



Repetitive control of functional electrical stimulation for induced tremor suppression



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ARTICLE INFO

Article history:

Received 28 March 2015

Accepted 17 October 2015

Available online 14 November 2015

Keywords:

Repetitive control
Electrical stimulation
Assistive technology
Optimization
Vibration suppression

ABSTRACT

Tremor is an involuntary, oscillating, debilitating movement which affects over 50% of people with Multiple Sclerosis. In this paper an advanced paradigm, combining linearising action and repetitive control (RC), is developed to suppress tremor using functional electrical stimulation (FES) applied to wrist extensors/flexors. This innovative biomechanical approach to tremor suppression embeds learning from experience, and its effectiveness is confirmed in tests performed with nine healthy adults who attended a single one-hour session. Using FES, pathological wrist tremors (2.5, 3 or 4 Hz) were induced via extensor digitorum and a validated mechanical wrist-rig was used to collect data. Results confirmed statistically significant reduction in pathological movement, measured by path-length wrist movement and single peak amplitude of tremor.

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1. Introduction

Intention or cerebellar tremor is an oscillatory involuntary motion which is perpendicular to visually-guided voluntary movement and increases in amplitude as the limb approaches a specific target [1,2]. Intention tremor occurs more frequently in the distal joints of the upper limb such as the wrist and fingers [3] hampering sufferers' independence in activities of daily living (ADL) such as eating, drinking and dressing [4]. In neurological conditions such as Multiple Sclerosis (MS) over 50% present with some form of tremor which can physically handicap and embarrass people with MS (PwMS) [5] promoting social isolation and depression [6]. Available interventions for MS intention tremor include medication, surgical and conservative options. However, the efficacy of medication is not assured [3] and the more effective surgery, thalamotomy, has associated permanent complications [7,8]. Conservative physical management to manage tremor in the periphery includes weights on tremulous limbs [6,9] and cooling upper peripheral joints [10], but fatigue and slowness in performing tasks may ensue. As current strategies are suboptimal for treating distal pathological tremor, this motivates exploration of conservative options used in motor disorders of other neurologically-impaired groups [11].

A defect of the cerebellar feedforward control of voluntary movement has been implicated in the cause of intention tremor [12]. The pathological movement can be controlled mechanically by contract-

ing opposing muscles to preserve only the voluntary movement. Controlled muscle contraction can be generated by functional electrical stimulation (FES) [13], but for effective suppression, a mechanism is required to identify the pathological movement and adjust timing and level of FES applied to appropriate muscles. One approach is a full characterization of both voluntary movement and tremor using electromyographic, electroencephalographic and kinematic signals [14,15], however in this paper control approaches are considered which only use kinematic signals, due to their potential simplicity, usability and reduced cost. Such approaches typically employ feedback of joint movement, using high/bandpass filter-based compensation designed to cancel the phase and magnitude of the disturbance [16,17]. For example, [18] showed significant reduction in intention tremor about the elbow in six PwMS, and [19] applied an impedance-based FES approach using proportional plus integral (PI) action. However, classical feedback schemes have limitations including trade-offs between control effort, stability margins and tracking performance [20]. In addition, a compensator to reject disturbances at the tremor frequency will inevitably distort low frequency dynamics, and this will increase with the degree of attenuation. These issues are reflected by the poor levels of suppression that have been achieved, leading [18] to conclude that they did not warrant further research. An alternative approach was used in [21], based on co-contracting an antagonist pair in order to increase resultant stiffness and viscosity, achieving significant reduction in tremor amplitude. However, suppressing tremor via co-contraction inherently reduces system gain at low frequencies, locking the joint and thereby requiring more voluntary control effort to perform tasks, as well as fatiguing co-activated muscles. In contrast to the previous approaches, this

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paper develops a model-based controller that, in the nominal case, (i) achieves complete tremor suppression without excessive stimulation, and (ii) does not interfere with patients' voluntary movement dynamics.

A necessary condition for any system output to asymptotically converge to a desired reference trajectory (or reject a disturbance acting on the system) is that the controller contains an internal model of the reference/disturbance within its structure [22]. Repetitive control (RC) embeds an internal model within its structure to enable exact cancellation of a repeating disturbance, and has been shown to provide high performance when applied experimentally in the presence of model uncertainty and noise [23,24]. RC hence has potential for control of human muscle where effects such as fatigue and spasticity [25] degrade model accuracy [13].

The aim of this paper is to establish whether RC can improve on conventional feedback control of tremor. A further contribution is the development of a linearising controller and associated identification procedure that enables the full scope of linear control design and analysis to be applied to the inherently nonlinear wrist dynamics. In addition, this paper introduces the use of a high-pass filter in RC design to ameliorate low frequency dynamic distortion.

The paper is arranged as follows: Section 2 develops the biomechanical model of the system and the RC scheme. The experimental test procedure is presented in Section 3, with results described and analysed in Section 4. Discussion and conclusions are set out in Sections 5 and 6, respectively.

2. RC for tremor suppression

The complexity of models for the human arm and hand has led to consideration of a single degree of freedom in previous work on FES for tremor suppression [16,17,26]. In this paper the wrist is targeted as it can be confined to a single plane of motion (extension/flexion) and is clinically relevant as distal upper limb joints are most frequently affected [3]. To provide direct comparison, the same set-up as [19] is used, involving suppression of FES-induced tremor in healthy adults while preserving smooth voluntary single-plane wrist motion.

The mechanical component of the wrist dynamics comprises inertia, damping and stiffness, and is assumed to be actuated by two prime (key) muscles comprising Flexor Capri Radialis (FCR) and the Extensor Carpi Radialis (ECR) [19]. Muscle force is widely modelled as the product of three experimentally measured factors: the force-length property, the force-velocity property and the nonlinear muscle activation dynamics under isometric conditions. The latter behaviour dominates in smooth/slow motion, and is almost uniformly represented by a static nonlinearity in series with linear dynamics [27]. The linear dynamics can be accurately modelled as a critically damped second order system [28], so that the linear component of the human arm dynamics $P_1(s) =$

$$\frac{y}{M+d+v} = \frac{\omega_n^2}{s^2 + 2s\omega_n + \omega_n^2} \cdot \frac{1}{(2ma^2 + I)s^2 + bs + k} \quad (1)$$

with natural frequency ω_n , mass m , inertia I , damping b , stiffness k and distance to centre of mass a . Likewise, the static isometric recruitment curve (IRC) nonlinearities can be accurately captured by the form

$$\begin{aligned} M &= P_{nl}((u_{fcr}, u_{ecr})^\top) := h_{IRC,fcr}(u_{fcr}) - h_{IRC,ecr}(u_{ecr}) \\ &= c_{1,fcr} \left| \frac{e^{c_{2,fcr}u_{fcr}} - 1}{e^{c_{2,fcr}u_{fcr}} + c_{3,fcr}} \right| - c_{1,ecr} \left| \frac{e^{c_{2,ecr}u_{ecr}} - 1}{e^{c_{2,ecr}u_{ecr}} + c_{3,ecr}} \right| \end{aligned}$$

where u_{fcr} and u_{ecr} denote the stimulation applied to the FCR and ECR respectively, and $c_{i,fcr}$, $c_{i,ecr}$, $i \in \{1, 2, 3\}$ are scalar parameters [28]. This produces the structure shown in Fig. 1(a), where w is the muscle innervation signal, y is the joint angle, v is the voluntary action, and d is the tremor disturbance.

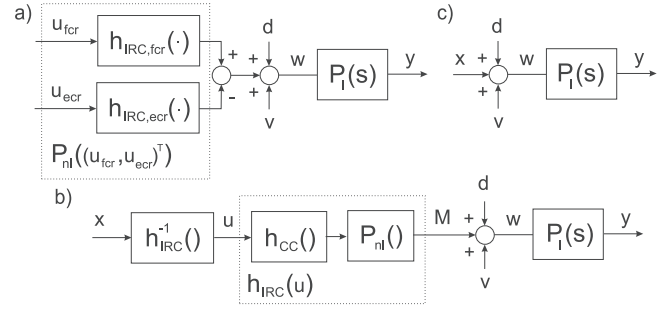


Fig. 1. (a) Biomechanical model, (b) additional controller functions $h_{IRC}^{-1}(\cdot)$, $h_{CC}(\cdot)$, (c) resultant linear system.

2.1. Linearising controller

Given the biomechanical system of Fig. 1(a), first define the coactivation function $h_{CC}(u(t))$ mapping a single control input $u(t)$ to the signals, $u_{fcr}(t)$, $u_{ecr}(t)$:

$$\begin{aligned} h_{CC}(u(t)) &:= (u_{fcr}(t), u_{ecr}(t))^\top : \\ u_{fcr}(t) &= \begin{cases} u(t) + u_{c,fcr}, & u(t) \in [0, 300 - u_{c,fcr}] \\ u_{c,fcr}, & u(t) \in [u_{c,ecr} - 300, 0] \end{cases} \\ u_{ecr}(t) &= \begin{cases} u_{c,ecr}, & u(t) \in [0, 300 - u_{c,fcr}] \\ u_{c,ecr} - u(t), & u(t) \in [u_{c,ecr} - 300, 0] \end{cases} \end{aligned} \quad (2)$$

where $u_{c,fcr}$ and $u_{c,ecr}$ are prescribed levels of co-activation for the FCR and ECR respectively. Function $h_{CC}(\cdot)$ is illustrated in Fig. 2(a). With the coactivation function in place, the summed muscle torque $M(t)$ can then be related to $u(t)$ by the static mapping

$$\begin{aligned} h_{IRC}(u(t)) &:= P_{nl}(h_{CC}(u(t))) \\ &= \begin{cases} h_{IRC,fcr}(u(t) + u_{c,fcr}) - h_{IRC,ecr}(u_{c,ecr}), & u(t) \in [0, 300 - u_{c,fcr}] \\ -h_{IRC,ecr}(u_{c,ecr} - u(t)) + h_{IRC,fcr}(u_{c,fcr}), & u(t) \in [u_{c,ecr} - 300, 0] \end{cases} \end{aligned}$$

Parameters $u_{c,fcr}$, $u_{c,ecr}$ are chosen such that $h_{IRC,fcr}(u_{fcr})$ is monotonically increasing for $u_{fcr} \geq u_{c,fcr}$, and likewise for ECR. If $u_{c,fcr}$ and $u_{c,ecr}$ are further selected such that $h_{IRC,fcr}(u_{fcr}) = h_{IRC,ecr}(u_{ecr})$, then mapping $h_{IRC}(u(t))$ is continuous, monotonic increasing and has a well-defined inverse. The co-activation has the effect of removing the dead zone inherent in each IRC, with an example shown in Fig. 2(b).

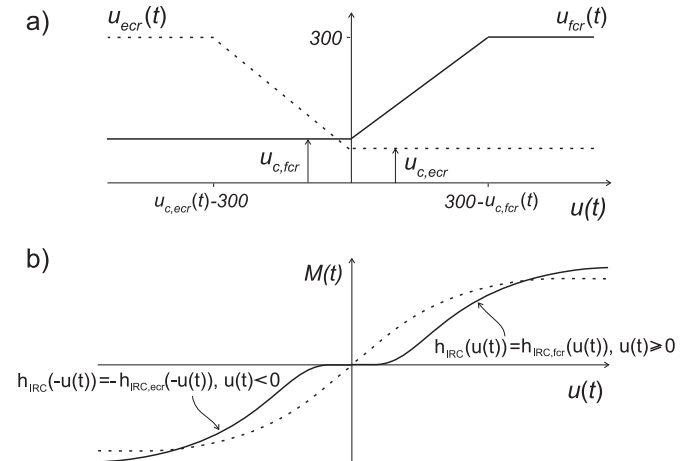


Fig. 2. Functional forms: (a) Co-activation mapping, $h_{CC}(\cdot)$, with parameters $u_{c,fcr}$ and $u_{c,ecr}$ chosen to remove respective IRC deadzones. (b) $h_{IRC}(u)$ function for cases: zero coactivation ($u_{c,fcr}, u_{c,ecr} = 0$) [solid line], and non-zero coactivation [dashed line].

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