Toward a genuine theoretical neuroscience of motor control

Daniel S. Levine

Department of Mathematics, University of Texas at Arlington, Arlington, TX 76019-0408
Electronic mail: *b344dsl@utarlg.uta.edu*

[GEA] The importance that **Alexander, DeLong & Crutcher** give to connectionist models heralds a welcome development in neuroscience. Experimental neuroscientists, working in motor control and other areas, are seeing neural network models not as toys or abstract exercises but as an increasingly significant contribution to the interpretation of their data. Yet these authors underestimate the progress that connectionist modeling of motor control has already made (see Levine 1991, Ch. 7).

Broadly speaking, I believe that in motor control, as in vision, conditioning, pattern recognition, or other areas, the most promising approach to models with genuine explanatory power is one that is well developed (see, e.g., Grossberg 1988, for many examples). Typically, the modeler sets out to explain a set of behavioral data using a network that obeys a dynamical system of differential equations. Often, parts of this network incorporate widely used network principles such as associative learning, lateral inhibition, opponent processing, or error correction. Then the network is refined to incorporate known anatomy, physiology, or neurochemistry of brain regions that are good candidates for analogy with some of the network nodes. With this approach, network theory has a "life of its own" as an equal partner with experiment. Experimental data can suggest alterations of models, and models can suggest new experiments to be performed.

I will give two examples of this approach as it has been applied to the role of the cortex and basal ganglia in motor control. Both of the models to be discussed rely on extensive feedback, and thereby synthesize the "bottom-up" and "top-down" approaches (target article, sect. 4). As **Alexander et al.** indicate, this makes such models more plausible as brain models than those that involve supervised learning or hybrids with a hardwired symbol-processing module.

Bullock and Grossberg (e.g., 1989; 1991) have developed a series of models for the performance of planned arm movements, particularly focusing on how positional control is independent of speed and force rescaling. The error-correction in their model requires a vector that computes the difference between a present position and a target position. They have noted that cells have been found in the shoulder-elbow zone of the precentral motor cortex with properties analogous to a difference vector (Georgopoulos et al. 1984; Schwartz et al. 1988). Another element that is required by the Bullock-Grossberg models is a "go" signal that is multiplied by the difference vector to generate a movement command. This enables the movement to be interrupted in the middle, by shutting off the go signal, and later resumed. Also, variable amplitude of the go signal allows the same movement to be performed, with essentially the same trajectory, at varying speeds. The neurophysiological requirements for a go signal are that it should (1) have nonspecific effects on a variety of muscles, (2) affect the rate but not the amplitude of muscle contractions, (3) not affect accuracy, but (4) be necessary for movement performance. Such properties were found in a class of globus pallidus neurons by Horak and Anderson (1984a; 1984b).

Higher-order controls on movement sequences involving the prefrontal cortex were modeled by Bapi and Levine (1990). Data on rhesus monkeys (Brody & Pribram 1978; Pinto-Hamuy & Linck 1965) had shown that monkeys with frontal lobe damage can learn one invariant sequence of movements if it is rewarded, but they cannot learn to perform any one of several variations of a sequence if all are rewarded. Modeling these data requires a sequence-generating circuit located elsewhere than the frontal cortex, but the frontal lobes must exert some sort of classificatory control on this circuit. Such a circuit would incorporate primacy and recency effects: The first of the movement sequence has to be performed first even though the later movement representations have stronger connections to the reward representation. Bapi and Levine have made the tentative suggestion that the sequence circuit is located in the caudate and putamen and that the higher control is exerted by connections from the frontal cortex to the striatum, and indirectly back from the striatum to the frontal cortex via the limbic system, nucleus accumbens, and mediodorsal thalamus (all of which are involved in reward representation).

These two examples are not meant to imply that the brain's motor control circuits are totally understood. Rather, they are meant to give some of the "flavor" of the most promising connectionist models, and of how future models might be generated. **Alexander et al.'s** call for models that are parallel, flexible yet mathematically analyzable, incorporate top-down controls, and can suggest further experiments is already being answered.

Past the equilibrium point

Gerald E. Loeb

Bio-Medical Engineering Unit, Queen's University, Kingston, Ontario, Canada K7L 3N6
Electronic mail: *loeb@biomed.queensu.ca*

[EB] Neurophysiologists are indebted to **Bizzi, Hogan,** and colleagues for having pointed out that muscles are endowed with certain intrinsic mechanical properties that are quite different from those of an "ideal actuator" (i.e., torque motor as used in robotics), and that these properties may actually augment or even simplify the neural control of limb motion under certain conditions. However, their claim that "the hypothesis . . . has been corroborated experimentally" lacks a clear statement of a testable hypothesis and a body of thoroughly analyzed data from well-designed and relevant experiments. I would argue that neither has been presented. Furthermore, Bizzi et al.'s notion has fundamental limitations that have been obvious from the beginning; these have been acknowledged briefly in their Conclusions but not yet addressed satisfactorily.

What is the hypothesis? In the alpha model (sect. 2.1), **Bizzi et al.** present "a central postulate" that the CNS generates signals specifying an equilibrium position. A postulate is a proposition presented without proof, either because it is self-evident or for purposes of exploring its implications. If the CNS outputs in question are merely the levels of recruitment of the muscles, then it is indeed self-evident that the "spring-like properties" of those muscles must define an equilibrium position for the limb as a whole. Perturbations away from this position will lead inexorably to restorative forces (e.g., McKeon et al. 1984). The implicit question of interest to neuroscientists is: Under what conditions and to what extent does the CNS actually rely on this physical fact to simplify the planning and execution of skeletal movements? This question cannot be answered by identifying a few simple situations in which the externally observable behavior happens to be qualitatively consistent with this general notion. Instead, at least two key questions must be addressed:

1. Given the mechanics of a particular task and a particular musculoskeletal system, how many different, equally simple motor strategies would produce adequate performance, and what kinesiological data would be needed to distinguish them?

2. What is the set of tasks and conditions for which the equilibrium-point strategy would be expected to fail and how well does the CNS then cope?

Exploring the implications of this postulate is daunting but necessary if any testable hypotheses are to be generated at all.

What are the behavioral data? Even for the simple tasks selected so far by these researchers, the data are fragmentary and their real implications largely unexplored. In Bizzi et al. (1984), the raw data cannot be examined in detail to separate intrinsic from reflexive responses because the figures are obviously hand-traced (a common necessity for the Visicorder technology the authors used, but not acknowledged in the paper or in the target article), time scales are highly compressed, and EMGs lack calibration bars. The one figure that depicts EMG from an antagonistic pair of muscles (reproduced in the target article as Fig. 2) suggests an unusually high degree of cocontraction in the deafferented limb. Cocontraction is a common default strategy for dealing with novel or difficult tasks. It is not surprising that deafferented monkeys recognize the utility of the "alpha" strategy for coping with their deficit, but this provides little insight into normal sensorimotor behavior.

In dismissing the lambda model (sect. 2.2), **Bizzi et al.** cite the low gain of the stretch reflex in the neck (Bizzi et al. 1978). It is now clear that the segmental feedback from the numerous proprioceptors in the neck is remarkably weak compared to the limbs (Anderson 1977; Brink 1988; Keirstead & Rose 1988; Rapoport 1979; Richmond & Loeb 1992), for reasons that remain obscure. The homonymous feedback in the limbs is much stronger and the total effects of the heteronymous feedback system are just starting to be considered (He et al. 1991; Loeb et al. 1989). Because of the diverse and powerful descending systems for modulating gain in these pathways, any extrapolation between species, limbs, surgical preparations, and behavioral tasks is fraught with uncertainty.

The curved trajectories measured and modeled by Flash (1987) apparently represent the only attempt to simulate the consequences of equilibrium-point planning in a realistic model of a limb. As such, the effort is commendable and the results worth considering in more detail than has been provided in section 3. The agreement that she obtained between the simulated and measured trajectories is often qualitatively impressive, but it is unfortunate and perhaps not accidental that the study was restricted to relatively slow movements where the differences between static and dynamic planning schemes will tend to be small. Any control scheme that undercompensated for intersegmental dynamics might well produce similar results, particularly if the velocity profile within the movement can be selected arbitrarily, as implied by Flanagan et al. (1990). Furthermore, to obtain even these results, it was necessary to increase the joint stiffness matrix beyond that obtained statically by Mussa-Ivaldi et al. (1985). This seemingly innocent "fix" implies an additional cocontraction of antagonist muscles that flies in the face of what is known about the reciprocal control of antagonist muscles during reasonably fast and unconstrained movements. The absence of systematic recording and interpretation of EMG data in the entire body of work on equilibrium-point control frequently leaves its advocates in the tenuous position of trying to infer actuator kinetics from end-point kinematics, a risky business indeed in a system this complex.

Bizzi et al. note that Flash's results (weak as they are) might be explained by CNS programming of the shape and orientation of the stiffness field, presumably by means of reflex gating rather than patterns of cocontraction. They dismiss this possibility by citing Mussa-Ivaldi et al. (1987), in which such changes were not seen in response to various perturbations during a static task. This is a fatally flawed argument in motor psychology. The question is not whether subjects happened to change the shape of the stiffness profile for the chosen task but whether they could change this stiffness if it suited their purposes. In the experiments cited, subjects had no way of knowing whether they were changing the stiffness profile nor did they have any particular incentive to change it.

What are the neurophysiological data? Various parts of the frog spinal cord have been "microstimulated" by **Bizzi et al.** to find neurons that produce the motor patterns associated with the wiping reflex. This seems to be one of the most naive electrophysiological experiments published since the invention of the inductorium. The spinal cord is responsible for many sensorimotor behaviors, none of which is known to be segregated anatomically. What topographic structure there is (and it is particularly weak in the frog; Lichtman et al. 1984) suggests elongated, columnar entities that could not be selectively recruited from a monopolar microelectrode thrust into the gray matter. No data are provided regarding the amount of distribution of EMG activity thus evoked in the various leg muscles, or whether these EMG patterns resemble those developed during naturally evoked wiping reflexes. Given the "spring-like" behavior of muscles, virtually any combination of muscle recruitment that was not strongly polarized toward one group of muscles would produce an "equilibrium point." Apparently, such conditions obtain when stimulating in the intermediate zones (where all the motoneurons and interneurons have densely intermingled dendritic trees) and not in the ventral horn (where there is some clustering of motoneuronal somata into motor nuclei). Bizzi et al.'s Figure 5, showing gradual shifts in this equilibrium point during a stimulation train whose parameters and temporal relationships are not given, is not interpretable.

What is the point? It seems clear that robotics engineers have derived some insights into the shortcomings of torque motors and servocontrollers from this work (although the cryptic allusion to the unpublished work of McIntyre resurrecting position-derivative control does not bode well). When it comes to elucidating biological mechanisms, we are given only the continuing development and promotion of dogma in place of critical thinking and experimentation. The value of simple models is that they focus our thinking on the various fundamental aspects of problems, not that they are likely to constitute universal solutions. The roles of modeling and experimental design are supposed to be complementary in that the latter strives to reveal the limitations of the former. We have here too many examples of experiments and phenomena that seem to be selected to flatter rather than to reveal.

To what extent are brain commands for movements mediated by spinal interneurones?

Anders Lundberg
Department of Physiology, University of Göteborg, S-413 90 Göteborg, Sweden

[EEF] The initial studies of unit recording in behaving animals seemed to be based on the assumption that the motor cortex is interested solely in motoneurones even though it was known at that time that corticospinal volleys influence many spinal mechanisms, including interneurones of almost all spinal reflex pathways to motoneurones, to ascending pathways, and to many of the pathways producing presynaptic inhibition by their action of primary afferent terminals. It was an important advance in this field when **Fetz** and Cheney (1980) introduced the method of recording from cortico-motoneuronal (CM) cells identified by their EMG postspike facilitation. Even so, caution is required and I believe that some interpretations are based on doubtful assumptions. An inherent assumption seems to be that only the *monosynaptic* pathways from the motor cortex and the red nucleus matter. The cat does very well without these connections and I am convinced that the major command also in primates is via spinal interneurones, implying that the monosynaptic pathways, though important, contribute only a fraction of the command, perhaps producing some fractionation of muscle activation and final adjustment of the movement. If so, a causal relationship between cell and muscle activity would not always be expected.