

Modular Control of Movement and Posture by the Corticospinal Alpha-Gamma Motor Systems

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Abstract—It is widely assumed that neural control of movement is carried out by the α motor system sufficiently. The role of the γ motor system in movement and posture has not been adequately addressed in motor control studies. Here, we propose a modular control model for movement and posture based on propriospinal neuronal (PN) network and spinal α - γ motor system. In the modular control model, the α and γ motor commands are divided into static and dynamic functions. The static commands are specified by the higher center of brain for posture control, and the dynamic commands for movement generation, respectively. Centrally planned kinematics based on the minimal jerk criterion is conveyed to the periphery via the γ motor system, while centrally programmed bi-phasic burst pattern of muscle activation is relayed to a pair of antagonistic muscles through the α motor system via the PN. Results of simulation showed that elbow kinematics and biceps and triceps activations displayed the similar kinematic and EMG features of fast reaching movement in human. This suggests a hypothesis that the α - γ motor systems can achieve modular control of movement and posture in parallel.

I. INTRODUCTION

Advancement in theory of neural control of movement and posture by human brain has not kept up with the pace of research in neurophysiology, biomedical and robotics engineering. Among different versions of equilibrium-point hypothesis, the λ -model [1] argued that posture was an equilibrium resulting from the length-dependent reflex forces generated by agonist-antagonist muscles, and a movement might be initiated by setting appropriate stiffness field of the arm, while shifting the equilibrium position of the joint. But the λ -model seemed insufficient in producing fast motor tasks. However, it demonstrated the importance of feedforward and feedback control of movement and posture.

Previous studies have suggested a dual control framework, in which posture and movement might be planned and controlled separately [2, 3]. This is in line with the finding that there are two cortico-motoneuronal descending pathways, i.e. mono-synaptic pathway and multi-synaptic pathway via propriospinal neurons (PN) in the C3–C4 spinal cord. In a modified α - γ control model [4], movement and posture control

was executed by the α and γ motor systems, respectively. This model has been shown to be capable of replicating the behavior of Parkinsonian tremor [4], and is consistent with the pattern of α - γ co-activation displayed in locomotion of decerebrate cat [5].

In the present study, we expand the α - γ control model [4] to show that it also allows modular control for movement and posture in the elbow joint. Movement trajectory and triceps/biceps EMGs were recorded from normal human subjects, and were used as template in the tuning of model behaviors. Descending cortical α and γ commands drove the virtual arm (VA) model [6, 7] to generate kinematic behaviors. The minimal jerk trajectory [8] was assumed to be centrally planned kinematics that was conveyed to muscle spindles by way of γ motor system [5, 9]. A bi-phasic pulse of α dynamic command dictated the acceleration and deceleration of joint motion [2, 10-12], in performing a reaching movement. Results confirmed that modular control of both posture and movement can be achieved by programming the descending α and γ commands simultaneously.

II. METHODS

A. Human Fast Reaching Experiment

7 subjects performed successive elbow movement with about 5 seconds' postural holding period in between fast reaching in the horizontal plane. As the sketch shown in Figure 1(A), the subjects extended their elbow following the 3 blocks: elbow angle changed from 0° to 90° with step sizes of 30° (block 1) and 45° (block 2) respectively, and from 10° to 90° with no pause (block 3). Between blocks, the subject had about 5 minutes' rest. And there were 5 trials for each block, with 10 seconds' rest between trials, the subject was told to hold on for 5 seconds at each stage of elbow angle. The human subject study was approved by the Internal Review Board (IRB) of University of Southern California (USC).

The raw EMG data of biceps and triceps were filtered with a band-pass filter with a cut-off frequency between 20 to 500 Hz to remove motion artifacts and high-frequency noise, and then with a notch filter at 50 Hz to remove power line noise. The steady postural EMG was picked out to calculate the average muscular activation levels for different angular position of elbow. The calibrated motion signals were low-pass filtered with a cut-off frequency of 20 Hz to remove high-frequency noise, and then utilized for calculation of joint kinematics. All the digital filters applied in the off-line processing were designed to process signals in both forward and reverse directions to achieve zero-phase shift in the filtered data.

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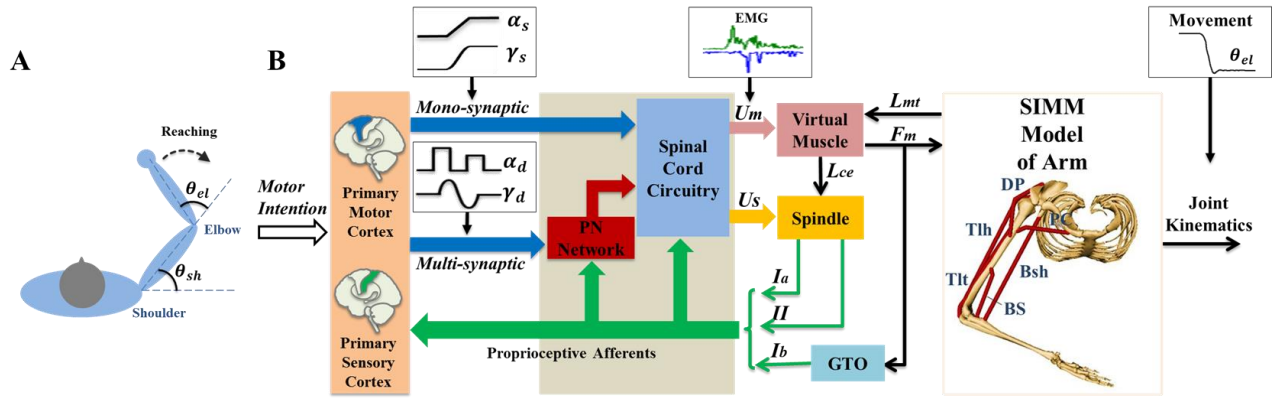


Figure 1. (A) A sketch of experimental settings: θ_{sh} is shoulder angle, and θ_{el} is elbow angle. (B) Corticospinal virtual arm model: U_m is muscle input, and U_s is spindle input; the virtual arm has two joints including shoulder and elbow in the horizontal plane, with two degrees of freedom. Shoulder flexion/extension and elbow flexion/extension. Three pairs of antagonistic muscles were used: shoulder flexor Pectoralis Major Clavicle (PC), extensor Deltoid Posterior (DP), elbow flexor Brachialis (BS), extensor Triceps Lateral (Tlt), and biarticular flexor Biceps Short Head (Bsh), extensor Triceps Long Head (Tlh).

B. Corticospinal Virtual Arm Model

The corticospinal virtual arm model (Figure 1(B)) is composed of primary motor cortex, propriospinal neuron (PN) network (see section II.C), spinal reflex (SR) circuitry and virtual arm (VA) model [4]. This model is based on previous work [6, 7], and extensive physiological studies on PN and SR [2, 3, 9, 13]. The VA model has also been validated to capture the neuromechanical properties of realistic human arm [7]. The three sub-systems have been integrated in SIMULINK/ MATLAB platform for simulation.

C. The PN-VA Model

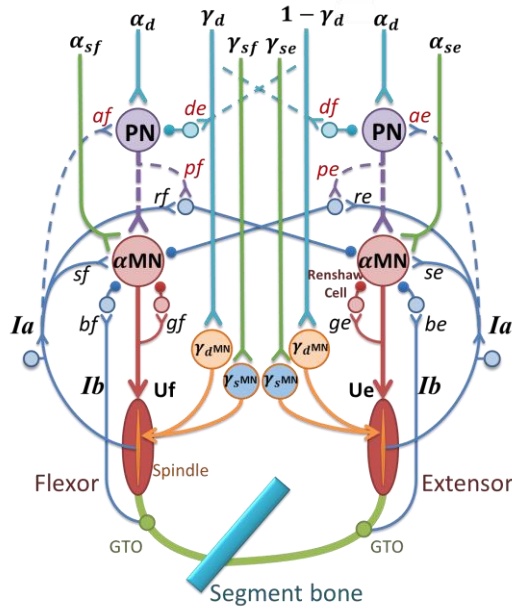


Figure 2. Dual control neural network model in a pair of antagonistic muscles. PN represents propriospinal interneuron; MN represents motor neuron pool; Subscript “d” and “s” of α and γ descending commands refer to “dynamic” and “static”, subscript “f” refers to flexor, “e” refers to extensor; U_f and U_e are muscle activation; a_f and a_e are gains on PN from Ia afferent; r_f and r_e are stretch reflex gains; r_f and r_e are Ia-reciprocal inhibition gains; b_f and b_e are Ib gains of Golgi tendon organ (GTO); g_f and g_e are Renshaw cell gains; r_f and r_e are reciprocal inhibition gains; p_f and p_e are PN related reciprocal gains; d_f and d_e are γ dynamic inhibition gains on PNs. The colored thin lines present axons in the neuron system, those result in branch represent excitation on the targeted neuron, while others result in filled dot represent inhibition on the targeted neuron, through the interneurons shown as small round cells.

According to dual control framework (Figure 2), static α and γ commands are sent to α motor neuron pools and γ static motor neuron pools directly, to recruit targeted muscles and spindles for posture control. In the movement module, central α dynamic commands are first regulated in PN, receiving primary afferent’s feedback and inhibition from antagonistic γ dynamic command, and then processed in alpha motor neuron pools together with α static commands, finally the integrated output will act as muscle activation. Meanwhile, α motor neurons will get feedback from both autologous and antagonistic peripheral afferents. The gamma static (γ_s) and dynamic (γ_d) innervate the spindle bag2, chain and bag1 fibers. The alpha static (α_s) and dynamics (α_d) converge to the motoneuronal pool of muscles. A pair of antagonistic muscles typically displays an inverse $\gamma - \theta$ relationship with each other.

D. Descending Cortical Commands

For posture control before and after the simulated fast reaching movement, we used the static EMGs calculated from human fast reaching movement as α_s input; and according to [9], the quadratic strategy of γ static modulation on shoulder and elbow equilibrium angles, as shown in eqs.(1)-(6), was applied here. θ_{sh} and θ_{el} are equilibrium angles of shoulder and elbow, and the varying trajectories of joint angles are decided by minimum jerk strategy [8], show in eq. (7),

$$\gamma_s(PC) = 6e - 5\theta_{sh}^2 + 0.002\theta_{sh} + 0.3835 \quad (1)$$

$$\gamma_s(DP) = 7e - 5\theta_{sh}^2 - 0.0116\theta_{sh} + 0.9595 \quad (2)$$

$$\gamma_s(Bsh) = 2e - 5(\theta_{sh} + \theta_{el})^2 - 0.0008(\theta_{sh} + \theta_{el}) + 0.3698 \quad (3)$$

$$\gamma_s(Tlh) = 8e - 6(\theta_{sh} + \theta_{el})^2 - 0.0044(\theta_{sh} + \theta_{el}) + 0.9292 \quad (4)$$

$$\gamma_s(BS) = 6e - 5\theta_{el}^2 - 0.001\theta_{el} + 0.3855 \quad (5)$$

$$\gamma_s(Tlt) = 2e - 5\theta_{el}^2 - 0.005\theta_{el} + 0.7772 \quad (6)$$

$$C = \frac{1}{2} \int_{t_0}^{t_1} \left(\frac{d^3\theta}{dt^3} \right)^2 dt \quad (7)$$

in which, θ is joint angle, t_0 and t_1 are the real time of movement initiation and termination. To minimize the objective function C , a fifth order polynomial shown in eq. (8) is required,

$$\theta(t) = \sum_{i=0}^5 a_i t^i \quad (8)$$

Also the initial and terminal joint angles, angular velocity and acceleration at time t_0 and t_1 (for our simulation, they were set to zero.) were required. This minimal jerk trajectory was used as γ_s commands in this model.

For movement control, we used a bi-phasic pulse of α_d activation to drive a pair of antagonistic muscles. γ_d input was equated to the 2nd derivative of equilibrium angle of eq. (8).

E. Simulation Experiment

Simulation trials with a duration of 15 seconds were typically performed. A random, signal dependent noise [14] was added to muscle activation at the time 5 (sec) after the system had converged at a stable initial state and the shoulder angle was fixed at 52° with a relatively strong static activation to lower its effect on elbow control. The elbow was driven at time 10 (sec) by cortical dynamic commands of α_d and γ_d to perform a fast reaching movement. The reflex gains used here were adopted from [4, 7] to keep the system stable. In these simulations, we chose biceps short and triceps long to drive elbow movement, and all α and γ commands applied were normalized with values between 0 and 1.

III. RESULTS

A. Experimental Ratio of Posture EMGs

The static EMGs of biceps and triceps during postural holding period in between fast reaching, recorded from all three blocks of subject 1, were found increasing and decreasing respectively as the elbow extended, and the ratio of them was fitted into a linear relationship with joint angle, plotted in Figure 3, which provided a reference in specifying α_s commands in simulation.

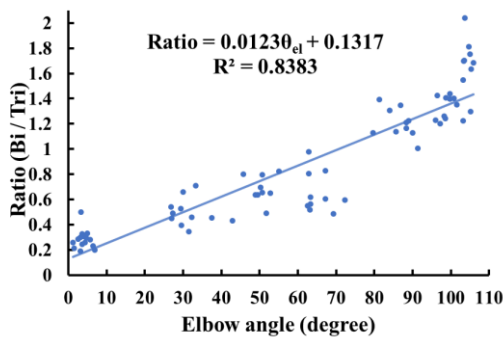


Figure 3. The relationship between the ratio of postural EMG of biceps and triceps, and elbow angle.

B. Programming Cortical Commands

A set of cortical commands, aimed at driving the elbow from 67° to 30° in accordance with the forth trial of block 1 of subject 1, was applied to the model in our simulation, as shown in Figure 4. The duration of cortical commands for reaching planning is 0.5 seconds, from time 10 (sec) to 10.5 (sec), especially, α_d included two pulses, the first is from time 10.03(sec), with a duration of 0.23 seconds, the amplitude of 0.63, and a second pulse with the duration of 0.15 seconds, the amplitude of 0.1, 0.1 seconds after the first pulse ends. To stabilize the joint angle after overshooting, a third pulse on α_s

of Tlh was utilized from time 10.4(sec), with the amplitude of 0.65 and pulse width 0.12 seconds, which was found of great significance in postural maintenance in our simulation, and could be easily observed in EMG activity during fast reaching movement. The initial and final postural α_s input of Bsh and Tlh came from experimental recordings, and those of BS and Tlt were adjusted to counteract the effect of gravity during the extension course, providing a background action for posture maintenance.

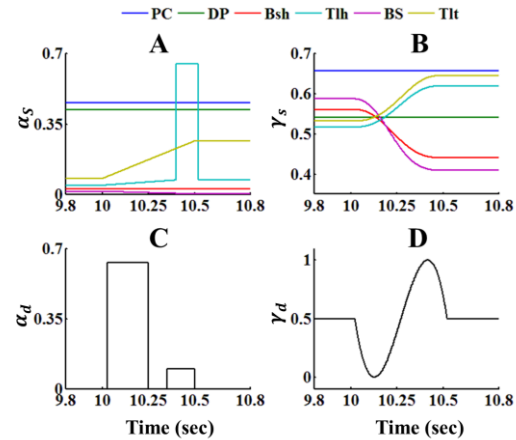


Figure 4. (A) α_s commands (the six colored lines represent six different muscles, respectively, so as in (B)); (B) γ_s commands; (C) α_d command (same for Tlh and Bsh, as shown in Figure 2); (D) γ_d command of Bsh.

C. Simulation of Movement and Postural Control

The simulation results were compared with experimental results (the forth trial of block 1 of subject 1) in Figure 5. It showed that before and after the reaching period, the elbow could be finely held at postures of experimental value by static cortical commands. During the movement, the elbow was driven to the final position of 30° with a slightly larger oscillatory stabilization. The angular velocity (Figure 5(B)) showed a similar bell-shaped profile to that of human in experiment. In general, the simulated kinematics was in agreement with the profiles of human movement in experiments.

The EMG of Tlh and Bsh in the simulation (Figure 5(C)) shared the same tri-phasic firing pattern with the experiment, in spite of the differences in amplitude. However, the rebounding of joint angle turned out to be much smaller in recorded human movement. The Ia and Ib afferents shown in Figure 5 (E) & (F) gave a reflex reaction to changes in muscle fascicle length and force. The Bsh lengthened during reaching, while Tlt shortened, with a much narrower length range. The tri-phasic muscle force corresponded to acceleration, deceleration and stabilization stages of a fast movement as seen in Figure 5(A).

IV. DISCUSSIONS AND CONCLUSION

Here we demonstrated the plausibility of modular control of posture and movement by separate descending commands to the $\alpha - \gamma$ motor systems. Simulation illustrated that posture could be maintained through static motor commands via mono-synaptic pathway and movement could be elicited by a doublet pulse in α_d command via propriospinal neuronal pathway. A relatively higher level of activation in the α_s of Tlt

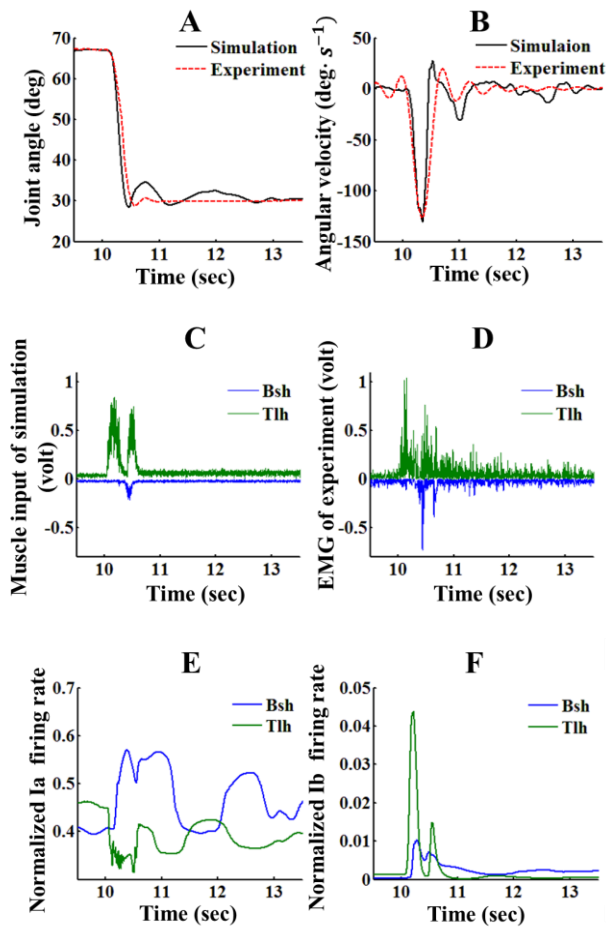


Figure 5. (A) Elbow angle of simulation comparing with experimental result; (B) Angular velocity; (C) Muscular activation of simulation (Bsh reversed); (D) EMG pattern of muscle activation in experiment. (E) & (F) Normalized Ia and Ib afferent firing rate in the simulation.

was needed to compensate for the effect of gravity on the joint. The minimal jerk trajectory [8] and its 2nd derivative served an adequate template for centrally planned kinematics and dynamics of movement, which was conveyed to muscle spindles via γ_s and γ_d fusimotor inputs, respectively. The propriospinal neuronal network in the multi-synaptic pathway played a pivotal role to topple the activation of agonist and antagonist during acceleration and deceleration of movement.

By adjusting the dynamic inputs of muscles and spindles, a single joint reaching movement could be reproduced with this dual control model. However, it was found that a bi-phasic firing pattern of Tlh and Bsh was not adequate to stabilize the elbow joint at the final position. The actions of reflexes seemed to be insufficient to stabilize the elbow joint at the final posture, since the reflex gains were relative low in these simulations [4, 7]. We thus used a pulse in the α_s of Tlh (Figure 4(A)) to help stabilizing the elbow joint at the new posture. This was found necessary to match the simulated profile of Figure 5(C) to the tri-phasic profile of experimental EMGs in Figure 5(D), indicating that the third phase of EMG bursts was essential to stabilize the joint after movement.

A pair of pulses of α_d cortical commands during reaching for agonistic and antagonistic muscles might have a physiological source from sub-cortical basal ganglia network,

which was found to emit two excitatory pulses in its output in the SNr neurons after the motor cortex had been electrically stimulated [15]. The descending cortical command might then be divided into activations to target a pair of flexor and extensor muscles to produce a movement by the PN circuitry in the spinal cord [4]. However, we did not use an optimization procedure to determine the descending commands in these simulations. All descending commands were chosen by an ad hoc approach. In actuality, cortical commands of a skilled movement by human may have been learned by sub-cortical structures, e.g. cerebellum and basal ganglia circuitry [16]. Yet, the learning mechanism performed by the sub-cortical neural circuitry remains unclear.

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