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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 P3δ.

Index Terms—Time-delay systems, stability and stabilization, multiplicity-induced-dominancy, muscle-tendon dynamics, P3δ 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 analyze 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 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.

Several mathematical models have been proposed to represent muscle dynamics, which are usually classified into three groups [10]. 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

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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-Voight (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 P3δ software. Section 5 concludes the contribution.

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

\[
D^n x = \left( \frac{d^n x}{dt^n}, \ldots, \frac{d^3 x}{dt^3}, \frac{d^2 x}{dt^2}, \frac{dx}{dt}, x \right)
\]

with \( n \in \mathbb{N} \).

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

\[
M(x, t) \frac{d^2}{dt^2} (x(t)) + B(D^2 x, t) = U(D^2 x, t)
\]

(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:

\[
M \ s^2 \ X(s) + B(s) = U(s)
\]

(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

\[
\frac{B(s)}{X(s)} = -\frac{k_t \ Z_m(s)}{k_t + Z_m(s)}
\]

(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

\[
Z_m = b_m \ s + k_m.
\]

(4)

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

\[
\frac{B(s)}{X(s)} = -\frac{k_t (b_m \ s + k_m)}{k_t + k_m + b_m \ s}
\]

(5)

which we rewrite in the following fashion

\[
B(s) = -\frac{k_t \ b_m \ s}{k_t + k_m} X(s) - \frac{k_t k_m}{k_t + k_m} X(s) - \frac{b_m}{k_t + k_m} s B(s)
\]

(6)

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

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

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

\[
U(s) = U_{\text{CNS}}(s, \tau) + U_{\text{EX}}(s)
\]

(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

\[
U_{\text{CNS}}(s, \tau) = (\alpha + \beta \ s) e^{-\tau s} X(s)
\]

(9)

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

\[
F(s) = \frac{X(s)}{U_{\text{EX}}(s)} = \frac{k_t + k_m + b_m s}{P(s) + Q(s) e^{-\tau s}}
\]

(10)

where

\[
\begin{cases}
\dot{P}(s) = M b_m s^3 + M (k_t + k_m) s^2 - k_t b_m s - k_t k_m,
\dot{Q}(s) = -\beta b_m s^2 - (\alpha b_m + \beta (k_t + k_m)) s - (k_t + k_m) \alpha.
\end{cases}
\]

(11)

**Numerical setting:**

We use bio-mechanical properties of muscles and tendon in the arm as reported in the literature (see [3], [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:

\[
\Delta(s, \tau) = P(s) + Q(s) e^{-\tau s}
\]

(12)

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

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>( k_t )</td>
<td>50.1 Nm/rad</td>
</tr>
<tr>
<td>( k_m )</td>
<td>18.9 Nm/rad</td>
</tr>
<tr>
<td>( b_m )</td>
<td>0.5 Nms/rad</td>
</tr>
<tr>
<td>( M )</td>
<td>0.002 kg/m²</td>
</tr>
</tbody>
</table>
The main idea of the delayed design based on the MID property is to force the quasipolynomial \( \Delta \) to have a root of a prescribed multiplicity, bounded by the quasipolynomial degree, 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 \( \alpha \) and \( \beta \) when the delay \( \tau \) 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 \( \Delta \) 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 \( \Delta \) becomes:

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

(13)

**Proposition 1.** The following assertions hold:

i) The multiplicity of a given root of \( \Delta \) is bounded by 4.

ii) The only admissible quadruple roots for \( \Delta \) are in the set \( s \in \{ -407.6856536, -22.86735033 \} \).

iii) The real number \( s_0 = -22.86735033 \) is a quadruple root of \( \Delta \) 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 \( \Delta \), 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 \( \Delta \) is equal to 6 and the Pólya and Szegö result from [27] asserts that 6 is the generic bound of the multiplicity of any root of \( \Delta \).

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

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

(14)

Next, to investigate potential roots with algebraic multiplicity 4, one substitutes the obtained identity \( \Delta \) in the ideal \( I_4 \) generated by the first three derivatives of \( \Delta \), i.e., \( I_4 = \langle \partial_4 \Delta, \partial_3^2 \Delta, \partial_3^3 \Delta \rangle \), which yields a manifold defined by three algebraic equations in four unknowns \( s, \alpha, \beta, \tau \). 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 \( \partial_3 \Delta = 0 \), 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:

\[
\Delta_e(s) = s^3 + 138 s^2 - 25050 s - 946890 \\
+ (207.1669924 s^2 + 35465.75084 s \\
+ 948985.4149 e^{-0.01059835599+\epsilon}) e^{-\tau s}
\]

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:

\[
- (207.1669924 \omega^2 - 948985.4149) \times \\
+ 35465.75084 \omega \sin(0.01059835599 \omega + \omega \epsilon) \\
- 946890 - 138 \omega^2 = 0, \\
35465.75084 \omega \cos(0.01059835599 \omega + \omega \epsilon) \\
+ (207.1669924 - 948985.4149 \omega^2) \times \\
\sin(0.01059835599 \omega + \omega \epsilon) - \omega^3 - 25050 \omega = 0
\]

Eliminating \( \cos(0.01059835599 \omega + \omega \epsilon) \) from the first trigonometric polynomial and feeding it into the second, we obtain the frequency \( \omega_c = 11.91836147 \). 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 P3δ 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 (P3δ for short), has been developed, see
Figure 3. The P3δ interface exhibiting (left) the spectrum distribution of $\frac{13}{13}$ and (right) the time-domain response of the muscle dynamics in the closed-loop system, in the presence of a quadruple spectral value for $\frac{13}{13}$ 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.

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 P3δ 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 MID property is exploited to model the CNS action and identify the critical delay inducing oscillations. The P3δ software is used to numerically illustrate the qualitative behavior of the muscle dynamics.

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