
\end{aligned} \tag{12}
$$

where:

$$
f(x) = \begin{pmatrix}
-\frac{1}{\tau_{act}}\beta x_1 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_1 + \frac{1}{\tau_{act}}\right)(f_{gas}^f(x_3,x_5)) \\
-\frac{1}{\tau_{act}}\beta x_2 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_2 + \frac{1}{\tau_{act}}\right)(f_{gas}^e(x_3,x_6)) \\
x_4 \\
\frac{1}{\Theta+ml_{com}^2}(M(x_1,x_2,x_3,x_4) + ml_{com}cos(x_3)g_y \\
c_P(l_{CE}^f(x_3) - x_5) \\
c_P(l_{CE}^e(x_3) - x_6)
\end{pmatrix}
$$

$$
g_1(x) = \begin{pmatrix}
-\frac{1}{\tau_{act}}(1-\beta)x_1 + \frac{1}{\tau_{act}} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0
\end{pmatrix}
\qquad
g_2(x) = \begin{pmatrix}
0 \\ -\frac{1}{\tau_{act}}(1-\beta)x_2 + \frac{1}{\tau_{act}} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0
\end{pmatrix}
$$

$$g_3(x) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ -c_G \\ 0 \end{pmatrix} \qquad g_4(x) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ -c_G \end{pmatrix}$$

We have to note, that the effect of the gamma-loop in the phase of model construction was treated as an input, but in this form it appears as a part of f, because here, we describe the complete system.

4.1.2 Model verification

At first, we analyze the behavior of the model without any controller, only with the feedback provided only by the gamma-loop mechanism as detailed above.

First simulation

At first we expect the gamma-loop mechanism to provide a more smooth movement of the limb, because it acts as a slow-down mechanism at big limb accelerations, which cause quick changes in muscle length. We suppose a situation, when the limb descends from $\alpha = \pi/2$ to $\alpha = 0$. In the following figures, we can see the simulation results in the case, if $u_d^f = u_d^e = u_s^f = u_s^e = 0$, and there isn't any feedback to the system ($u^f = u^e = 0$).

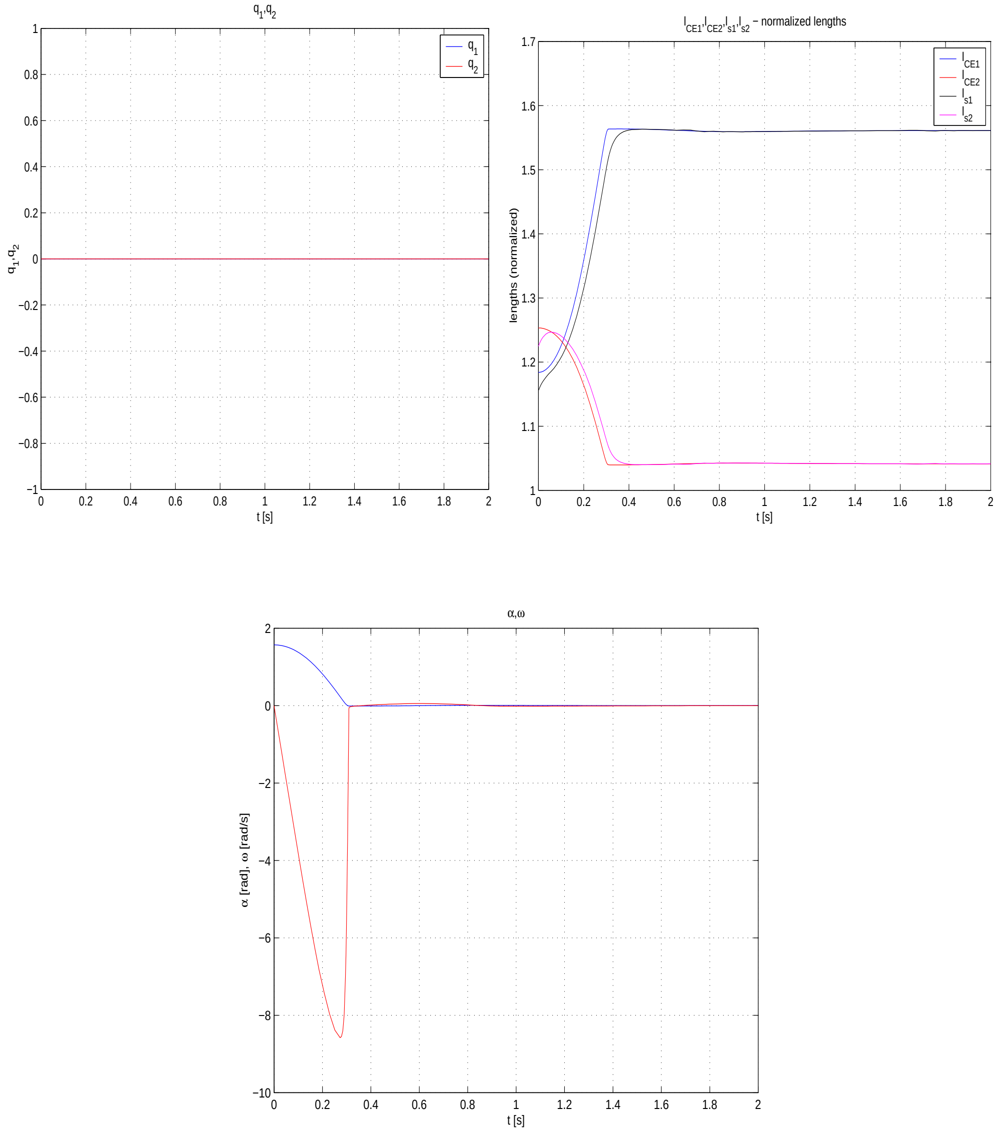

Figure 6: Muscle activation states, and muscle lengths, α and ω without gamma-loop feedback

The oscillations around 0 rad are caused by the passive forces of the muscles, and by the the forces of ligaments.

Second simulation

In the following figures, we can see the simulation results of the system with the passive gamma-loop feedback, in the case, if $u_d^f = u_d^e = u_s^f = u_s^e = 0$.

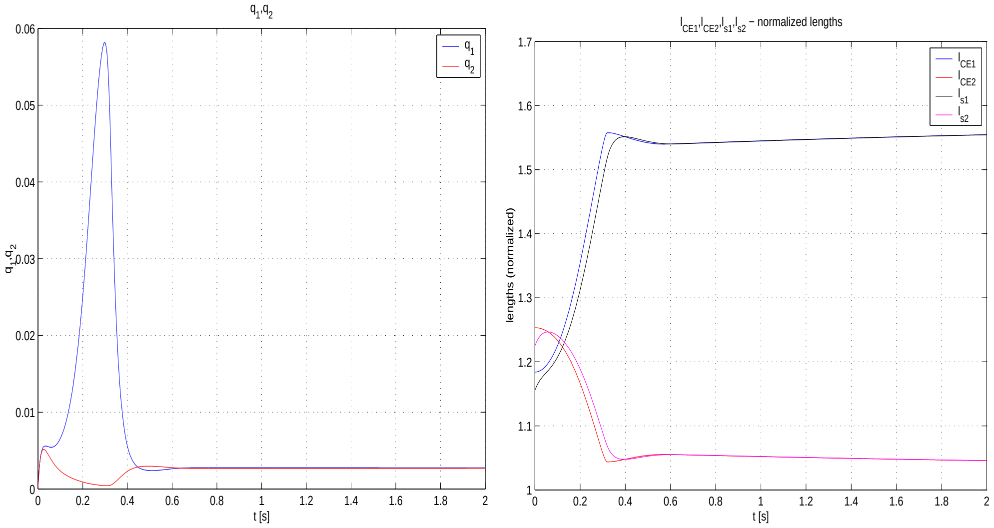

Figure 7: Muscle activation states, and muscle lengths

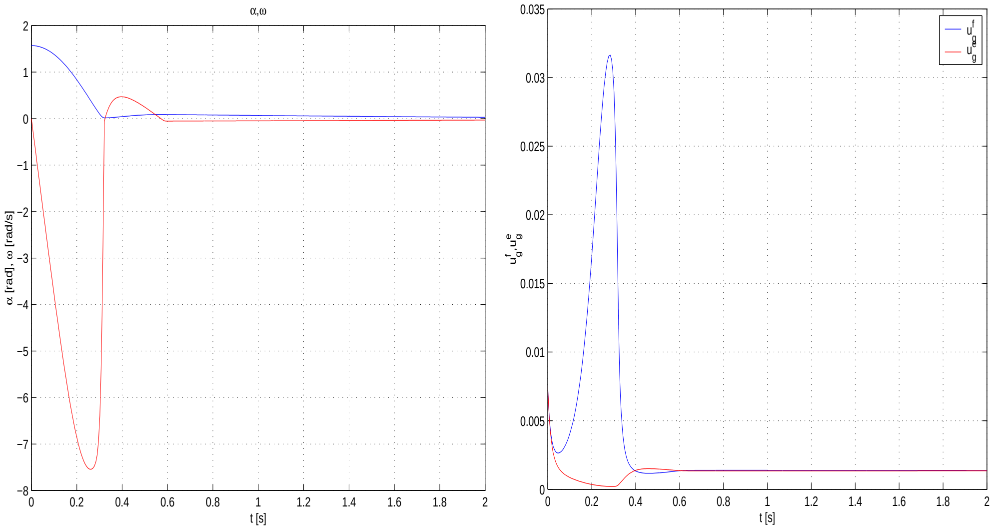

Figure 8: α, ω, u_g^f and u_g^e with passive gamma-loop feedback

We can see in the figures in the significant values of ω, that the movement became a bit smoother, and the descending of the limb takes a bit more time.

Third simulation

In the following figures, we can see the simulation results of the system with the active gamma-loop feedback, in the case, if $u_d^f = u_d^e = 0$, $u_s^f = u_s^e = 0.2$. In this case the sensitivity of the spindles increase, because of the properties of the function depicted in 5.

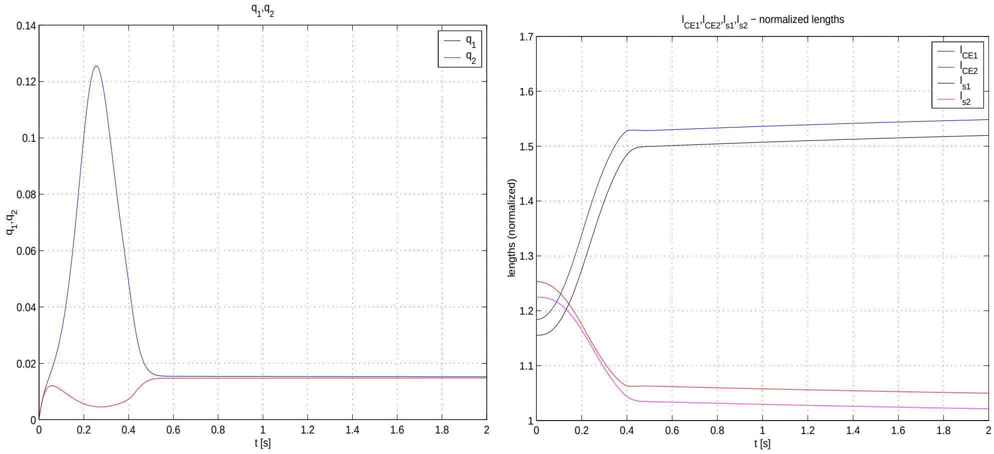

Figure 9: Muscle activation states, and muscle lengths

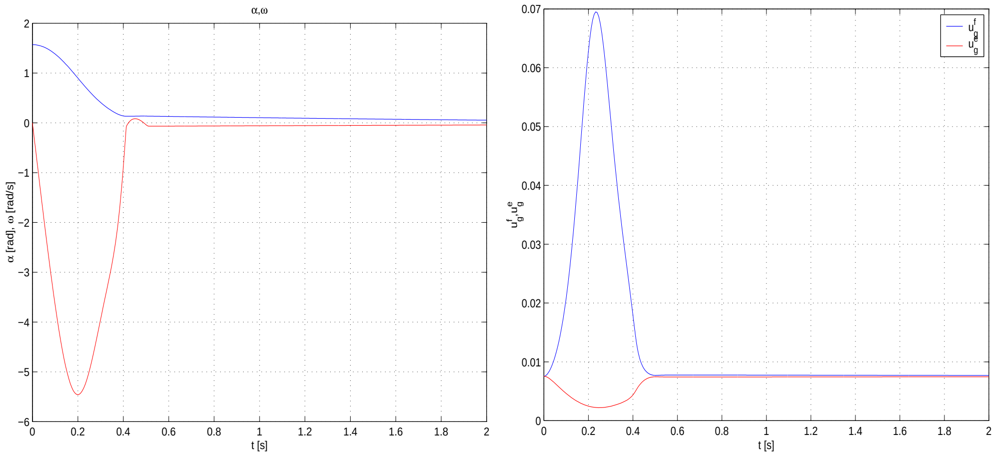

Figure 10: α, ω, u_g^f and u_g^e with active gamma-loop feedback

We can see, that the movement became more smooth, and a constant tone is appearing in the muscles.

Fourth simulation

Similar to the patellar reflex, we can encroach into the dynamics of the limb, and make a very good example of the gamma-loop's functioning. We can increase the muscle length by artificial ways (i.e. without change in the joint angle), and analyze the reaction of the system. In this simulation we generated an extra increment in the flexor muscle's length, at $\alpha = 0$ joint angle, approximately at the time $t = 1.3s$. The simulation results are depicted in the figures below:

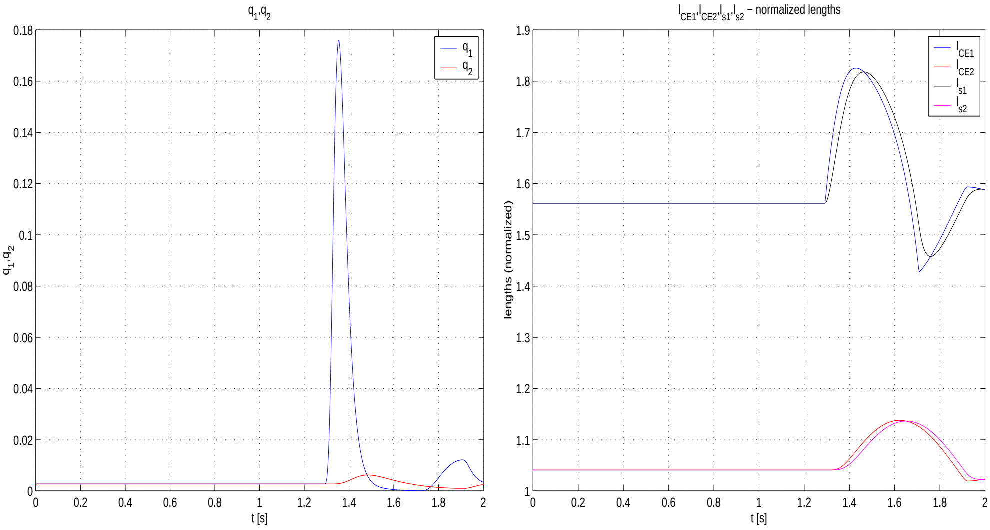

Figure 11: Muscle activation states, and muscle lengths

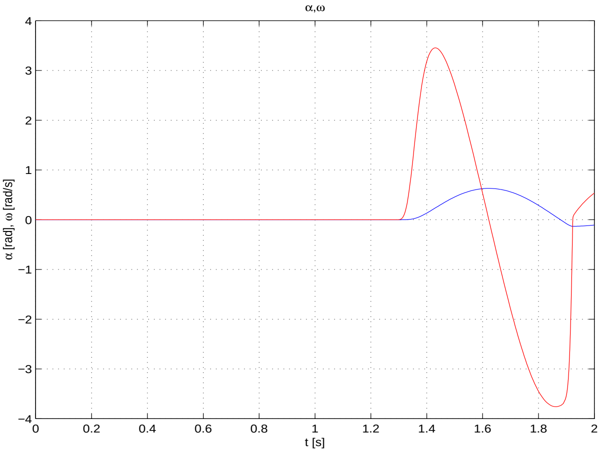

Figure 12: α and ω at stretch reflex

4.2 The second simplified model of the gamma-loop

In the second model of the muscle spindle an intrafusal fiber is composed of a linear elastic element representing the sensory part (the equatorial region) and a muscle fiber representing the muscular part (the polar region), as it can be seen in the next figure 13.

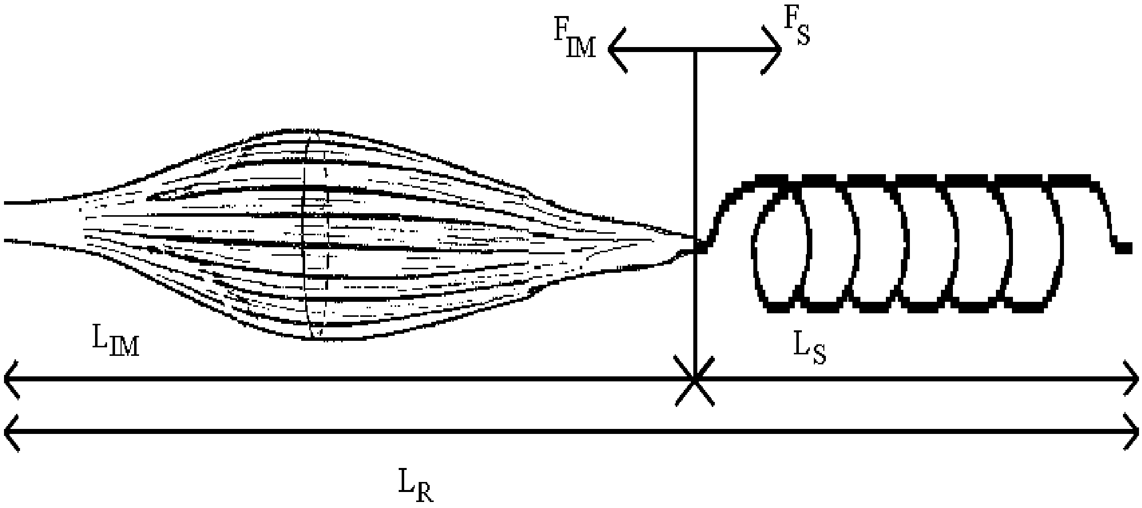

Figure 13: The second model of the muscle spindle

where l_{IM} denotes the length of the muscular part, l_S denotes the length of the spring, l_R denotes the length of the whole system, which is determined by the length of the surrounding fibers of the working muscle (which depend only on $\alpha = x_3$). F_{IM} is the notation of the force generated by the muscle fiber, F_S is the force of the spring. These two forces are always equal in our model.

Simplifying assumptions

- In the model, we are only interested in the motor component of the system. We suppose, that by sensory ways all the state-space variables are available for a controller. In our case the controller models the adequate functions of the CNS.
- The muscle spindle in the model refers to the summation of the spindles.
- The force generated by the muscular part of the spindle depends only on it's activation state (we neglect the force-length, and the force-contraction velocity characteristics in the intrafusal case).
- In the case of the spindles, only gamma innervation is modeled.
- Only static bag fibers are taken into account: The activation signal originating from the gamma-loop effect in this case is a sigmoid function of $\Delta L = L_S - L_{S0}$ (L_{S0} denotes the natural length of the spring).
- At first we do not model alpha-gamma coactivation.
- At first, we do not model polysynaptic control.

4.2.1 Model equations

We can describe the functioning of the model with the following equations:

$$F_S = D(L_S - L_{S0}) \tag{13}$$

where D denotes the spring constant, L_{S0} denotes the natural length of the spring.

$$F_{IM} = q_{IM} F_{IM}^{max} + F_{IPE}(L_{IM}) \tag{14}$$

In this equation q_{IM} denotes the activation state of the intrafusal muscle, F_{IM} is the maximum of the active force and $F_{IPE}(L_{IM})$ is the passive force generated by the intrafusal muscle. This function can be seen in figure 14

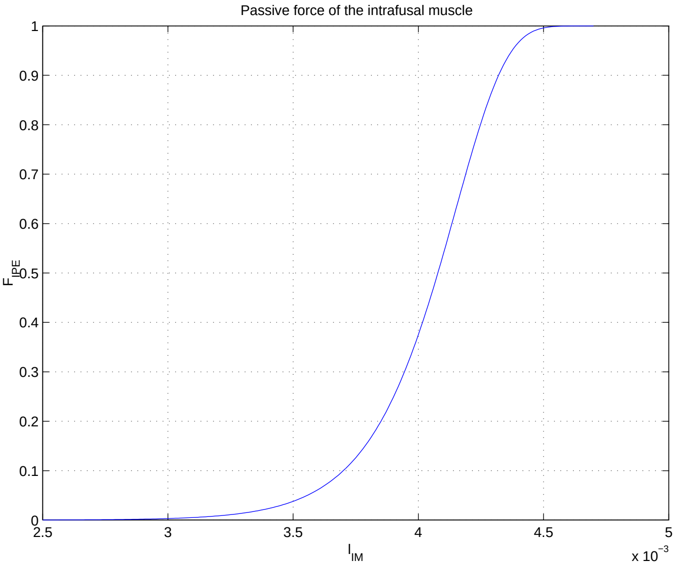

Figure 14: The passive force generated by the intrafusal muscle

Furthermore we can write:

$$L_S + L_{IM} = L_R \qquad \Rightarrow \qquad L_S = L_R - L_{IM} \tag{15}$$

So we can write:

$$D(L_R - L_{IM} - L_{S0}) = q_{IM} F_{IM}^{max} + F_{IPE}(L_{IM}) \tag{16}$$

$$q_{IM} F_{IM}^{max} + F_{IPE}(L_{IM}) - D(L_R - L_{IM} - L_{S0}) = 0 \tag{17}$$

If we take the time-derivative of the equation 17 (see [1]), and define $z = L_{IM}$, we get:

$$\dot{q}_{IM} F_{IM}^{max} + \frac{dF_{IPE}(z)}{dz}\dot{z} - D(\frac{dL_R(x_3)}{dx_3}\dot{x_3} - \dot{z}) = 0 \tag{18}$$

If we rearrange the equation above, we get an equation which can be fit to our state-space model with z as the new state-space variable.

$$\dot{z} = \frac{\dot{q}_{IM} F_{IM}^{max} - D \frac{dL_R(x_3)}{dx_3} x_4}{-(D + \frac{dF_{IPE}(z)}{dz})} \tag{19}$$

If we define $z^f = x_7$ for the flexor muscle and $z^e = x_8$ for the extensor muscle, and we suppose first order activation dynamics for the muscular part of the spindles, the state-space equations will take the following form:

$$\begin{aligned}
\dot{x_1} &= -\tfrac{1}{\tau_{act}}\beta x_1 + \left(-\tfrac{1}{\tau_{act}}(1-\beta)x_1 + \tfrac{1}{\tau_{act}}\right) u^f(t) \\
\dot{x_2} &= -\tfrac{1}{\tau_{act}}\beta x_2 + \left(-\tfrac{1}{\tau_{act}}(1-\beta)x_2 + \tfrac{1}{\tau_{act}}\right) u^e(t) \\
\dot{x_3} &= x_4 \\
\dot{x_4} &= \tfrac{1}{\Theta + ml_{COM}^2}(M(x_1, x_2, x_3, x_4) + ml_{COM} cos(x_3 + \xi) g_y) \\
\dot{x_5} &= -\tfrac{1}{\tau_{act}}\beta x_5 + \left(-\tfrac{1}{\tau_{act}}(1-\beta)x_5 + \tfrac{1}{\tau_{act}}\right) u_s^f(t) \\
\dot{x_6} &= -\tfrac{1}{\tau_{act}}\beta x_6 + \left(-\tfrac{1}{\tau_{act}}(1-\beta)x_6 + \tfrac{1}{\tau_{act}}\right) u_s^e(t) \\
\dot{x_7} &= \tfrac{\dot{x_5} F_{IM}^{max} - D \frac{dL_R^f(x_3)}{dx_3} x_4}{-(D + \frac{dF_{IPE}(x_7)}{dx_8})} \\
\dot{x_8} &= \tfrac{\dot{x_6} F_{IM}^{max} - D \frac{dL_R^e(x_3)}{dx_3} x_4}{-(D + \frac{dF_{IPE}(x_8)}{dx_8})}
\end{aligned} \tag{20}$$

where $u^f(t)$ denotes the total activation signal of the flexor muscle, $u^e(t)$ denotes the total activation signal of the extensor muscle, u_s^f denotes the activation signal originating from the spindle of the flexor muscle and u_s^e denotes the activation signal originating from the spindle of the extensor muscle.

$$u^f = u_d^f + u_g^f \tag{21}$$

where u_d^f denotes the direct activation signal of the flexor muscle, and u_g^f denotes the activation signal, which originates from the gamma-loop mechanism.

Let us define the following notation once more:
Let u_1 be the direct activation signal of the flexor muscle (u_d^f) (not originating from the gamma loop mechanism), u_2 the direct activation signal of the extensor muscle (u_d^e), both $\in [0,1]$, u_3 the activation signal of the flexor muscle's spindle (u_s^f), u_4 the activation signal of the extensor muscle's spindle (u_s^e)

In this case the gamma-feedback depends only on the actual length of the spring in the model.

With this notation for example u_g^f can be computed as:

$$u_g^f = f_{gas2}^f(x_3, x_7) \doteq 1 - \frac{1}{(1 + exp(10000(L_R^f(x_3) - x_7) - 61))^{30}} * UG_{max} \tag{22}$$

L_R denotes the total length of the muscle and the spring, and depends only on x_3. UG_{max} is the maximal value of the gamma-loop based activation signal. Of course in the case of the extensor muscle the equations are similar.

The function [22] can be seen in the next figure:

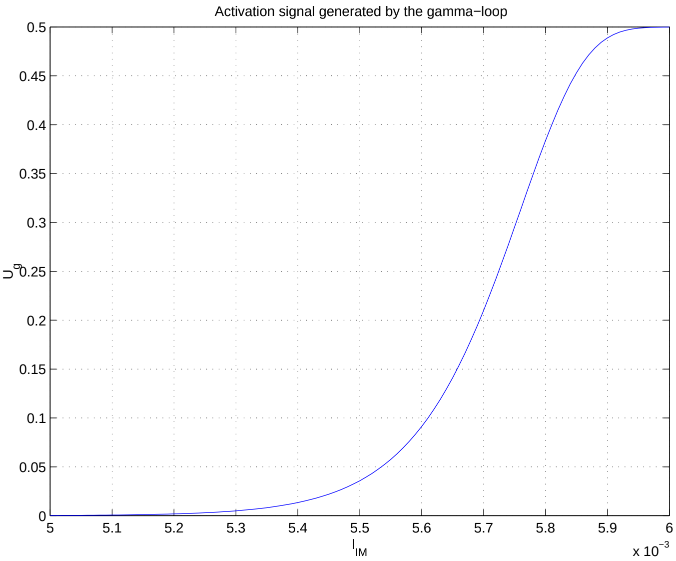

Figure 15: The activation signal generated by the gamma loop mechanism as a function of the spring's length

We can substitute these expressions in equation (6) to get the following form:

$$\begin{aligned}
\dot{x}_1 &= -\frac{1}{\tau_{act}}\beta x_1 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_1 + \frac{1}{\tau_{act}}\right)(f^f_{gas2}(x_3, x_5) + u_1) \\
\dot{x}_2 &= -\frac{1}{\tau_{act}}\beta x_2 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_2 + \frac{1}{\tau_{act}}\right)(f^e_{gas2}(x_3, x_6) + u_2) \\
\dot{x}_3 &= x_4 \\
\dot{x}_4 &= \frac{1}{\Theta + ml^2_{COM}}(M(x_1, x_2, x_3, x_4) + ml_{COM}cos(x_3 + \xi)g_y) \\
\dot{x}_5 &= -\frac{1}{\tau_{act}}\beta x_5 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_5 + \frac{1}{\tau_{act}}\right)u^f_s(t) \\
\dot{x}_6 &= -\frac{1}{\tau_{act}}\beta x_6 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_6 + \frac{1}{\tau_{act}}\right)u^e_s(t) \\
\dot{x}_7 &= \frac{\left(-\frac{1}{\tau_{act}}\beta x_5 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_5 + \frac{1}{\tau_{act}}\right)u^f_s(t)\right)F^{max}_{IM} - D\frac{dL^f_R(x_3)}{dx_3}x_4}{-(D + \frac{dF_{IPE}(x_7)}{dx_7})} \\
\dot{x}_8 &= \frac{\left(-\frac{1}{\tau_{act}}\beta x_6 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_6 + \frac{1}{\tau_{act}}\right)u^e_s(t)\right)F^{max}_{IM} - D\frac{dL^e_R(x_3)}{dx_3}x_4}{-(D + \frac{dF_{IPE}(x_8)}{dx_8})}
\end{aligned} \tag{23}$$

If we rearrange the equation (25) we can get the following general form of input-affine systems:

$$\begin{aligned}
\dot{x} &= f(x) + \textstyle\sum_{i=1}^{m} g_i(x)u_i(t) \\
y &= h(x)
\end{aligned} \tag{24}$$

where:

$$f(x) = \begin{pmatrix} -\frac{1}{\tau_{act}}\beta x_1 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_1 + \frac{1}{\tau_{act}}\right)(f_{gas2}^{f}(x_3,x_5)) \\ -\frac{1}{\tau_{act}}\beta x_2 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_2 + \frac{1}{\tau_{act}}\right)(f_{gas2}^{f}(x_3,x_5)) \\ x_4 \\ \frac{1}{\Theta+ml_{com}^2}(M(x_1,x_2,x_3,x_4) + ml_{com}cos(x_3)g_y \\ -\frac{1}{\tau_{act}}\beta x_5 \\ -\frac{1}{\tau_{act}}\beta x_6 \\ \frac{-\frac{1}{\tau_{act}}\beta x_5 F_{IM}^{max} - D\frac{dL_R^f(x_3)}{dx_3}x_4}{-(D+\frac{dF_{IPE}(x_7)}{dx_7})} \\ \frac{-\frac{1}{\tau_{act}}\beta x_6 F_{IM}^{max} - D\frac{dL_R^e(x_3)}{dx_3}x_4}{-(D+\frac{dF_{IPE}(x_8)}{dx_8})} \end{pmatrix}$$

$$g_1(x) = \begin{pmatrix} -\frac{1}{\tau_{act}}(1-\beta)x_1 + \frac{1}{\tau_{act}} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \qquad g_2(x) = \begin{pmatrix} 0 \\ -\frac{1}{\tau_{act}}(1-\beta)x_2 + \frac{1}{\tau_{act}} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

$$g_3(x) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ -\frac{1}{\tau_{act}}(1-\beta)x_5 + \frac{1}{\tau_{act}} \\ 0 \\ \frac{\left(-\frac{1}{\tau_{act}}(1-\beta)x_5 + \frac{1}{\tau_{act}}\right)F_{IM}^{max}}{-(D+\frac{dF_{IPE}(x_7)}{dx_7})} \\ 0 \end{pmatrix} \quad g_4(x) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ -\frac{1}{\tau_{act}}(1-\beta)x_6 + \frac{1}{\tau_{act}} \\ 0 \\ \frac{\left(-\frac{1}{\tau_{act}}(1-\beta)x_6 + \frac{1}{\tau_{act}}\right)F_{IM}^{max}}{-(D+\frac{dF_{IPE}(x_8)}{dx_8})} \end{pmatrix}$$

4.2.2 Model verification

Because the properties of the second model differ from the first and it does not include the *alpha-gamma coactivation mechanism* we get different results for the simulation tasks.

If we study the system with passive gamma-loop feedback, we get the following result: (in this case $u_d^f = u_d^e = u_s^f = u_s^e = 0$).

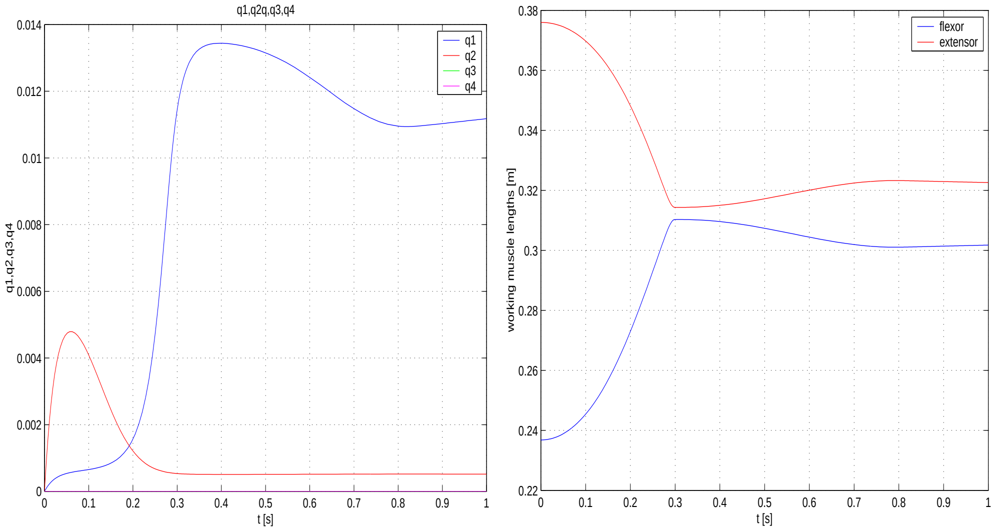

Figure 16: Muscle activation states, and muscle lengths

Because this model is not sensitive to quick changes in muscle length the gamma-feedback does not slow the descent of the limb in passive, or in active case.

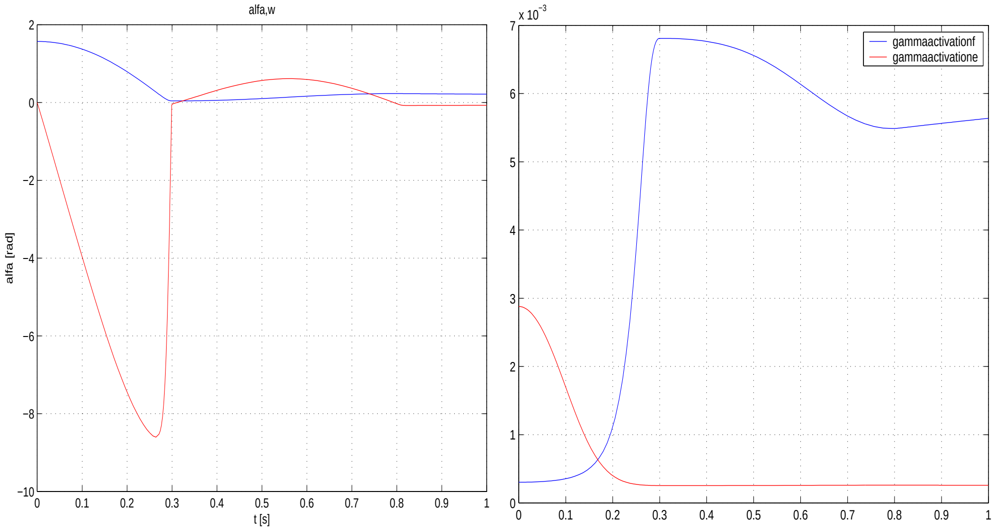

Figure 17: α, ω, u_g^f and u_g^e with passive gamma-loop feedback

4.3 Alpha-gamma coactivation

In this subsection we try to create a solution for the alpha-gamma coactivation problem appearing in the case of the second model. As we mentioned above, in the case of the first model this problem does not appear, because the intrafusal muscle length always follows the extrafusal length automatically (with a small delay).

In the case of the second model, the aim of the alpha-gamma coactivation is to keep the spring in the same length state, while the limb itself is moving, and the length of the intrafusal, and extrafusal muscles are changing. For this we have to determine the activation signal of the intrafusal muscles. But this signal depends not only in the position of the limb, but on the state of the muscle spindle model itself. This means if we are to keep a higher tone (what means, we need to keep the spring in a more stretched state), we need a completely different activation signal for the same movement pattern. So the explicit expression of the needed activation signal would be very difficult.

For this reason, we use the following method:

- We analyze only the *effect* of the alpha-gamma coactivation - because we suppose that it plays a key role in control mechanism, which has to be investigated in the future.
- We do not care about which way the the correct activation signal in physiological terms is determined (anyway a similar simple negative neural feedback with a reference signal is imaginable).
- We modify (expand) the model with two more differential equations, which are only to calculate the needed activation signal of the intrafusal muscles. The new state space variables (x_9, x_{10}) are the activation signals of the intrafusal muscles, and the new inputs (u_3, u_4) are the desired spindle lengths.

The equations are, as follows:

$$
\begin{aligned}
\dot{x_1} &= -\frac{1}{\tau_{act}}\beta x_1 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_1 + \frac{1}{\tau_{act}}\right)(f^f_{gas2}(x_3,x_5)+u_1) \\
\dot{x_2} &= -\frac{1}{\tau_{act}}\beta x_2 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_2 + \frac{1}{\tau_{act}}\right)(f^e_{gas2}(x_3,x_6)+u_2) \\
\dot{x_3} &= x_4 \\
\dot{x_4} &= \frac{1}{\Theta + ml^2_{COM}}(M(x_1,x_2,x_3,x_4) + ml_{COM}cos(x_3+\xi)g_y) \\
\dot{x_5} &= -\frac{1}{\tau_{act}}\beta x_5 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_5 + \frac{1}{\tau_{act}}\right)x_9 \\
\dot{x_6} &= -\frac{1}{\tau_{act}}\beta x_6 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_6 + \frac{1}{\tau_{act}}\right)x_{10} \\
\dot{x_7} &= \frac{\left(-\frac{1}{\tau_{act}}\beta x_5 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_5 + \frac{1}{\tau_{act}}\right)x_9\right)F^{max}_{IM} - D\frac{dL^f_R(x_3)}{dx_3}x_4}{-(D+\frac{dF_{IPE}(x_7)}{dx_7})} \\
\dot{x_8} &= \frac{\left(-\frac{1}{\tau_{act}}\beta x_6 + \left(-\frac{1}{\tau_{act}}(1-\beta)x_6 + \frac{1}{\tau_{act}}\right)x_10\right)F^{max}_{IM} - D\frac{dL^e_R(x_3)}{dx_3}x_4}{-(D+\frac{dF_{IPE}(x_8)}{dx_8})} \\
\dot{x_9} &= c_g(u_3 - (L^f_R(x_3) - x_7) \\
\dot{x_{10}} &= c_g(u_4 - (L^e_R(x_3) - x_8)
\end{aligned} \tag{25}
$$

We can easily determine f and g_i-s in this case:

$$f(x)=\begin{pmatrix}
-\frac{1}{\tau_{act}}\beta x_1+\left(-\frac{1}{\tau_{act}}(1-\beta)x_1+\frac{1}{\tau_{act}}\right)(f^f_{gas2}(x_3,x_5)) \\
-\frac{1}{\tau_{act}}\beta x_2+\left(-\frac{1}{\tau_{act}}(1-\beta)x_2+\frac{1}{\tau_{act}}\right)(f^f_{gas2}(x_3,x_5)) \\
x_4 \\
\frac{1}{\Theta+ml_{com}^2}(M(x_1,x_2,x_3,x_4)+ml_{com}cos(x_3)g_y \\
-\frac{1}{\tau_{act}}\beta x_5+\left(-\frac{1}{\tau_{act}}(1-\beta)x_5+\frac{1}{\tau_{act}}\right)x_9 \\
-\frac{1}{\tau_{act}}\beta x_6+\left(-\frac{1}{\tau_{act}}(1-\beta)x_6+\frac{1}{\tau_{act}}\right)x_10 \\
\dfrac{\left(-\frac{1}{\tau_{act}}\beta x_5+\left(-\frac{1}{\tau_{act}}(1-\beta)x_5+\frac{1}{\tau_{act}}\right)x_9\right)F_{IM}^{max}-D\frac{dL_R^f(x_3)}{dx_3}x_4}{-(D+\frac{dF_{IPE}(x_7)}{dx_7})} \\
\dfrac{\left(-\frac{1}{\tau_{act}}\beta x_6+\left(-\frac{1}{\tau_{act}}(1-\beta)x_6+\frac{1}{\tau_{act}}\right)x_{10}\right)F_{IM}^{max}-D\frac{dL_R^e(x_3)}{dx_3}x_4}{-(D+\frac{dF_{IPE}(x_8)}{dx_8})} \\
C_g\left(-(L_r^f(x_3)-x_7)\right) \\
C_g\left(-(L_r^e(x_3)-x_8)\right)
\end{pmatrix}$$

$$g_1(x)=\begin{pmatrix}
-\frac{1}{\tau_{act}}(1-\beta)x_1+\frac{1}{\tau_{act}} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0
\end{pmatrix}
\qquad
g_2(x)=\begin{pmatrix}
0 \\ -\frac{1}{\tau_{act}}(1-\beta)x_2+\frac{1}{\tau_{act}} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0
\end{pmatrix}$$

$$g_3(x) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ C_g \\ 0 \end{pmatrix} \quad g_4(x) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ C_g \end{pmatrix}$$

4.4 Simulation results

In the following figures a simulation of a simple descending movement can be seen, applying the alpha-gamma coactivation model.

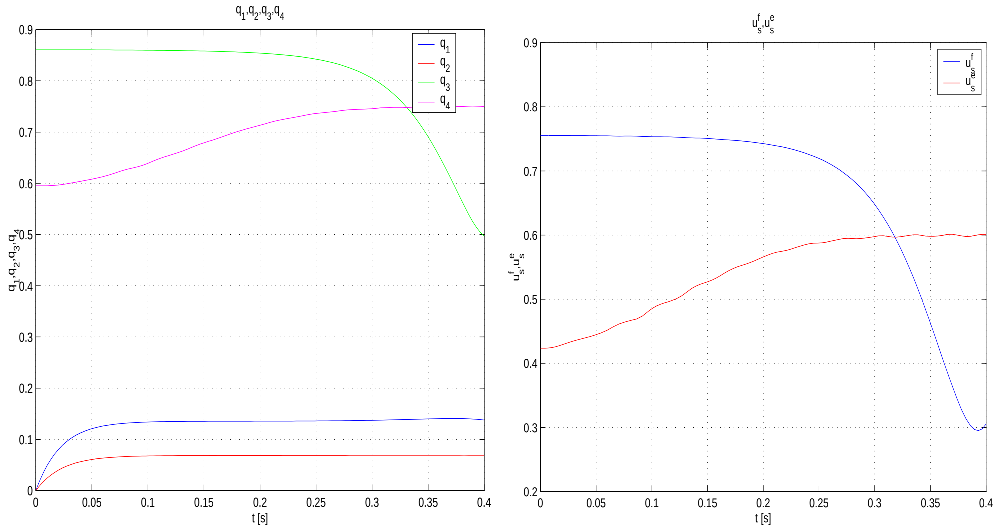

Figure 18: Muscle activation states, and the activation signals of the intrafusal muscles

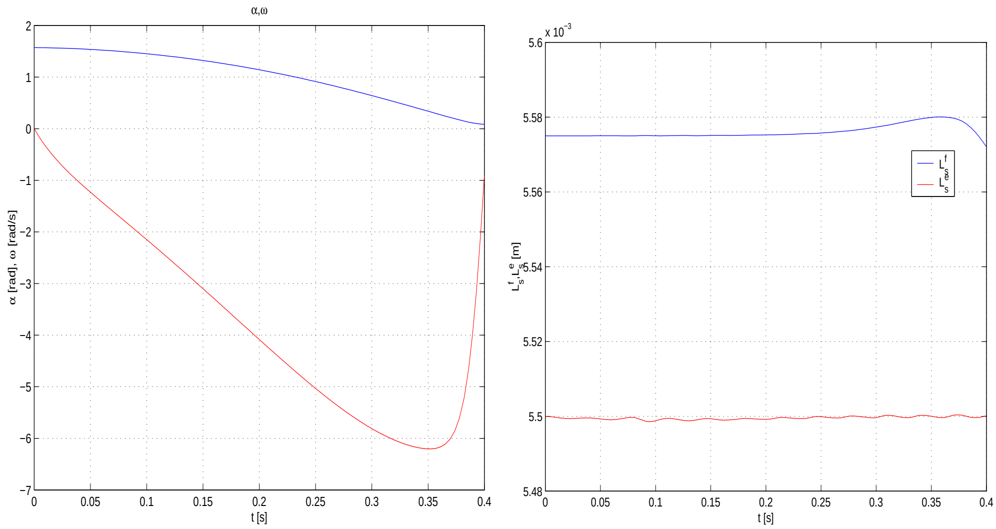

Figure 19: α, ω, and L_s^f, L_s^e

As we can see in the figures above, in the case of a simple descending movement, the length of the springs stay in the close range of the desired spring lengths (0.005575 and 0.0055 m), and the intrafusal activation signal changes. The beginning oscillation is caused by the value of C_g - if we choose a lesser value, less oscillation can be seen, but the length of the spring convergates with less speed to the prescribed value.

5 Discussion

The properties of the two model show some important differences:

- The first model is more simple, but takes more dynamical properties into account (the alpha-gamma coactivation is included in the basic model, and because of the length of the intrafusal fibers follow the length of the extrafusal ones with delay, some more dynamical properties appear). Due to its simplicity external interventions, as an external change in the muscle length (in the case of the stretch-reflex), can be easily done. This would be more difficult in the case of the second model, because the state-space equations which describe the length of the intrafusal fibers (equation 19) contain terms which are derived from the function which describes the muscle length in normal conditions. In a such case, all these functions have to be modified. In fact in the case of the first model there is no information about the stretch and the forces inside the intrafusal fibers.

- The second model is based on the real physical properties of the muscle spindle (and so we can suppose, that the parameters of the model can be more easily measured), but becomes more sophisticated as more dynamical properties are taken into account (alpha-gamma coactivation, etc.). In fact it is more realistic and it can describe the stretch states and the forces inside the intrafusal fibers. Moreover the muscle spindle works also as a receptor of stretch-difference between the intrafusal and the extrafusal fiber. Due to this, if in future studies a model will be constructed which also takes the the spindle as a receptor into account, it will be probably based on the second model.

Taking it all round, the first model can more easily show the dynamical properties which are to investigate in control-design researches, but the second model can describe the functioning of any actuation or control method in a more accurate way, and it is more suitable for extending the model with receptor functions.

6 Conclusions and future work

In this short article two different models of the gamma-loop mechanism were constructed. The models take the phenomenon of the spinal chord feedback, originating from the signals of static bag fibers, and the alpha-gamma coactivation into account. The aim of the work described in this article was to propose a principle model for future research, which aim will be to investigate the role of the gamma-loop mechanism in human locomotion control.

6.1 Future work

- The models can be easily expanded to deal with polysynaptic control.
- The models can be expanded with the equations describing the functioning of dynamic bag fibers.
- The physical model of the muscle spindle can be improved, to better describe the properties of a real spindle. Parameters of such models can be identified with in-vitro experiments.
- The dynamic properties of the models have to be analyzed for future control purposes.
- The models can be completed with a controller, which describes the adequate functions of the higher level structures of the CNS (Cerebellum, Cortex, etc.).
- The models can be extended with the equations describing the receptor functions of the muscle spindles, and the Golgi tendon organs.

7 Acknowledgements

The author would like to thank Katalin M. Hangos, László Simon and Csaba Fazekas for their help in the research.

References

[1] Cameron, I.T. and Hangos, K.M. *Process Modelling and Model Analysis.* Academic Press, London, 2001.

[2] Csercsik, D. *Analysis and Control of a Simple Nonlinear Limb Model.* Degree Thesis, Budapest University of Technology and Economics, Budapest, Hungary, http://daedalus.scl.sztaki.hu, 2005.

[3] Enoka, R.M. *Neuromechanics of Human Movement.* Human Kinetics, 2002.

[4] Haines, D.E. *Fundamental Neuroscience.* Elsevier, 2001.

[5] Hasan, Z. A model of spindle afferent response to muscle stretch. *Journal of Neurophysiology*, 49:989–1006, 1983.

[6] Loeb, G.E., Brown, I.E., and Cheng E.J. A hierarchical foundation for models of sensorimotor control. *Experimental brain research*, 126:1–18, 1999.

[7] Loeb, G.E. and Levine, W.S. Linking musculoskeletal mechanics to sensorimotor neurophysiology. *Multiple Muscle Systems: Biomechanics and Movement Organization, J.M.*, pages 165–181, 1990.

[8] Maltenfort, M. G. and Burke, R. E. Spindle model responsive to mixed fusimotor inputs and testable predictions of beta feedback effects. *Journal of Neurophysiology*, 89:797–809, 2003.

[9] Paul, C., Bellotti, M., Jezernik, S., and Curt, A. Development of a human neuro-musculo-skeletal model for investigation of spinal cord injury. *Biological Cybernetics*, 93:153–70, 2005.

[10] Prochazka, A., Gillard, D., and Bennett, D.J. Positive force feedback control of muscles. *Journal of Neurophysiology*, 77:3226–3236, 1997.

[11] Stein, R.B. and Oguztoreli, M.N. The role of gamma-motoneurons in mammalian reflex systems. *Biological Cybernetics*, 39:171–9, 1981.

[12] Szentágothai, J. *Functionalis Anatomia.* Medicina Press, 1975.

[13] Szirmai, I. *Neurológia.* Medicina Press, 2005.

[14] van Schaafsma, A., van Otten, E., and Van Willigen, J.D. A muscle spindle model for primary afferent firing based on a simulation of intrafusal mechanical events. *Journal of Neurophysiology*, 65:1297–312, 1991.