Co-Contraction of Antagonist Muscles of Human Limb using Neural Network-based Control

Yasunori Kawai, Keita Ejiri, and Hiroyuki Kawai

Abstract—This paper considers co-contraction of antagonist muscles of human limb using neural network (NN)-based control. Some experimental results indicate that the antagonist muscle and agonist muscle work at the same time during the knee extension and flexion, then it is called co-contraction. However, control laws to adjust electrical impulses for the antagonist muscle by neuromuscular electrical stimulation (NMES) are almost not shown in previous researches. This paper proposes that the control of agonist muscle and antagonist muscle is controlled independently. Moreover, NN-based control is applied to control of antagonist muscle, because the learning components is needed for the unknown disturbances. The set-point regulation control and trajectory tracking control are implemented with the no co-contraction and NN-based co-contraction. It is shown that the co-contraction is useful to the convergence of the joint angle by the attenuation of the overshoot.

I. INTRODUCTION

Neuromuscular electrical stimulation (NMES) which is also called functional electrical stimulation (FES) is used to improve the motor function of human. As the similar way as electrical impulses from the brain, NMES makes muscles contraction by causing electrical impulses, and if the stimulation is controlled, a desired movement can be done [1].

The human limb can be moved by pairs of muscles, the muscles are composed of the agonist muscle and antagonist muscle. Actually, the human limb can be moved by only the agonist muscle due to electrical stimulus. However, some experimental results indicate that the antagonist muscle and agonist muscle work at the same time [2], [3], it is called co-contraction or coactivation. This co-contraction mechanism may be important for the stability of the knee joint. In [2], the amount of antagonist co-activation and the resultant moment of force generated by the hamstring muscles during maximal quadriceps contraction in slow isokinetic knee extension were quantified. In [3], to elucidate how able-bodied individuals use co-contraction by analyzing the electromyogram (EMG) activity of antagonist muscles during voluntary knee extension against the gravity was shown.

However, control laws to adjust electrical impulses for the antagonist muscle by NMES are almost not shown in previous researches. In [4], optimal stimulation patterns via an inverse model by taking the muscle contraction dynamics into account are proposed. In [5], [6], the antagonist muscle force in the co-contraction is given as a constant and variable ratio of agonist muscle force, simulation and experimental results are shown. It is shown that the co-contraction is useful to the convergence of the joint angle. In [7], the role of the co-contraction is shown by using four muscles.

This paper considers co-contraction of antagonist muscles of human limb using neural network (NN)-based control. In [6], it is indicated that the difference between the co-contraction and no co-contraction is small. This paper proposes that the control of agonist muscle and antagonist muscle is controlled independently. Moreover, NN-based control is applied to control of antagonist muscle, because the learning components is needed for the unknown disturbances. In [8], the effectiveness of NN-feedforward for control of the agonist muscle is shown. The knee extension and flexion movements are implemented by NMES. In this paper, the set-point regulation control and trajectory tracking control are implemented with the co-contraction and NN-based co-contraction. It is shown that the co-contraction is useful to the convergence of the joint angle in transient states.

The organization of this paper is as follows. The human limb model with co-contraction is shown in Section 2. The control system design by RISE control and NN-based control is indicated in Section 3. In Section 4, experimental results are shown. Finally, we present our conclusions.

II. HUMAN LIMB MODEL WITH CO-CONTRACTION

The knee-joint model of the human limb is shown as Fig. 1. The dynamics of total knee-joint is modeled in [9] as

\[
\begin{align*}
J\ddot{q}(t) + k_1 e^{-k_2 q(t)} - k_3 + mgl \sin(q(t)) & = M_f
\end{align*}
\]

where \(q(t), \dot{q}(t), \ddot{q}(t) \in R\) are the joint angles, velocities, and accelerations, respectively. \(J \in R\) is the unknown inertia of the combined shank and foot, \(m \in R\) is the unknown combined mass of the shank and foot, \(l \in R\) is the unknown distance between the knee-joint and lumped center of mass of the shank and foot, and \(g \in R\) is gravitational acceleration. \(k_1, k_2, k_3 \in R\) are unknown positive coefficients, \(B_1, B_2, B_3 \in R\) are unknown positive constants.

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1. Y. Kawai is with Department of Electrical Engineering, National Institute of Technology, Ishikawa College, Kitacyujo, Tsubata, Ishikawa 929-0392, Japan kawai@ishikawa-nct.ac.jp
2. K. Ejiri is with Advanced Course of Electronic and Mechanical Engineering, National Institute of Technology, Ishikawa College, Kitacyujo, Tsubata, Ishikawa 929-0392, Japan
3. H. Kawai is with Department of Robotics, Kanazawa Institute of Technology, Ishikawa 921-8501, Japan
In Eq. (1), $M_I$ is the inertial effects of the shank-foot complex about the knee joint, $M_c$ is the elastic effects due to joint stiffness, $M_g$ is the gravitational component, $M_v$ is viscous effects due to damping in the musculotendon complex, $\tau_d \in R$ is considered as an unknown bounded disturbance which represents an unmodeled reflex activation of the muscle (e.g., muscle spasticity) and other unknown unmodeled phenomena (e.g., dynamic fatigue, electromechanical delays), and $\tau \in R$ is the torque produced at the knee joint [9].

As shown in Fig. 1, a couple of the antagonistic bi-articular muscle $e_3$ and $f_3$, and the mono-articular muscle $e_2$ and $f_2$ are attached, $e_2$ and $e_3$ mean extensor muscles, $f_2$ and $f_3$ are flexor muscles, where $e_2$ is vastus medialis, $e_3$ is rectus femoris, $e_2$ and $e_3$ are also called as quadriceps femoris, $f_2$ is short head of biceps femoris muscle and $f_3$ is hamstring muscles. In this paper, $e_2$ and $e_3$ are stimulated together with electrical stimulus by using an electrode pad at the same time. In a similar way, $f_2$ and $f_3$ are also stimulated by an electrode pad at the same time.

The muscle contraction force are defined as follows [10], [11]

$$u_e = K_{an,e}(q) \cdot K_{ve,e}(\dot{q}) \cdot V_e,$$  \hspace{1cm} (2)
$$u_f = K_{an,f}(q) \cdot K_{ve,f}(\dot{q}) \cdot V_f,$$  \hspace{1cm} (3)

where $K_{an,e}(q)$, $K_{an,f}(q)$, $K_{ve,e}(\dot{q})$, and $K_{ve,f}(\dot{q})$ indicate muscle contraction models, $V_e$ and $V_f$ are the force by extensor muscles and flexor muscles, respectively. The relations between angles and the force ratio is represented as $K_{an,e}(q)$ and $K_{an,f}(q)$. It is assumed that they are defined as

$$K_{an,e}(q) = K_{an,f}(q) = e^{-\left(\frac{q_1-q_2}{l_2}\right)^2}. \hspace{1cm} (4)$$

$K_{ve,e}(\dot{q})$ and $K_{ve,f}(\dot{q})$ are relations between velocities and the force ratio. The relations are assumed to be

$$K_{ve,e}(\dot{q}) = -K_{ve} \dot{q} + 1,$$  \hspace{1cm} (5)
$$K_{ve,f}(\dot{q}) = K_{ve} \dot{q} + 1,$$  \hspace{1cm} (6)

$K_{an,e}(q)$, $K_{an,f}(q)$, $K_{ve,e}(\dot{q})$, and $K_{ve,f}(\dot{q})$ are illustrated in Fig. 2-3.

During the knee extension, the contraction force $u_e$ is needed in order to move the leg to upper direction. However, the antagonistic muscle contraction force $u_f$ works at the same time [2], [3], it is called co-contraction. Then the torque for lower direction works in the leg by the contraction force $u_f$. The joint torque $\tau(t)$ is given as the difference of the agonist force and the antagonist force

$$\tau(t) = (u_e - u_f)l_p,$$  \hspace{1cm} (7)

where $l_p$ represent radius of the joint as shown in Fig. 4.

Substituting (2)-(6) into the joint torque (7), the joint torque becomes

$$\tau(t) = (K_{ve,e} V_e - K_{ve,f} V_f) l_p e^{-\left(\frac{q_1-q_2}{l_2}\right)^2} \cdot (K_{ve} \dot{q} + 1) V_e - (K_{ve} \dot{q} + 1) V_f) l_p e^{-\left(\frac{q_1-q_2}{l_2}\right)^2}.$$

$$\tau(t) = \{V_e - V_f\} K_{ve} l_p e^{-\left(\frac{q_1-q_2}{l_2}\right)^2}.$$  \hspace{1cm} (8)

From the joint torque (8), muscle contraction model could be indicated as a mass dumper model as shown in Fig. 4. The block diagram of Eq. (8) is represented as Fig. 5. In Eq. (8), increasing the co-contraction $V_f$, the dumper term $K_{ve} \dot{q}(V_e + V_f)$ acts for stability, then the joint stiffness is increased. However, the contractile torque $(f_e - f_f)$ decreases. As a result, the more large torque $\tau$ is needed to compensate $-V_f$ in the agonist muscle.
The discrete time of the backpropagation algorithm of $W_1$ is

$$W_1(k+1) = W_1(k) - \sum_{i=1}^{3} \eta_i (x(k) \cdot \hat{\sigma}^T(W_1x) \cdot W_2(k))^T E_i(k),$$

where $\eta_i \in \mathbb{R}$ ($i = 1, 2, 3$) is positive definite design parameter matrices governing the speed of convergence of the algorithm, the error $E_i \in \mathbb{R}$ ($i = 1, 2, 3$) is defined as

$$E_1 = \begin{bmatrix} q_d - q \\ \dot{q}_d - \dot{q} \\ \ddot{q}_d - \ddot{q} \end{bmatrix},$$

$$E_2 = \begin{bmatrix} q_d - q \\ \dot{q}_d - \dot{q} \end{bmatrix},$$

$$E_3 = \begin{bmatrix} \ddot{q}_d - \ddot{q} \end{bmatrix}.$$ (16)

The another backpropagation algorithm of $W_2$ is

$$W_2(k+1) = W_2(k) - \sum_{i=1}^{3} \eta_i \cdot \sigma^T(W_1x) \cdot E_i(k).$$ (17)

Thus, NN has two layers of weights.

The joint torque Eq. (8) can be represented as

$$\tau(t) = \tau_s \left(\frac{x}{x_2 K_2}\right)^2 [K_{ve\theta_x} K_{ve\theta}] \begin{bmatrix} V_e \\ -V_f \end{bmatrix},$$

$$= \Omega V,$$ (18)

where

$$\Omega = \tau_s \left(\frac{x}{x_2 K_2}\right)^2 [K_{ve\theta_x} K_{ve\theta}],$$

$$V = \begin{bmatrix} V_e \\ -V_f \end{bmatrix}^T.$$ (19)

$\Omega > 0$, $V_e$ and $V_f$ are the control inputs of agonist muscle and agonist muscle as shown (11) and (12), respectively. It is assumed that $V_e$ and $V_f$ work when $V_e > 0$ and $V_f > 0$, respectively. The feedback control system of the human limb with the co-contraction of antagonist muscles is shown in Fig. 6. The system is composed of the segmental dynamics (1), co-contraction and muscle contraction model (18), RISE controller (11), and NN-based controller (12).

For the stability, this paper don’t show the proof. However, the closed-loop system with RISE controller can guarantee that the tracking error $e_1$ is asymptotically stable on the equilibrium point [9]. The NN-based controller with RISE controller is used in [8] for only the agonist muscle, the positive definite function including NN weights is proposed. However, assumptions $V_e > 0$, $V_f > 0$ exist in this paper, the proof of stability will be considered in the future work.
IV. EXPERIMENTS

The knee extension and flexion movements by the proposed RISE control and NN-based control method are verified. Fig. 7 shows the experimental equipment, the human sits on the leg extension machine, two pairs of electrode pad are set on the muscle of the human leg. The encoder is set on the leg extension machine in order to measure the joint angle of human leg. A weight 5LBS(=2.3 [kg]) is attached in the leg extension machine.

![Experimental equipment](image)

The block diagram of control of human limb is indicated in Fig. 8. The joint angle of the human leg can be measured by using the encoder which is set on the leg extension machine. The encoder is connected to the encoder port on the signal processing board DS1104 (dSPACE). The signal processing board is also connected to the PC (Personal Computer). In PC, the control input $V_e$ and $V_f$ are calculated by using the information of the joint angle $q$ from the encoder and references $q_d$. The angle velocity $\dot{q}$ and acceleration $\ddot{q}$ are calculated by using low path filter in PC instead of measurement. The control input $V_e$ and $V_f$ are sent to the electrical stimulus machine RehaStim (HASOMED Gmbh). In the RehaStim, the control input $V_e$ and $V_f$ are transformed into actual electrical stimulus signals. The signals are sent to two pairs of electrode pad, then the muscle contraction is caused.

![Block diagram](image)

The parameters of RISE controller are designed as $k_e = 9.1$, $\alpha_1 = 4.25$, $\alpha_2 = 0.525$, $\beta = 3$. The parameters of NN-based controller are designed as $\eta_1 = 2.5 \times 10^{-2}$, $\eta_2 = 2.5 \times 10^{-3}$, $\eta_3 = 2.5 \times 10^{-4}$. The initial condition of weight $W_1$ and $W_2$ are given as random number. All parameters of RISE and NN-based controller are decided by the tuning from experimental results. The initial conditions of joint angles are given as $q(0) = 0$ [rad], $\dot{q}(0) = 0$ [rad/s].

A male in his 30s participated in this trial. The participant give the informed consent under the supervision of the Institutional Review Board (IRB) at National Institute of Technology, Ishikawa College.

A. Set-point regulation control

The set-point regulation control are shown in Figs. 9-13. The reference is selected as

$$q_d(t) = \begin{cases} 0 \text{[rad]} & (0 \leq t < 3 \text{[s]}) \\ \frac{\pi}{2} \text{[rad]} & (3 \leq t < 10.5 \text{[s]}) \\ \frac{\pi}{4} \text{[rad]} & (10.5 \leq t < 15.5 \text{[s]}) \\ \frac{\pi}{8} \text{[rad]} & (15.5 \leq t < 23 \text{[s]}) \\ 0 \text{[rad]} & (23 \leq t \text{[s]}) \end{cases}$$

In Figs. 9, 11, dashed line is $q_d$, solid line is $q$. Figs. 9-10 show the no co-contraction ($V_f = 0$), Figs. 11-13 show the co-contraction.

From the comparison between Fig. 9 and Fig. 11, the effect of damping for about $t = 4 - 7$ [s], $11.5 - 13$ [s], $17 - 20$ [s] appear in Fig. 11. The control input of antagonist $V_f$ works as shown in Fig. 13. Moreover, $V_f$ support to move the limb downward for $t = 15.5$ [s], $23$ [s]. Because $V_e$ in Fig. 10 almost looks like same as $V_e$ in Fig. 12, we can indicate the effect of co-contraction by NN. Note that though the magnitude of $V_f$ is smaller than $V_e$, the force $V_f$ for hamstring muscle is smaller than the force $V_e$ for quadriceps femoris, the electrical stimulus cause a pain in case of the same magnitude of control input of $V_e$.

![Joint angle](image)

![Control input](image)
Fig. 11. Joint angle with co-contraction of set-point regulation control (dashed: reference $q_d$, solid: joint angle $q$).

Fig. 12. Control input $V_e$ with co-contraction of set-point regulation control.

Fig. 13. Control input $V_f$ with co-contraction of set-point regulation control.

**B. Trajectory tracking**

The trajectory tracking control are shown in Figs. 14-18. The reference $q_d$ is selected as

$$q_d(t) = \begin{cases} 0 & (0 \leq t < t_1) \\ \frac{\pi}{2} + \frac{\pi}{2} \sin\left(\omega t' - \frac{\pi}{2}\right) & (t_1 \leq t < t_2) \\ \frac{\pi}{2} + \frac{\pi}{2} \sin\left(\omega t' - \frac{\pi}{2}\right) & (t_2 \leq t < t_3) \\ \frac{\pi}{2} + \frac{\pi}{2} \sin\left(\omega t' - \frac{\pi}{2}\right) & (t_3 \leq t < t_4) \\ 0 & (t_4 \leq t) \end{cases} ,$$

$$\omega t' = 2\pi f (t - t_1), \quad f = 1/2.5 \text{ [Hz]}, \quad t_1 = 3 \text{ [s]},$$

$$t_2 = t_1 + 1.25, \quad t_3 = t_2 + 18.75, \quad t_4 = t_3 + 1.25.$$  

In Figs. 14, 16, dashed line is $q_d$, solid line is $q$. Figs. 14-15 show the no co-contraction ($V_f = 0$), Figs. 16-18 show the co-contraction. For $18 - 23 \text{ [s]}$, it can be shown that the overshoot of the maximal value of joint angle $q$ is attenuated in Fig. 16 by using the antagonist muscle. Fig. 18 indicates that $V_f$ is adjusted by NN-based control.

Therefore, the co-contraction by NN-based control is useful to the convergence of the position tracking error $e_1$ by the attenuation of the overshoot in transient states.
This paper considered the co-contraction of antagonist muscles of human limb using NN-based control. This paper proposed that the control of agonist muscle and antagonist muscle is controlled independently. RISE control and NN-based control was applied to the control of agonist muscle and antagonist muscle, respectively. The knee extension and flexion movements were implemented by NMES. The set-point regulation control and trajectory tracking control were implemented with the no co-contraction and NN-based co-contraction. It was shown that the co-contraction is useful to the convergence of the joint angle by the attenuation of the overshoot.

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