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► **To cite this version:**

Ali El Ati, Islam Boussaada, Silviu-Iulian Niculescu, Pierre-Henry Poret. Analysis of the Delayed Central Nervous System Action in the Regulation of a Third-order Muscle-Tendon Model. 24th International Conference on System Theory, Control and Computing (ICSTCC 2020), Oct 2020, Sinaia, Romania. 10.1109/icstcc50638.2020.9259775 . hal-02944531

HAL Id: hal-02944531

<https://hal.science/hal-02944531>

Submitted on 21 Sep 2020

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Analysis of the Delayed Central Nervous System Action in the Regulation of a Third-order Muscle-Tendon Model

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Abstract—This paper presents the analysis of a third-order linear time-invariant delay differential equation representing the regulation of a muscle-tendon system. The Central Nervous System action is modeled as a delayed proportional-derivative controller exploiting the multiplicity-induced-dominancy property. The stability analysis is illustrated via the software P3 δ .

Index Terms—Time-delay systems, stability and stabilization, multiplicity-induced-dominancy, muscle-tendon dynamics, P3 δ software.

I. INTRODUCTION

The control of human motricity is one of the paramount tasks of our Central Nervous System (CNS). To understand the human body movement, one needs not only to establish a good model of the muscle dynamics but also to analyse the control circuit including the structural form of the CNS instruction that controls the dynamic of muscle forces [1]. For instance, abnormal movements caused by neuromuscular diseases, such as Parkinson’s disease or sclerosis, result from abnormal muscle tone. Furthermore, maintaining the balance is a vital ability for humans: falls are leading causes of accidental death and morbidity in the elderly, a fact which provides a strong motivation to understand the functioning of the CNS, [2]. Actually, the CNS generates neural commands to activate the muscles. The intern muscles’ force combined with inertia and external forces, generate observable movements. The position and velocity of the musculoskeletal system are measured and transmitted to CNS to close the loop with the required information to take appropriate control decisions [3]. However, there is a substantial time-delay caused by

This work is partially supported by a public grant overseen by the French National Research Agency (ANR) as part of the “Investissement d’Avenir” program, through the iCODE project funded by the IDEX Paris-Saclay, ANR-11-IDEX0003-02. The authors wish to acknowledge the work of the full P3 δ development team as well as the Cyb’Air Association.

the finite speed of signal and the performance of motor tasks is affected by the presence of time-delayed sensory feedback [4], [5]. Moreover, the intrinsic CNS functioning is complex itself, being inherently generator of high-dimensional and nonlinear dynamics. As such, a substantial time delay of signal propagation in the nervous system has to be considered in the input signals. One intuitive and simple way to model such a CNS response is to identify it as a system of propagation which is justified by the necessary lag-time for an information to get through the neuronal axon. We refer the reader to [6] for a summary of the different kinds of delays occurring in neural systems.

The mathematical models that take into account the delay effects in the nervous system are delay functional differential equations which are of infinite dimension and may exhibit a complex dynamical behaviour due to the delay effect. It has been recently shown (see, e.g., [7]–[12]) that, for some quasipolynomials occurring in systems with time-delays, multiple real roots are often dominant, a property usually referred to as *multiplicity-induced-dominancy* (MID for short). If, in addition, this multiple dominant root is negative, exponential stability is guaranteed. Namely, a control-oriented MID approach was first proposed in [10] for second-order delay equations then extended in [13] for general n^{th} -order linear time-invariant dynamical systems with a single delay. Indeed, it was shown that under appropriate conditions, the MID property may assess the critical delay established in previous works [9], [14], [15]. In this paper, we consider that the CNS acts as a delayed PD-controller to regulate the muscle dynamics by exploiting the MID property.

Several mathematical models have been proposed to represent muscle dynamics, which are usually classified into three groups [16]. The first one is based on black box approximation where the inputs are either the neural signal or the external load, and the output corresponds to either the joint position

or torque. Hence, the muscle-tendon mechanism may be regarded as a second order system [1]. The second group are Huxley-based distributed-parameter models that seek to explain faithfully the mechanism of contraction with great accuracy, however, its use is not recommended in movement studies because the mathematical complexity of this type of models is paramount and involves ever-increasing numbers of rate functions to describe the chemical and mechanical processes at play in the muscle contraction. Finally, Hill-type lumped-parameter models [17] are the most commonly used to study human movement and postural stability [18], [19], [20]. As a matter of fact, these models describe the mechanical behaviour of the muscle tissue by means of the parallel between the elastic element (PEE), accounting for the passive elastic properties of the muscle fibers, and the contractile element (CE) responsible of the active force generated in the muscle. The elastic properties of the tendon are represented by a spring attached in series with the Hill-type muscle model [21], [22]. Extensive and detailed accounts of Hill-based muscle models are accessible in [16] and [23]. Fig. 1 gives a schematic representation of the muscle-tendon unit using the Hill-type muscle model. In the literature, various linear approximations of Hill models are developed. In particular, the Kelvin-Voigt (KV) muscle-tendon model interprets the mechanical network by the parallel between a single stiffness and a damping element, and the system dynamic is viewed as a second-order equation. It was observed that KV models present some limits and are particularly not adapted to represent tendon and muscle contractile elements independently [24]. Therefore, a third-order system is proposed in the form of a Poynting-Thomson (PT) model. In this model, a supplementary elastic element is placed in series with a KV model to represent the tendon, bringing them closer to the physiological muscle-tendon system. However, the presence of the tendon element can produce an oscillatory behavior that needs to be compensated by the CNS controller. For the reasons stated above, we shall use a third-order PT model to investigate the stability of a muscle-tendon system acting in an arm through the wrist joint.

The paper is organized as follows. Section 2 is dedicated to the modeling of the muscle-tendon interaction as well as to the description of the delayed CNS action. In Section 3, we present the main result where the stability of the muscle response is analysed. Also, the oscillation-inducing critical delay is characterized. Section 4 illustrates the main result by using the recently established P3 δ software. Section 5 concludes the contribution.

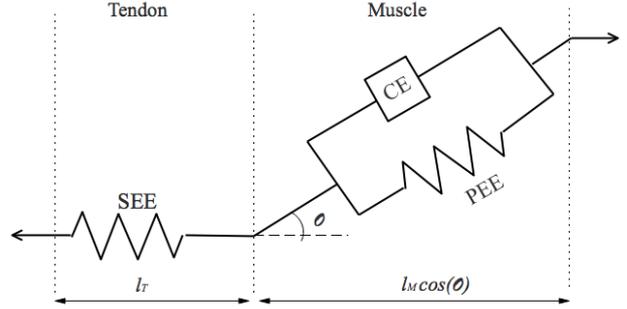


Figure 1. Nomenclature used in the Hill-type muscle model. Each musculotendon actuator is represented by muscle elements in series with an elastic tendon (SEE). The mechanical behavior of muscle is described by a Hill-type contractile element (CE) that models the muscle's force-length-velocity property, and a parallel-elastic element (PEE) that models the muscle's stiffness. (Inspired from [23])

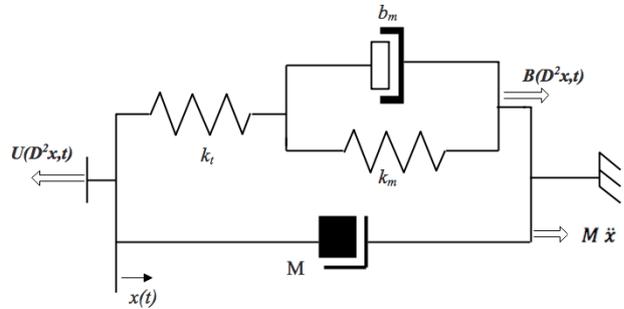


Figure 2. PT model of muscle-tendon system. The mechanical behavior of muscle is described by the parallel between k_m and b_m that represent the stiffness and viscosity of muscle fibers. The stiffness of the tendon is represented by k_t . The diagram depicts the different force fields of the D'Alembert equation. (Inspired from [25])

II. MUSCLE-TENDON MODELS:

The Poynting-Thomson (PT) model depicted in Fig. 2 is a linear, time-invariant and third-order system. This mechanical network is an extension of the Kelvin-Voigt model and includes tendon elasticity (Hill-type passive model). The PT model includes two separate elastic elements, the stiffness k_t of the tendon in series with the muscle fibers and the parallel between k_m and b_m represents the stiffness and viscosity of the muscle fibers (b_m is a contractile element accounting for muscle damping). Most identification techniques proposed in the literature assume the damping and stiffness to be time-invariant. One can show that the interaction of the muscle-tendon model with an inertial system (e.g., the mass of bones and connective tissues) yields a third-order system [24].

Consider $x(t)$ as a vector of generalized variations of position coordinates (angles, Cartesian co-

ordinates,...) describing the motion of a mechanical system and define $D^n x$ as the set representing the position coordinates' variations and their derivatives with respect to time up to the n -th order so that

$$D^n x = \left(\frac{d^n x}{dt^n}, \dots, \frac{d^2 x}{dt^2}, \frac{dx}{dt}, x \right)$$

with $n \in \mathbf{N}$.

Owing to the Lagrange-D'Alembert principle, the mechanical system may be written as follows

$$M(x, t) \frac{d^2}{dt^2}(x(t)) + B(D^2 x, t) = U(D^2 x, t) \quad (1)$$

where $M(x, t)$ is the inertial matrix of the system in the chosen coordinate frame, $B(D^2 x, t)$ is the internal force field generated by the mechanical network, and $U(D^2 x, t)$ is the external force field, (see [24], [25] for more details).

Assuming the system is stationary, classical Laplace transform techniques can be used to recast equation (1) as:

$$M s^2 X(s) + B(s) = U(s) \quad (2)$$

According to the PT model represented in Fig. 2, the transfer function of the internal force generated by the mechanical network is the transfer function of the series of tendon and muscle fibers, namely

$$\frac{B(s)}{X(s)} = - \frac{k_t Z_m(s)}{k_t + Z_m(s)} \quad (3)$$

where $Z_m(s)$ is the transfer function of the muscle fibers composed by the parallel between the stiffness and viscosity of the muscle fibers, which is given by

$$Z_m = b_m s + k_m. \quad (4)$$

Substituting equation (4) in (3) yields the following transfer function

$$\frac{B(s)}{X(s)} = - \frac{k_t(b_m s + k_m)}{k_t + k_m + b_m s} \quad (5)$$

which we rewrite in the following fashion

$$B(s) = - \frac{k_t b_m s}{k_t + k_m} X(s) - \frac{k_t k_m}{k_t + k_m} X(s) - \frac{b_m}{k_t + k_m} s B(s). \quad (6)$$

Combining equations (2) and (5), we infer

$$\left(M s^2 - \frac{k_t(b_m s + k_m)}{k_t + k_m + b_m s} \right) X(s) = U(s) \quad (7)$$

In the absence of external force, it was demonstrated that the PT model with a parameters range in the normal physiological scales often exhibits an oscillatory free response [25]. That is why, the central nervous system (CNS) forces controller is required to cope with such a bio-mechanical constraint by way of a delayed feedback regulation. Hence, we propose that the external force field consists of the CNS action

Parameters	value
k_t	50.1 Nm/rad
k_m	18.9 Nm/rad
b_m	0.5 Nms/rad
M	0.002 kg/m ²

Table I
NUMERICAL SETTING FOR MUSCLE-TENDON MODEL

$U_{CNS}(s, \tau)$ and an external impulsive perturbation U_{EX} , i.e.

$$U(s) = U_{CNS}(s, \tau) + U_{EX}(s) \quad (8)$$

Neural feedback control mechanisms for muscle are time-delayed. In other words, there is a significant time interval between the measurement of the variables the application of the forces. Consequently, the CNS input applied to the muscle-tendon system can be represented by

$$U_{CNS}(s, \tau) = (\alpha + \beta s) e^{-\tau s} X(s) \quad (9)$$

Then, simple calculations lead to the closed-loop transfer function

$$F(s) = \frac{X(s)}{U_{EX}(s)} = \frac{k_t + k_m + b_m s}{\tilde{P}(s) + \tilde{Q}(s)e^{-\tau s}}, \quad (10)$$

where

$$\begin{cases} \tilde{P}(s) = M b_m s^3 + M(k_t + k_m) s^2 - k_t b_m s - k_t k_m, \\ \tilde{Q}(s) = -\beta b_m s^2 - (\alpha b_m + \beta(k_t + k_m)) s \\ \quad - (k_t + k_m)\alpha. \end{cases} \quad (11)$$

Numerical setting :

We use bio-mechanical properties of muscles and tendon in the arm as reported in the literature (see [5], [25], [26],). In [25], authors give a good estimation of stiffness parameters for 5 main muscles across both the wrist and elbow. Under the assumption that each muscle contributes to the rotational stiffness of the joint on which it acts, they infer the equivalent joint stiffness at the wrist. Moreover, the arm's moment is calculated where both the wrist and the elbow are completely extended (the joint angle $\theta = 0$). The numerical parameters of PT muscle-tendon model in the wrist joint are presented in Table 1.

III. MAIN RESULTS

We investigate the stabilizing delayed-controller modeling the CNS action, as well as the range of the delay up to the limit of stability, which corresponds to the so-called *delay margin*. In closed-loop, the corresponding normalized characteristic function reads:

$$\Delta(s, \tau) = P(s) + Q(s) e^{-\tau s} \quad (12)$$

where P is a unitary polynomial, i.e., $P = \tilde{P}/(M b_m)$ and $Q = \tilde{Q}/(M b_m)$

The main idea of the delayed design based on the MID property is to force the quasipolynomial (12) to have a root of a prescribed multiplicity, bounded by the *quasipolynomial degree*¹ allowing to tune the controller gains according to a prescribed exponential decay of the closed-loop system solutions. More precisely, the control-oriented MID suggests a root of multiplicity equal to the order of the polynomial associated to the delay plus one. In our case, the CNS acts as a delayed PD-controller.

As a matter of fact, this multiplicity constraint defines a manifold in the parameter space enabling the tuning of the gains α and β when the delay τ is left-free and guaranteeing the exponential stability of the closed-loop system solution. Hereafter, the next theorem, which is based on the MID property, states the explicit conditions on the parameters' values guaranteeing the targeted multiplicity. Recall that the multiplicity of a given root of the generic quasipolynomial (12) is bounded by its degree, so that is 6 is the bound of multiplicity in our case. Introducing the parameters' settings as in Table 1, the quasipolynomial function (12) becomes:

$$\Delta(s) = s^3 + 138 s^2 - 25050 s - 946890 + (-500 \beta s^2 - (69000 \beta + 500 \alpha) s - 69000) e^{-\tau s} \quad (13)$$

Proposition 1. *The following assertions hold:*

- i) *The multiplicity of a given root of (13) is bounded by 4.*
- ii) *The only admissible quadruple roots for (13) are in the set $s \in \{-407.6856536, -22.86735033\}$;*
- iii) *The real number $s_0 = -22.86735033$ is a quadruple root of (13) if, and only if, $\tau_0 = 0.01059835599, \alpha_0 = -13.75341181, \beta_0 = -0.4143339847$;*
- iv) *If $s_0 = -22.86735033$ is a quadruple root of (13) then s_0 is also the corresponding spectral abscissa;*
- v) *For a positive delay perturbation $\tau = \tau_0 + \epsilon$ with $\epsilon > 0$, the first crossing frequency $\omega = 11.91836147$ occurs for $\epsilon = 0.0002807867460$.*

Proof. The degree of the quasipolynomial function defined in (13) is equal to 6 and the Pólya and Szegő result from [27] asserts that 6 is the generic bound of the multiplicity of any root of (13).

One first investigates the vanishing of the quasipolynomial Δ . It yields the elimination of the exponential term as a rational function in s :

$$e^{-\tau s} = \frac{-s^3 - 138 s^2 + 25050 s + 946890}{-500 \beta s^2 + (-69000 \beta - 500 \alpha) s - 69000 \alpha}. \quad (14)$$

¹The degree of a given quasipolynomial is defined as the sum of the degrees of the involved polynomials plus the number of corresponding delays.

Next, to investigate potential roots with algebraic multiplicity 4, one substitutes the obtained identity (14) in the ideal \mathcal{I}_4 generated by the first three derivatives of Δ , i.e., $\mathcal{I}_4 = \langle \partial_s \Delta, \partial_s^2 \Delta, \partial_s^3 \Delta \rangle$, which yields a manifold defined by three algebraic equations in four unknowns (s, α, β, τ) . Solving it and substituting the obtained solution in (14) determines the two admissible solutions given in ii). Furthermore, one easily checks that both of them are not roots of $\partial_s^4 \Delta = 0$, which concludes the proof of i).

Items ii-iii) follow directly from the proof of i).

Item iv) follows directly from [13]. Indeed, the main result therein provides sufficient conditions for the dominance of a multiple root, which is essentially based on the real-rootedness of the delay-free polynomial P . Namely, one easily proves this fact by computing the discriminant of P the positivity of which guarantees three real roots.

To show item v), the sensitivity analysis with respect to the delay uncertainties is studied, which brings one to investigate the quasipolynomial:

$$\Delta_\epsilon(s) = s^3 + 138 s^2 - 25050 s - 946890 + (207.1669924 s^2 + 35465.75084 s + 948985.4149) e^{-(0.01059835599 + \epsilon)s}$$

By substituting $s = i\omega$ in the above quasipolynomial, one deduces the corresponding crossing frequencies. Indeed, separating real and imaginary parts, the following system of trigonometric polynomials is satisfied:

$$\begin{aligned} & - (207.1669924 \omega^2 - 948985.4149) \times \\ & \quad \cos(0.01059835599 \omega + \omega \epsilon) \\ & + 35465.75084 \omega \sin(0.01059835599 \omega + \omega \epsilon) \\ & - 946890 - 138 \omega^2 = 0, \\ & 35465.75084 \omega \cos(0.01059835599 \omega + \omega \epsilon) \\ & + (207.1669924 - 948985.4149 \omega^2) \times \\ & \quad \sin(0.01059835599 \omega + \omega \epsilon) - \omega^3 - 25050 \omega = 0 \end{aligned}$$

Eliminating $\cos(0.01059835599 \omega + \omega \epsilon)$ from the first trigonometric polynomial and feeding it into the second, we obtain the frequency $\omega_c = 11.9183614$. It is then substituted into the expression of $\cos(0.01059835599 \omega + \omega \epsilon)$ to recover the value for $\epsilon_c = 0.0002807867460$, which concludes the proof of v).

IV. NUMERICAL SIMULATIONS USING P3 δ SOFTWARE

Based on recent results from [10], [13], [28] on the multiplicity-induced-dominancy property for systems with single time-delays, a Python toolbox for the parametric design of stabilizing feedback laws with time-delays, called Partial Pole Placement via Delay Action (P3 δ for short), has been developed, see

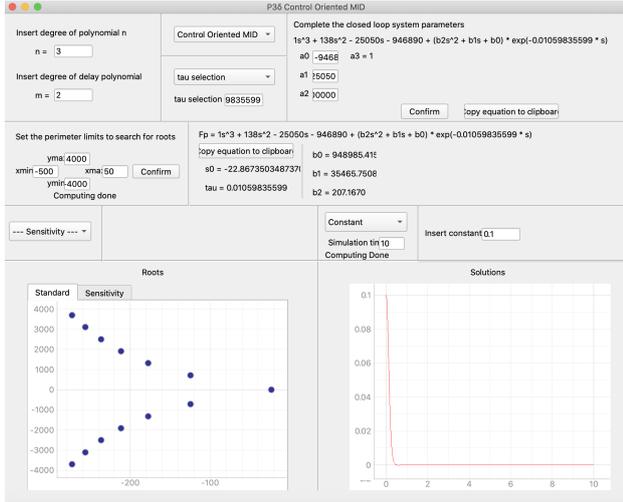


Figure 3. The P3 δ interface exhibiting (left) the spectrum distribution of (13) and (right) the time-domain response of the muscle dynamics in the closed-loop system, in the presence of a quadruple spectral value for (13) at $s = -22.86735033$.

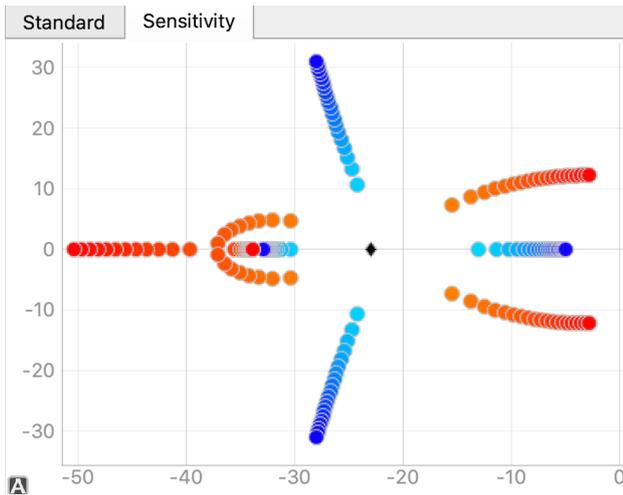


Figure 4. The Sensitivity of the multiple dominant spectral value with respect to delay uncertainty which illustrates item v) of Proposition 1.

[29]. The software is freely available for download on <https://cutt.ly/p3delta>, where installation instructions, video demonstrations, and a user guide are also available.

In this section, we illustrate the use of P3 δ to numerically investigate the problem we consider; the design of the CNS action as well as the sensitivity of its regulation with respect to the uncertain delay are displayed.

V. CONCLUDING REMARKS

A third-order muscle-tendon model is revisited taking into account the delay in the CNS action. The

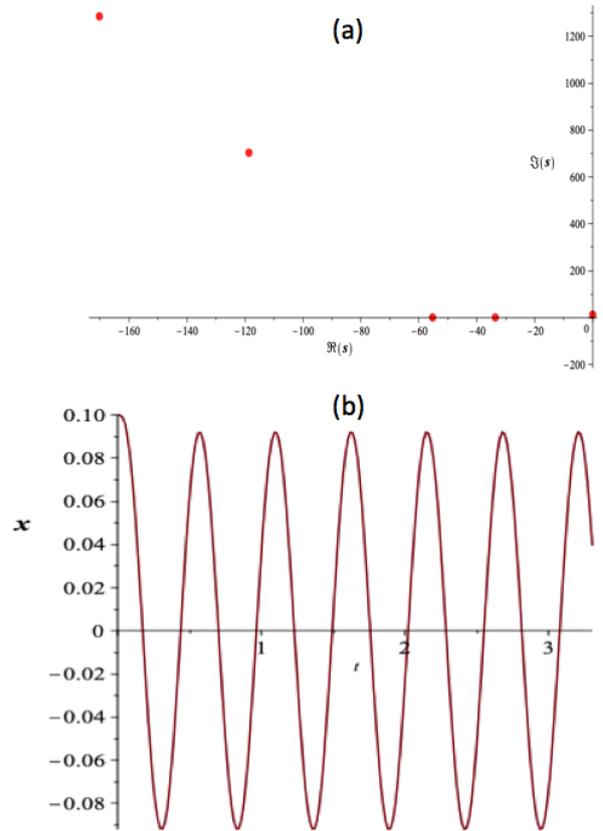


Figure 5. (a) Spectrum distribution for $\tau = 0.01087914274$ showing the crossing frequency $\omega = 11,91836147$ rad/sec. (b) Muscle oscillating response occurring with frequency $\omega = 11,91836147$ rad/sec for the delay value $\tau = 0.01087914274$.

MID property is exploited to model the CNS action and identify the critical delay inducing oscillations. The P3 δ software is used to numerically illustrate the qualitative behavior of the muscle dynamics.

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