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Research Article

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The costs of position, velocity and force requirements in optimal control induce triphasic muscle activation during reaching movement

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Abstract

The nervous system activates a pair of agonist and antagonist muscles to determine the muscle activation pattern for a desired movement. Although there is a problem with redundancy, it is solved immediately, and movements are generated with characteristic muscle activation patterns in which antagonistic muscle pairs show alternate bursts with a triphasic shape. To investigate the requirements for deriving this pattern, this study simulated arm movement numerically by adopting a musculoskeletal arm model and an optimal control based on the minimization of neural input. The simulation reproduced the triphasic electromyogram (EMG) pattern observed in a reaching movement using a cost function that considered three terms: end-point position, velocity, and force required. The first, second and third bursts of muscle activity were generated by the cost terms of position, velocity and force, respectively. Thus we concluded that the costs of position, velocity and force requirements in optimal control can induce triphasic EMG patterns. Therefore we suggest that the nervous system may control the body by using

an optimal control mechanism that adopts the costs of position, velocity and force required, which serve to initiate, decelerate and stabilize movement, respectively.

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Introduction

Mammalian biomechanical motor control comprises multiple joints and muscles that form redundant systems with multiple degrees of freedom. To move the nervous system must overcome the problem of redundancy and determine a movement trajectory and muscle activation pattern that involves pairs of agonist and antagonist muscles. The muscle activation pattern (i.e. the electromyogram, EMG) is equivalent to other movements loaded by an external force when the movements are identical kinematically and the dynamics are well learned, even if the amount of muscle activation is increased¹. In both single-joint and multi-joint reaching movements, the agonist and antagonist muscles typically burst alternately in triphasic patterns^{2,3}. Thus the muscles seem to be tuned selectively in accordance with the movement direction, similar to isometric force production⁴. Then the agonist and antagonist muscles are activated alternately in a triphasic pattern (Figure 1). An agonist muscles are strongly activated at movement onset (Figure 1, AG1). Next, the antagonist muscles show single peaks at the midpoint of the movement (Figure 1, ANT). The timing is synchronized with the peaks in the velocity profiles. Subsequently, the agonist muscles are activated again, albeit weakly (Figure 1, AG2). The AG1 and ANT burst pair is thought to determine the increase and decrease in acceleration, respectively, and the AG2 burst increases deceleration⁵ or dampens the oscillations that might occur at the end of the movement⁶. Thus the triphasic EMG pattern does not relate directly to the movement amplitude, speed or duration, but to the acceleration and deceleration⁷. Such a simple, general relation between the EMG and movement implies that the nervous system can simply determine the muscle activation patterns needed to produce movements with desired

characteristics ⁸. Indeed single-neuron and population-level activity in the primary motor cortex (M1) also show triphasic changes in temporal pattern and instantaneous directionality similar to the EMG ^{9,10}, although it is thought that the neurons represent the speed and direction of hand motion ¹¹.

Figure 1. Triphasic muscle activation pattern during reaching movement. AG1, AG2 and ANT indicate the sequence of bursts for the pair of agonist (AG1 and AG2) and antagonist (ANT) muscles.

In the computational motor control domain, optimal feedback control (OFC) theory has been proposed ¹²⁻¹⁴, which predicts not only various movement phenomena, such as obstacle avoidance ¹⁵, adaptation to novel tasks ¹⁶, stiffness modulation ¹⁷ and the manipulation of complex objects ¹⁸, but also neural representation in M1 ¹⁹. An optimal control framework using an OFC-like cost function based on minimization of neural input can also reproduce muscle co-contraction depending on the task; this is impossible using previous models ^{20,21}, although most motor-planning models, such as the minimum-jerk ²² and minimum-torque-change ²³ models, are thought to be unable to predict such specific muscle actions ²⁴. In neural studies of primates, M1 may provide a neural substrate for integrating shoulder and elbow motion information into joint torque

for fast feedback control ^{25,26}. The neural activity in M1 is thought to be optimized for the musculoskeletal structure using an OFC-like cost function ²⁷⁻²⁹. However, it has been suggested that optimal control cannot predict the EMG patterns in wrist movement, despite the fact that the patterns are produced to minimize end-point variance, rather than neural input ³⁰. On the other hand, some studies have evaluated several cost functions to generate a point-to-manifold reaching task that leaves the target underdetermined ³¹⁻³³. Although they suggested the reaching trajectories are generated by the optimality principle with compositions of multiple costs including kinematic smoothness and mechanical energy consumption, it has been remained the question which cost function can predict the EMG patterns in arm movement.

In this study, we re-examined the triphasic muscle activation patterns observed in EMGs during arm movement from the perspective of optimal control using an OFC-like cost function that consisted of terminal requirement costs with minimized neural input to predict the muscle activation patterns. Then we performed numerical simulations applying an iterative linear–quadratic–Gaussian (ILQG) method ³⁴, which approximates OFC, to physiological arm dynamics with a realistic muscle model. This showed that the optimal control could selectively tune muscles in accordance with the movement direction and indicated an interlaced cost based on combinations of the terminal requirement of the end-point position, velocity and force under minimization of neural input during movement. The control induced triphasic muscle activation patterns like those in the recorded EMGs, only under certain conditions validating the position, velocity and force costs. Thus each cost corresponded to each burst of the agonist and antagonist muscles (i.e. AG1, ANT and AG2). Consequently, we suggest that the neural system controls the body by using an optimal control mechanism based

on a cost function that consists of position, velocity and force requirements, and that they appear to correspond to the first (AG1), second (ANT) and third (AG2) muscle activation bursts, which serve to initiate, decelerate and stabilize the movement, respectively.

Results

We simulated movement using an approximate OFC ³⁴ and a two-joint six-muscle arm model ^{17,35,36} (i.e. shoulder flexor (SF), shoulder extensor (SX), elbow flexor (EF), elbow extensor (EX), biarticular flexor (BF) and biarticular extensor (BX) muscles). We set the simulation duration to 500 ms and determined the terminal time of the movement at 400 ms to fit the experimental data ^{3,17,37}. Then we assumed four situations involving the various cost weights in equation (9) as Cases 1–4. In Case 1, the cost weights were zero, except the positional cost. The movement was constrained only in the terminal position. In Case 2, the cost weight of the end-point force was zero. This required movement under kinematic constraints. In Case 3, the cost weight of the end-point velocity was zero to inhibit the end-point force at the target position. Finally, Case 4 required the regulation of the end-point position, velocity and force states at the movement end. The task required stopping at the target without force generation. They showed that the hand pathways varied slightly, forming curves or almost-straight lines in all cases in accordance with the direction (Figure 2, left column).

Figure 2. The centre-out movement for 16 targets. The columns from left to right are the hand paths, hand speed profiles, root mean squared hand force profiles and joint torque profiles. In the hand paths, the target positions are shown as filled circles. In the joint torque profiles, solid and dotted lines indicate the torques of the shoulder and elbow joints, respectively. Cases 1–4 are in rows (a)–(d), respectively.

Movement parameters

In Case 1, the hand passed through on the targets and the speed did not converge on zero at the time of the movement end (i.e. 400 ms); the joint torques peaked at the onset of movement to generate the hand force triggering the movement (Figure 2a). Then the joint torques and hand force diverged slightly at the movement end because a passive tensile force was generated by stretching the muscles. In Case 2, the hand stopped on the targets and the speed was almost set to zero at the movement end, although they had slight motions after the movement end (Figure 2b). The hand force and joint torques showed biphasic peaks. The second peak of the joint torques was generated in the direction opposite the movement, to decelerate the movement and set the hand speed to zero at the movement end. However, the hand force and joint torques did not converge on zero at the movement end but gradually decreased after then. In Case 3, the hand passed through on the targets and the speed was not zero at the movement end time, similar to Case 1, and the hand force and joint torques converged on zero at this time in contrast to Case 1 (Figure 2c). In Case 4, the hand stopped on the targets and the speed was close to zero with gradual curves and formed clear bell-shaped profiles (Figure 2d). Although the hand force and joint torques showed biphasic peaks similar to Case 2, they converged on zero at the movement end.

Muscle activation patterns

Although the muscles were activated selectively in accordance with the movement direction, regardless of the case following the first muscle activation (AG1), the second (ANT) and third (AG2) bursts of the muscles varied temporally among the cases (Figure 3).

Figure 3. Muscle activation patterns plotted as a function of time and target direction. Cases 1–4 are shown in (a)–(d), respectively. The values were normalized using the highest activation level in each muscle across all cases.

In Case 1, agonist muscles were activated once to initiate the movement (Figure 3a). Then the monoarticular elbow muscles EF and EX showed no activation. In Case 2, the activation of the agonist and antagonist muscles alternated like a burst of AG1 and ANT, occurring immediately after the beginning and at the end of the movement, respectively (Figure 3b). The antagonist muscles might contribute the torque in the direction opposite to that of the movement to decelerate the movement. However, EF and EX were activated only as the antagonist muscles synchronizing with the biarticular muscles BF or BX. In Case 3, the agonist muscles were activated twice, like bursts of AG1 and AG2 (Figure 3c). The second activations might suppress the development of

the hand force and joint torques at the end of the movement. Then muscles EF and EX had weak activation to supplement BF or BX. In Case 4, the muscle activations showed a triphasic pattern resembling the superposition of those in Cases 2 and 3, with the agonist and antagonist muscles activated alternately, although the agonist muscles were occasionally activated twice (Figure 3d). Of note, when the SF muscle acted as an agonist muscle (e.g. 90° movement direction), it was activated twice, at the start and end of the movement, while it was activated only once in the middle of the movement when it was an antagonist muscle (e.g. 270° movement direction). The later activation of agonist muscles was thought to contribute to stabilization of the movement because the later torque observed in Case 2, which the antagonist muscles generated, may induce unnecessary movement after reaching the target. Thus, the additional torque generated by the agonist muscles was required to counteract the opposite torques and stabilize the movement after completing the task.

Stabilization control

We suggested that the force cost reactivates the agonist muscle to counteract the breaking torques, for stabilization. An alternate hypothesis is that to achieve a reaching movement in a certain time, it is necessary to optimize the stability of the terminal kinematic state (i.e. position, or position and velocity) after the movement phase but not to minimize only the terminal cost at the movement end. To examine this hypothesis, we define the extra two cost functions for stabilization control as equation (11).

As a result, although the cost function in equation (11) showed the biphasic muscle pattern seen in Case 2 (Figure 4c), the cost function in equation (12) reproduced the triphasic muscle pattern seen in Case 4 (Figure 4d). Accordingly, we could not reject

the hypothesis of the stabilization control represented by equation (12). However, the stabilization control may regulate a differential of the cost terms, because the stabilization of equation (11), which adopts only the position cost, decreased the hand speed similar to Case 2 (Figure 4a). Therefore we postulate the stabilization control of equation (12) suppressed hand acceleration during the stabilization period (Figure 4c), and it played a role like the force cost in Case 4.

Figure 4. Stabilization control. The controls of only the position cost, and a pair of the position and velocity costs are shown in (a, c) and (b, d), respectively. (a, b) The centre-

out movement for 16 targets. The format is the same as in Figure 2. (c, d) Muscle activation pattern. The format is the same as in Figure 3.

Discussion

The purpose of this study was to investigate the requirements for deriving muscle activation patterns in which antagonistic muscle pairs show alternate bursts with triphasic shapes. To examine it, we carried out simulations of arm movement to apply four types of cost function that considered the end-point position, velocity and force requirements under minimization of the neural input. To summarize the results, the position, velocity and force costs were postulated to play following roles: (i) the position cost led to activation of the first agonist muscle, to trigger movement; (ii) the velocity cost activated the antagonist muscles and generated braking torques to decelerate the movement; and (iii) the force cost reactivated the agonist muscle to negate the braking torques and the passive tensile force of the stretched muscles, for stabilization.

Our control methodology was not truly optimal, but approximately optimal, since solving the optimal control problem analytically for nonlinear systems such as the musculoskeletal system is difficult. According to the original OFC model for a linear dynamics plant whose state variable $\mathbf{x}(t)$ comprises the position, velocity and force ¹⁴, the motor command is represented as $\mathbf{u}(t) = \mathbf{K}(t) \cdot \hat{\mathbf{x}}(t)$, where $\mathbf{K}(t)$ is the feedback gain and $\hat{\mathbf{x}}(t)$ is the estimated state integrating the prediction of the internal model with the sensory feedback. The components of the feedback gain for the position, velocity and force peak at varied timings according to different cost functions corresponding to

Cases 1–4 (Figure 5). They show a peak at simultaneous timing in Case 1, twice peaks in Case 2 and Case 3, and the position, velocity and force peak in turn in the early, mid and late phases of the movement, respectively. Thus the position, velocity and force feedback gains serve to initiate, decelerate and stabilize the movement, respectively, which fits our suggestion regarding the roles of the cost terms.

Figure 5. Typical feedback gains of the optimal feedback control (OFC) model for a linear system. Each of Case 1–4 corresponds to the same formation of the cost function above results. Each value of the position, velocity and force gains is normalised by that of Case 4.

It seems that the force cost can be replaced with an acceleration term. Although the acceleration term actually could reproduce the triphasic muscle pattern shown in Case 4, it does not seem natural to use the acceleration cost instead of the force. The muscle spindles detect the muscle length and velocity, and the Golgi tendon organ provides the muscle tension. However, the acceleration cannot be gained directly from proprioceptive systems. A previous study reported producing the same muscle pattern to solve an optimal control problem of muscles with boundary conditions, which velocities

and forces were zero at the beginning and end of the movement ³⁸. Thus we postulate that the neural system adopts not the acceleration, but the force.

Proprioceptive feedback is not essential to generate the triphasic pattern ⁶, although it contributes to achieving precise movements. Thus the triphasic pattern of muscles might be programed in the motor planning phase. In the OFC model, the neural system is also assumed to compute the feedback gain before movement onset, and execute the feedback control for estimated states. The common point implies that the muscle activation pattern serves as feedback gain reflecting muscle properties such as stiffness and viscosity.

Several studies in patients with motor disorders have reported that the basal ganglia may have a role in scaling the size of AG1, reinforcing the voluntary command and inhibiting inappropriate EMG activity ⁶. Therefore we assume that in the basal ganglia module, the positional cost weight w_p is related to AG1. The cerebrum may regulate the other cost weights w_v and w_f to balance w_p , because the cerebellum has a role in timing the voluntary bursts of ANT and AG2. Indeed, cerebellar patients are unable to perform accurate movements ³⁹. This deficit is known as dysmetria, which is a lack of coordination of movement that results in overshooting or undershooting a target during reaching tasks.

In previous studies that used similar muscle models ^{28,34}, the tensile force of the passive elastic component was excluded from the muscle model (i.e. F_{PE2} in equation (8)), although the force is not small. We assume that this was done because of the sensitivity of parameter selection for the muscle model, such as the moment arms, optimal muscle lengths and optimal joint angles, which affect the muscle length and generate a tensile force without muscle activation. In the model, the tensile force

requires initial muscle activation to maintain the initial position before movement onset, and this affects the hand trajectories, causing marked distortion. Thus we set the initial hand position to an equilibrium point balancing the forces, to set the initial muscle activations to zero. With such sensitive effects, how do we generate and control movement? Previous studies suggested that the neurons are optimized for physical mechanics^{19,27,28}. However, if subjects learned fine motor tasks once, such learning tend to persist, even when clearly suboptimal⁴⁰. Subsequently, the subjects learn to overcome real and virtual changes in their biomechanics, but prefer rescaling their prior motor habits to recomputing to optimize the control policy⁴¹. The prior motor habits may be learned from muscle activation patterns generated by lower sensorimotor circuitry that is functionally suboptimal. However, we ignored the lower sensorimotor circuitry, which is a limitation of our model. Recently, a model of the spinal circuitry and musculoskeletal system was developed⁴², and seems to be useful for studying habitual motor control systems.

Dehghani and Bahrami suggested that arm movements are planned with some principle patterns of muscle synergies, and the plans are divided into few phases to reduce the dimension of the control space^{43,44}. Also, Sakaguchi et al. have proposed a computational model that brain adaptively divides the continuous-time axis into discrete segments and executes feedforward control in each segment to allow sensorimotor delays⁴⁵. However, the OFC could realize the control of the muscle synergies through feedback gains, and the segmentation of motor execution may be identified as some steady-state feedback gains computed by a model predictive control under the framework of the OFC¹⁹.

In summary, the results of this study imply that a triphasic muscle activation pattern can be predicted by an optimal control mechanism, e.g. by adopting an OFC-like cost function. Furthermore, the costs of position, velocity and force requirements may be the critical parameters for the physiological control of movements and correspond to the triggering, braking, and stabilizing of movement, respectively.

Methods

We simulated arm movements using the ILQG method³⁴ together with a two-joint six-muscle arm model^{17,35,36}. The ILQG method constructs an affine feedback control law by minimizing a quadratic approximation to optimize a cost function. The muscles were modelled by nonlinear dynamics reflecting the force–length and force–velocity relationships of mammalian skeletal muscle⁴⁶. We examined a centre-out reaching movement task in this study. The hand position at the start of movement was located 22 cm in front and 8 cm to the right of the shoulder, which is nearly equal to an equilibrium point balanced by passive elastic muscle forces. Then, 16 targets were aligned 8 cm away from the initial hand position. All of the simulations in this paper used simple Euler integration with a 5-ms time step for 500 ms, and we defined the movement duration as 400 ms according to the experimental measurement in monkeys^{3,17,37}.

Musculoskeletal arm model

We considered the monkey’s arm to be a two-joint arm composed of the shoulder and elbow joints. The joint angles were defined as vector $\boldsymbol{\theta} = [\theta_1, \theta_2]^T$, where θ_1 and θ_2

indicate the shoulder and elbow variables, respectively. Suppose joint torque $\boldsymbol{\tau} \in \mathbb{R}^2$, the dynamics of the monkey's arm in horizontal plane are denoted by

$$\boldsymbol{\tau} = \mathbf{M}(\boldsymbol{\theta}) \cdot \ddot{\boldsymbol{\theta}} + \mathbf{C}(\boldsymbol{\theta}, \dot{\boldsymbol{\theta}}) + \mathbf{D} \cdot \dot{\boldsymbol{\theta}}, \quad (1)$$

where $\mathbf{M}(\cdot) \in \mathbb{R}^{2 \times 2}$, $\mathbf{C}(\cdot) \in \mathbb{R}^2$, and $\mathbf{D} \in \mathbb{R}^{2 \times 2}$ are the inertia matrix, Coriolis force vector, and viscosity matrix, respectively, and are given by

$$\begin{aligned} \mathbf{M}(\boldsymbol{\theta}) &= \begin{bmatrix} s_1 + 2s_2 \cos \theta_2 & s_3 + s_2 \cos \theta_2 \\ s_3 + s_2 \cos \theta_2 & s_3 \end{bmatrix}, \\ \mathbf{C}(\boldsymbol{\theta}, \dot{\boldsymbol{\theta}}) &= \begin{bmatrix} -\dot{\theta}_2(2\dot{\theta}_1 + \dot{\theta}_2) \\ \dot{\theta}_1^2 \end{bmatrix} \cdot s_2 \sin \theta_2, \quad \mathbf{D} = \begin{bmatrix} d_{11} & d_{12} \\ d_{21} & d_{22} \end{bmatrix}, \\ s_1 &= I_1 + I_2 + m_2 l_1^2, \quad s_2 = m_2 l_1 l_{g2}, \quad s_3 = I_2. \end{aligned}$$

They are represented by the link parameters: i.e. mass m_i , length l_i , distance from the joint centre of mass l_{gi} , moment of inertia I_i , joint friction d_{i1} , d_{i2} ($i = 1$, upper arm; $i = 2$, forearm). The parameters are shown in Table 1, and were estimated from our measurements of a Japanese macaque. Because many muscles act on the arm in the horizontal plane, we modelled only two degrees of freedom actuated by six muscle groups: SF, shoulder flexor; SX, shoulder extensor; BF, biarticulate flexor; BX, biarticulate extensor; EF, elbow flexor; and EX, elbow extensor (Figure 6a). The joint torque is a function of its moment arms $\mathbf{A} \in \mathbb{R}^{2 \times 6}$ and the muscle tension vector $\mathbf{T} = [T_1, T_2, \dots, T_6]^T$, and it is given by $\boldsymbol{\tau} = \mathbf{A} \cdot \mathbf{T}$. The moment arm is defined as the perpendicular distance from the muscle line of action to the joint centre of rotation, given by

$$\mathbf{A} = \begin{bmatrix} 1.5 & -1.5 & 0 & 0 & 1.5 & -1.5 \\ 0 & 0 & 1.5 & -1.5 & 1.5 & -1.5 \end{bmatrix} / 100. \quad (2)$$

The j^{th} muscle activation a_j ($j = 1, 2, \dots, 6$) is not equal to the instantaneous neural input u_j , but is generated by passing u_j thorough a filter that describes the calcium dynamics modelled with a first-order nonlinear filter⁴⁷:

$$\alpha_j = \frac{u_j - a_j}{f(u_j, a_j)}, \quad (3)$$

where

$$f(u_j, a_j) = \begin{cases} t_{deact} + u_j(t_{act} - t_{deact}) & u_j > a_j \\ t_{deact} & u_j \leq a_j \end{cases}.$$

The time constant parameters were set as $t_{act} = 0.05$ [s] and $t_{deact} = 0.066$ [s] because the input-dependent activation dynamics are faster than the constant deactivation dynamics (Figure 6b).

Figure 6. Simulation model. (a) Allocation of muscles in the two-link, six-muscle arm model. The SF models the pectoralis major, coracobrachialis and deltoid anterior muscles. The SX models the posterior and middle deltoid muscles. The BF models the long and short biceps muscles. The BX models the long triceps muscle. The elbow flexor (EF) models the brachialis, brachioradialis and extensor carpi radialis longus muscles. The elbow extensor (EX) models the lateral and medial triceps muscles. (b) Muscle activation dynamics. (c) Length–velocity–tension curve of the muscle model.

Table 1. Link parameters.

Link	m_i [kg]	l_i [m]	l_{gi} [m]	I_i [kg·m ²]	d_{i1} [N·m·s/rad]	d_{i2}
$i = 1$	0.3	0.15	0.07	5.0×10^{-3}	5.0×10^{-3}	2.5×10^{-3}
$i = 2$	0.3	0.21	0.12	9.0×10^{-3}	2.5×10^{-3}	5.0×10^{-3}

Mammalian muscles have remarkable scaling properties, meaning that all are similar after proper normalization: length is expressed in units of the length at which the maximum isometric force is generated ⁴⁸, and velocity is expressed in the units per second ⁴⁶. Thus we assume the units are unified across all muscles as $L^0 = 0.08$ [m], and denote a normalized muscle length L_j as follows:

$$L_j = 1 + A_j^T (\boldsymbol{\theta}_j^0 - \boldsymbol{\theta}) / L^0, \quad (4)$$

where A_j is the j^{th} row vector of $\mathbf{A}$, $\boldsymbol{\theta}_j^0 \in \mathbb{R}^2$ is the optimal joint angle vector of the j^{th} muscle for generating the maximal torque in the shoulder and elbow, respectively. Then the muscle tension T_j is given to scale the unit-less tension by the absolute muscle force of the physiological cross-sectional area (PCSA) to yield the physical tension ⁴⁹:

$$T_j = F_a \cdot P \cdot \bar{T}_j, \quad (5)$$

where F_a is the absolute muscle force and is set to $F_a = 32$ [N/cm²] based on measurements in monkeys ⁵⁰, and P is the PCSA which is assumed to unify across the muscles as $P = 10$ [cm²]. According to a model of mammalian skeletal muscle ^{46,51}, the unit-less muscle tension $\bar{T}_j$ is produced by a nonlinear muscle model composed of the functions of contractile element $F_{CE}(\cdot)$ and passive elastic element $F_{PE}(\cdot)$:

$$\bar{T}_j = F_{CE}(a_j, L_j, \dot{L}_j) + F_{PE}(L_j), \quad (6)$$

where

$$F_{CE}(a_j, L_j, \dot{E}_j) = A_f(a_j, L_j) \cdot F_L(L_j) \cdot F_V(L_j, \dot{E}_j), \quad (7)$$

$$F_{PE}(a_j, L_j) = F_{PE1}(L_j) + A_f(a_j, L_j) \cdot F_{PE2}(L_j). \quad (8)$$

Here $A_f(\cdot)$, $F_L(\cdot)$, $F_V(\cdot)$ are the functions of the activation-frequency relationship, the tetanic force-length relationship, and the tetanic force-velocity relationship, respectively. The passive elastic force is represented by two separate functions, $F_{PE1}(\cdot)$ and $F_{PE2}(\cdot)$, which exert a tensile force and resists compression force, respectively. They are defined as follows:

$$A_f(a_j, L_j) = 1 - \exp \left[- \left\{ \frac{a_j}{0.56(2.12 + 3.31(1/L_j - 1))} \right\}^{2.12 + 3.31(1/L_j - 1)} \right],$$

$$F_L(L_j) = \exp \left(- \left| \frac{L_j^{1.55} - 1}{0.81} \right|^{2.12} \right),$$

$$F_V(L_j, \dot{E}_j) = \begin{cases} \frac{-7.39 - \dot{E}_j}{-7.39 - (3.21 - 4.17L_j)\dot{E}_j} & \dot{E}_j \leq 0 \\ \frac{1.05 + 1.53\dot{E}_j}{1.05 + \dot{E}_j} & \dot{E}_j > 0 \end{cases},$$

$$F_{PE1}(L_j) = 0.15 \log \left\{ \exp \left(\frac{L_j - 1.54}{0.059} \right) + 1 \right\},$$

$$F_{PE2}(L_j) = \min \left[-0.02 \left\{ \exp(18.7(L_j - 0.79)) - 1 \right\}, 0 \right].$$

The dependence of force on the length and velocity of a muscle is often referred to as the force-length and force-velocity curves, respectively (Figure 6c). The muscle model parameters were shown in Table 2, which are assigned based on the results of anatomical measurements of macaque monkeys^{50,52}.

Table 2. Equilibrium joint angles of each muscle. *does not affect the muscle length regardless of the value, because the moment arms are set to zero so as not to transform the effects.

	$j = 1$	$j = 2$	$j = 3$	$j = 4$	$j = 5$	$j = 6$
$\frac{180}{\pi} \theta_j^0$ [deg]	15	5	—*	—*	15	5
	—*	—*	90	110	100	100

Approximately optimal feedback control

We transformed the two-joint six-muscle model into a state–space model. The control object was denoted by the state vector $\mathbf{x} \in \mathbb{R}^{10}$ as

$$\mathbf{x} = \begin{bmatrix} \boldsymbol{\theta} \\ \dot{\boldsymbol{\theta}} \\ \mathbf{a} \end{bmatrix}, \quad (9)$$

where $\mathbf{a} = [a_1, a_2, \dots, a_6]^T$ is the muscle activation vector. Here we define the state vector at time t as $\mathbf{x}(t)$. Then the dynamics of the musculoskeletal arm model can be written as a state–space equation described as

$$\dot{\mathbf{x}}(t) = F(\mathbf{x}(t)) + G(\mathbf{x}(t)) \cdot \mathbf{u}(t).$$

The nonlinear functions $F(\cdot)$ and $G(\cdot)$ are defined for descriptive purposes to represent the dynamics in an affine form. In practice, they are given as locally linearized forms around each state at time t to obtain an approximately OFC law. The motor command $\mathbf{u}(t)$ is computed iteratively using the Levenberg–Marquardt algorithm to optimize the following cost function:

$$q_1 = \min_{\mathbf{u}} \left(w_p \left\| \mathbf{p}(T_s) - \mathbf{p}^* \right\|^2 + w_v \left\| \mathbf{v}(T_s) \right\|^2 + w_f \left\| \mathbf{f}(T_s) \right\|^2 + \int_0^T \left\| \mathbf{u}(t) \right\|^2 dt \right), \quad (10)$$

where T_s and T are the terminal time of the movement and simulation duration, respectively (i.e. $T_s = 0.4$ [s] and $T = 0.5$ [s]). The vectors $\mathbf{p}(T_s) \in R^2$, $\mathbf{v}(T_s) \in R^2$ and $\mathbf{f}(T_s) \in R^2$ are the end-point position, velocity and force in Cartesian space at the movement end T_s , respectively. They are calculated from the joint angles, angular velocities and torques as follows:

$$\mathbf{p}(t) = \begin{bmatrix} l_1 \cos \theta_1(t) + l_2 \cos(\theta_1(t) + \theta_2(t)) \\ l_1 \sin \theta_1(t) + l_2 \sin(\theta_1(t) + \theta_2(t)) \end{bmatrix}, \quad \mathbf{v}(t) = \mathbf{J}(t) \cdot \dot{\boldsymbol{\theta}}(t),$$

$$\mathbf{f}(t) = (\mathbf{J}(t)^T)^{-1} \cdot \boldsymbol{\tau}(t),$$

where $\mathbf{J}(t) \in R^{2 \times 2}$ is the Jacobian matrix

$$\mathbf{J}(t) = \begin{bmatrix} -l_1 \sin \theta_1(t) - l_2 \sin(\theta_1(t) + \theta_2(t)) & -l_2 \sin(\theta_1(t) + \theta_2(t)) \\ l_1 \cos \theta_1(t) + l_2 \cos(\theta_1(t) + \theta_2(t)) & l_2 \cos(\theta_1(t) + \theta_2(t)) \end{bmatrix}.$$

In addition, $\mathbf{p}^*$ is a target position in Cartesian space, and w_p , w_v and w_f are cost weights of the position, velocity and force requirements at the movement end, respectively. On the right-hand side of equation (10), the first, second and third terms evaluate the requirements of the end-point position, velocity and force, respectively, at the end of movement in achieving the desired state, whereby the end-point is close to the target position with zero values for the end-point velocity and force. The fourth term, which is the sum of the squares of the neural inputs during the movement, evaluates the metabolic cost of the neural input.

Cost weight parameters

We simulated four situations requiring movement under minimized neural input by balancing the cost weights in equation (10) (Table 3). These were determined heuristically to achieve the task. We confirmed that the results in this study are robust to

changes in the parameters according to the sensitivity analysis (see Supplementary Note).

Table 3. Cost weight parameters.

	w_p (position)	w_v (velocity)	w_f (force)
Case 1	1000	0	0
Case 2	1000	100	0
Case 3	1000	0	10
Case 4	1000	100	10

Stabilization control

We examined an alternate hypothesis that stabilizes the terminal position, or position and velocity after the movement phase but not to minimize only the terminal cost at the movement end. Thus, we additionally defined following two cost functions for the stabilization control:

$$q_2 = \min_{\mathbf{u}} \left[\frac{1}{T - T_s} \int_{T_s}^T w_p \|\mathbf{p}(t) - \mathbf{p}^*\|^2 dt + \int_0^T \|\mathbf{u}(t)\|^2 dt \right], \quad (11)$$

and

$$q_3 = \min_{\mathbf{u}} \left[\frac{1}{T - T_s} \int_{T_s}^T \left(w_p \|\mathbf{p}(t) - \mathbf{p}^*\|^2 + w_v \|\mathbf{v}(t)\|^2 \right) dt + \int_0^T \|\mathbf{u}(t)\|^2 dt \right]. \quad (12)$$

Then, we set the time at the movement end and stabilization period as 0.5 s and 0.1 s, respectively (i.e. $T_s = 0.4$ [s], $T = 0.5$ [s]).

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Author contributions

All of this study was conducted by Y.U.

Competing interests

The authors declare no competing interests.

Data availability

The MATLAB (MathWorks, Natick, MA, USA) codes and data sets that support the findings of this study are available in Github (<https://github.com/yuki-ueyama/Muscle-Activation-Pattern>).

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Figures

Figure 1

Triphasic muscle activation pattern during reaching movement. AG1, AG2 and ANT indicate the sequence of bursts for the pair of agonist (AG1 and AG2) and antagonist (ANT) muscles.

Figure 2

The centre-out movement for 16 targets. The columns from left to right are the hand paths, hand speed profiles, root mean squared hand force profiles and joint torque profiles. In the hand paths, the target positions are shown as filled circles. In the joint torque profiles, solid and dotted lines indicate the torques of the shoulder and elbow joints, respectively. Cases 1–4 are in rows (a)–(d), respectively.

Figure 3

Muscle activation patterns plotted as a function of time and target direction. Cases 1–4 are shown in (a)–(d), respectively. The values were normalized using the highest activation level in each muscle across all cases.

Figure 4

Stabilization control. The controls of only the position cost, and a pair of the position and velocity costs are shown in (a, c) and (b, d), respectively. (a, b) The centre-out movement for 16 targets. The format is the same as in Figure 2. (c, d) Muscle activation pattern. The format is the same as in Figure 3.

Figure 5

Typical feedback gains of the optimal feedback control (OFC) model for a linear system. Each of Case 1–4 corresponds to the same formation of the cost function above results. Each value of the position, velocity and force gains is normalised by that of Case 4.

Figure 6

Simulation model. (a) Allocation of muscles in the two-link, six-muscle arm model. The SF models the pectoralis major, coracobrachialis and deltoid anterior muscles. The SX models the posterior and middle deltoid muscles. The BF models the long and short biceps muscles. The BX models the long triceps muscle. The elbow flexor (EF) models the brachialis, brachioradialis and extensor carpi radialis longus muscles. The elbow extensor (EX) models the lateral and medial triceps muscles. (b) Muscle activation dynamics. (c) Length–velocity–tension curve of the muscle model.

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