A Computational Model of Adaptation to Novel Stable and Unstable Dynamics

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Introduction

Humans have exceptional abilities to produce movements and interact with objects in the environment. When faced with novel tasks, they adapt to environmental disturbances in a way that indicates they are forming an internal representation of the external mechanics or an internal model. Adaptation under mechanically stable conditions appears to involve the acquisition of an inverse dynamics model through feedback error learning [4]. However, many tasks that humans perform, particularly those involving tool use, are inherently unstable and the mechanism of adaptation to these environments is not well understood. One form of adaptation to instability, which we have been investigating, is the selective control of endpoint stiffness [1].

Our recent research suggests that inverse dynamics models and impedance control are combined during motor learning. An inverse dynamics model is a controller which computes feedforward commands of the net joint torques for movement, based on the estimated effects of internal and external dynamics. An impedance controller, in contrast, modifies the impedance of the limb by co-contraction of agonist and antagonist muscles without changing net joint torque. Recent experiments have provided evidence that inverse dynamics models and impedance control may function independently [6;7]. In our most recent work we have investigated the adaptation to various stable and unstable environments which has led to a proposed computational mechanism whereby such adaptation can be realized by the central nervous system (CNS).

Methods

Subjects sat in a chair and moved a 2D robotic manipulator (PFM) in a series of forward reaching movements performed in the horizontal plane. Trajectory, joint torque and EMG changes were investigated during and following adaptation to a stable velocity dependent (VF) and an unstable position dependent (DF) force field and were compared to performance in a null force field (NF).

Subjects initially performed movements in the NF. After 50 to 70 trials, one of the novel force fields was unexpectedly substituted for the NF. Subjects proceeded to adapt to this force field over the subsequent 100 to 200 trials. Both after effect and before effect trials were recorded to check that subjects had adapted to the force field and to aid in interpreting adaptive changes in EMG.

Results

Initially subjects' trajectories were disturbed by the force fields, however after training they tended to produce straight movements to the target. Two parallel processes were identified from the evolution of the muscle activation patterns. One was an activation process involved in increasing the endpoint stiffness of the arm by means of muscle co-contraction during the early stages of learning. The other was a deactivation process, which led to gradual reduction in muscle activity as learning progressed. Adaptation to the VF was characterized by fast increases in muscle activity contributing to the inverse dynamics model and a slower increase in generalized co-contraction. Kinematic error was reduced with a time course similar to the increase in co-contraction but adaptation of the joint torque (formation of inverse dynamics model) took longer. Later, unnecessary co-contraction was reduced. The change between the initial and final patterns of muscle activation was similar to the reflex responses observed during before effects, suggesting that the reflex patterns provided a template for the feedforward command. Learning in the DF was characterized by initial slow increase in activation of all muscles (co-contraction) which occurred at a similar rate to the decrease in kinematic error. After adaptation to the force field, unneeded co-contraction was gradually reduced. Kinematic data for the first few trials in the DF suggest that the CNS attempts to learn the DF using an inverse dynamics model. Therefore, it appears that the CNS utilizes both an inverse dynamics model and impedance control in adapting to any novel environment.

Discussion of Computational Model

Osu et al. [5] showed that the classical feedback-error-learning algorithm [3] applied to joint torque can explain inverse dynamics model learning in the VF, but cannot explain impedance learning in the DF. The following natural extension of feedback error learning could coherently unify the two learning processes, and at least qualitatively reproduce the current results, as well as other recent data on motor learning [1;2;6]. First, centrally generated feedforward motor commands would comprise both a reciprocal component for agonist and antagonist muscle pairs (difference in muscle activation similar to net joint torque) and a co-activation component (summation of agonist and antagonist muscle activation similar to joint stiffness). Since these two components sum at the muscle level it is not necessary that they be separately represented in the brain. Second, the feedforward co-activation signal to antagonists should increase on trials following perturbation of the hand path during early learning even when only agonist muscles are stretched. Third, the feedforward co-activation signal decays with a large time constant as manifested by the deactivation time constants of all muscles. Synaptic plasticity equations can be constructed by extending the feedback-error-learning algorithm based on the proposed theory. Such a model could explain both the development of net joint torque to compensate for external forces and the selective adaptation of endpoint impedance to environmental instability. Computer simulations of this computational model qualitatively reproduce the experimental results and tentatively confirm this conceptual model.

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