Wagenaar, R.C., & Beek, W.J. (1992). Hemiplegic gait: A kinematic analysis using walking speed as a basis. *Journal of Biomechanics*, **25**, 1007-1015.
Wagenaar, R.C., & Van Emmerik, R.E.A. (1994). Dynamics of pathological gait. *Human Movement Science*, **13**, 441-471.
Wulff, H.R. (1976). *Rational diagnosis and treatment*. Oxford, UK: Blackwell.

Acknowledgments

We gratefully acknowledge stimulating discussions with Rob Bongaardt (cf., 1966), Claudine Lamoth, Chantal Leemrijse, and all members of the research group "Efficacy of Physical Therapy and Rehabilitation" at the Departments of Physical Therapy and Occupational Therapy, and the Faculty of Human Movement Sciences, Vrije Universiteit, Amsterdam.

Accepted for publication: March 16, 1998

POINT OF VIEW

Motor Control, 1998, **2**, 189-205

Recent Tests of the Equilibrium-Point Hypothesis (λ Model)

Anatol G. Feldman, David J. Ostry, Mindy F. Levin, Paul L. Gribble, and Arnold B. Mitnitski

The λ model of the equilibrium-point hypothesis (Feldman & Levin, 1995) is an approach to motor control which, like physics, is based on a logical system coordinating empirical data. The model has gone through an interesting period. On one hand, several nontrivial predictions of the model have been successfully verified in recent studies. In addition, the explanatory and predictive capacity of the model has been enhanced by its extension to multimuscle and multijoint systems. On the other hand, claims have recently appeared suggesting that the model should be abandoned. The present paper focuses on these claims and concludes that they are unfounded. Much of the experimental data that have been used to reject the model are actually consistent with it.

The most complete account of the λ model (a version of the equilibrium-point [EP] hypothesis) has been presented in a recent target article (Feldman & Levin, 1995) together with numerous commentaries and our response to them. Based on experimental data, starting from Matthews (1959) and Asatryan and Feldman (1965), the model represents an approach to understanding how the nervous system controls movement that integrates both physical and physiological concepts. This process has been continuous since the initial formulation of the model over 30 years ago (Asatryan & Feldman, 1965). As a result, the current model offers specific views on several areas: the definition of central commands or control variables; the principles of sensorimotor integration or the relationship between central control signals, reflexes, and biomechanical properties of the motor apparatus in movement production; the origin of electromyographic (EMG) patterns, forces and kinematics; the relationship between posture and movement; the redundancy problem in multimuscle and multijoint control; the coding of movement direction, speed, and distance; spatial frames of reference for movement control and kinesthesia; dynamical versus computational approaches to motor control; and applicability of linear models to movement analysis.

Although significant progress has been made, our ability to explain the neural control of movement remains limited. Nevertheless, the model has defined the

Anatol G. Feldman, Mindy F. Levin, and Arnold B. Mitnitski are with the Centre de Recherche, Institut de réadaptation, Université de Montréal, Montréal, PQ, Canada H3S 2J4. David J. Ostry and Paul L. Gribble are with McGill University, Department of Psychology, 1205 Dr. Penfield Ave., Montreal, PQ, Canada H3A 1B1.

minimal requirement for future theorizing in the field in the sense that it outlines a range of phenomena for which any full model must account. However, has the time actually come to abandon the λ model, as explicitly suggested by Gottlieb (1998)? We will show that rejections of the model have been flawed. Recent publications by Gottlieb (1995, 1998), Gomi and Kawato (1996), and Lackner and Dizio (1994) will be used to illustrate this point.

In the following sections we address a number of criticisms of the λ model. The criticisms can be essentially divided into two types. Some authors disagree with the definition and the organization of control patterns as reflected in the time-varying form of the equilibrium shift. Others question the ability of the model to explain the output characteristics of active movements such as EMG, kinematic, stiffness, and damping patterns. The issues of control patterns and output characteristics, although interrelated, are considered separately in the present paper.

Control Patterns Underlying Movement Production

The Dynamical Nature of the Concepts of Equilibrium State and Control Variables

Gottlieb (1995) proposed that a major idea of the λ model, that motor control is produced by shifts in the system's equilibrium state, is only applicable to the maintenance of posture or, at best, the production of slow movements. However, many examples of simulations of fast movements in the λ model have been reported (Feldman, Adamovich, Ostry, & Flanagan, 1990; Feldman & Levin, 1995; Flanagan, Ostry, & Feldman, 1993; Gribble, Ostry, Sanguineti, & Laboissière, 1998; St-Onge, Adamovich, & Feldman, 1997). A brief clarification of the dynamic nature of the concept of equilibrium state may be helpful.

We will begin by distinguishing between state variables and parameters of a physical system. In moving systems, state variables are kinematic variables (i.e., the current system's coordinates and their time derivatives) and any other variables dependent on them. Those quantities of the system that remain constant or may be changed independently of the state variables are called parameters.

By definition, the equilibrium state is the state of a moving system that may be achieved by nullifying the velocity of positional change. We refer to this state as a virtual one since the actual state of the system may never coincide with it, as for example in an oscillating pendulum. The concept of equilibrium state came to the λ model from physics, in which it is applied to the description of not only the static but also the dynamic behavior of the system (Glansdorf & Prigogine, 1971). The applicability to dynamics is justified since the forces generated in the system are substantially defined by the difference between the actual and the equilibrium values of state variables. According to the λ model, the nervous system has the capacity to intentionally create such a difference by modifying the equilibrium state of the neuromuscular system. The forces emerging from this process will produce movement.

Which factors determine the equilibrium state of a system? The answer to this question is based on the distinction between the state variables and parameters of the system. According to physical law, the equilibrium state is determined by the system's parameters, not by state variables. For many physical systems (a pendulum, a mass-spring device, an electrical generator, etc.), the equilibrium state is

unique; that is, it remains the same despite continuous changes in state variables. This is because the parameters that determine the equilibrium state in these systems are constant. For a pendulum, these are the coordinates of the suspension point, the length of the rope from which the mass is suspended, and the direction of gravity. Since these parameters are constant, the equilibrium state is not modified despite the system's motion. Also, the tension in the rope of the pendulum is a state variable since it depends on the current position of the mass. Consistent with the above law, the equilibrium position at any phase of motion is the same despite the changes in this variable. To change the equilibrium position in the pendulum, the observer may modify the parameters listed above with the obvious result that the system will make a transition to oscillate about a new equilibrium position.

The distinction between state variables and parameters is also essential in the λ model. In this model, state variables are variables describing the current motor output of the neuromuscular system. These are kinematic variables and any other variables dependent on them (e.g., muscle forces).

The λ model postulates that active movements, no matter how fast, may be produced by the nervous system only by changing the system parameters influencing its equilibrium state. The parameters determining the equilibrium state that are typically constants in nonliving systems thus become actively controlled variables in living systems. These variables are called control variables or central commands in the λ model.

Control variables differ from state variables since the latter cannot influence the system's equilibrium state. In living systems, an example of a state variable is EMG activity, since well-documented observations (e.g., Bigland & Lippold, 1954) show that it depends on kinematic variables (position and velocity). As a state variable, the EMG activity cannot itself influence the equilibrium state of the system.[1] The same is true for muscle forces related to EMG activity. In a linear mass-spring system, stiffness is a constant parameter. In neuromuscular systems, muscle stiffness depends on muscle length, which makes it a state variable not influencing the system's equilibrium state.

The concept of control variables implies that they can be changed independently of state variables and, in particular, remain invariant regardless of changes in state variables. The λ model started from the experimental demonstration (Asatryan & Feldman, 1965) of the capacity of the nervous system to maintain invariant values of control variables despite changes in elbow position and muscle torques elicited by unloading (the "do not intervene" paradigm). Hence the term *invariant torque/angle characteristic* was used.[2]

The λ model also defines the notion of the equilibrium point (EP), the combination of equilibrium values of joint angles with the associated values of load torques. As a consequence, not only control variables but also characteristics of load torques may determine the EP. The EP is thus an integral variable characterizing the interaction of all components of the system with the environment. Such understanding of the EP concept is helpful in explaining both movement and isometric torque production in the model (Feldman, 1986; Latash, 1993).

Load perturbations may or may not affect the EP of the system depending on the motor task and the parameters of the perturbation. For example, a steplike change in the load torque instantly changes the system's EP. Such changes are considered involuntary, whereas active changes in the EP elicited by control signals are considered voluntary. Control systems may or may not react to load per-

turbations. Each load perturbation is a multifaceted phenomenon, and the selection of an appropriate control strategy to deal with it depends on more than its mechanical aspects. For example, from a mechanical point of view, velocity-dependent perturbations do not affect the system's EP. However, even in this case, the system may actively modify control variables for the sake of, for example, movement stability. It is important to have this in mind when discussing the effects of Coriolis force perturbations on arm movements (see Movement Equifinality section).

The model defines control variables that modulate the transition from one equilibrium state to another or, generally, deal with system stability. Several control variables were defined in the model, for example, the reciprocal (R), coactivation (C), and μ commands. Of these, only the first is responsible for voluntary changes in the EP, whereas the transition process and system stability depend on them all. Changes in the C command, by definition, do not affect the existing EP or its active shifts elicited by the R command regardless of the external load or the asymmetries of agonist and antagonist muscle action. Involuntary changes in the EP elicited by an external force depend on the existing R and C commands (see the section Muscle Asymmetries in the λ Model).

Several conclusions related to this section can be made. The concept of the equilibrium state does not apply only to situations where muscle and external forces are balanced. Thus, the λ model applies to both slow and fast movement. The λ model also tells us that active movement production cannot be explained in terms of state variables (i.e., variables characterizing the motor output—EMG activity, muscle forces, torques, stiffness, damping, etc.). Studies showing correlations between cortical neuronal activity and state variables may be important in describing motor behavior. Explaining how behavior is controlled, however, requires an extension of the analysis beyond variables characterizing the system's motor output.

Monotonic Control Signals May Underlie Point-to-Point Arm Movements: Functional Significance

A number of empirical findings have been proposed supporting the idea that, in contrast to what is suggested in the λ model, the EP shifts specified by the nervous system to produce active arm movement have a "complex," nonmonotonic, time-varying form (Gomi & Kawato, 1996; Latash & Gottlieb, 1991). In particular, Gomi and Kawato (1996) analyzed the effects of small perturbations of arm movements from one position to another without corrections. Based on the assumption of the system's linearity, they experimentally estimated stiffness and damping of the limb and used these data to compute shifts in the equilibrium position of the arm. They concluded that the pattern of the shifts is complex and involves multiple velocity peaks, in contrast to what the λ model suggests.

A simple inspection of Gomi and Kawato's (1996) data reveals a problem in their method: Their Figure 3 shows shifts in the equilibrium position ending substantially (about 250 ms) after the end of movement. The result is paradoxical if one takes into account that, physically, a shift in the equilibrium state of the system provokes movement and, consequently, the movement can only cease after, not before the end of, the EP shift.

The paradoxical result of Gomi and Kawato brings into question the validity of their conclusion that the pattern of shift in the equilibrium position is complex, with multiple velocity peaks. Simultaneously, this questions the applicability of

linear methods to the description of motor system behavior (see the section Neuromuscular Systems Are Fundamentally Nonlinear). Indeed, Gribble et al. (1998) demonstrated that Gomi and Kawato's conclusion resulted from their simplified assumptions on the force-generating mechanism. Gribble et al. (1998) also showed that a monotonic EP shift might be sufficient to simulate Gomi and Kawato's empirical findings when an adequate force generating mechanism is used.

One of Gottlieb's (1998) arguments against the λ model is based on the observation of a nonmonotonic ("N-shaped") shift pattern of the net torque/angle characteristic during fast elbow point-to-point movements (Latash & Gottlieb, 1991). Assuming that shifts in the EP of the system are similar to those in the torque/angle characteristics, Latash and Gottlieb concluded that the EP shifts in fast movements are also N-shaped rather than monotonic. In contrast, Gribble et al. (1998) showed that monotonic EP shifts in the λ model produce N-shaped shifts in torque/angle characteristics similar to those shown by Latash and Gottlieb (1991). The difference in the two types of shifts can be explained in the following way.

According to the λ model, the shifts in the EP are defined by the R command producing independent changes in the agonist and antagonist thresholds, whereas the shifts in joint characteristic result from the superposition of these changes with the reflex changes in the thresholds that depend on movement speed. The latter is nonmonotonic (bell-shaped) for point-to-point movements. Because of the superposition of the two processes, the shifts in the joint characteristic may be N-shaped even though the EP shift defined by the R command is monotonic. In essence, this has been demonstrated by Gribble et al. (1998) by numerical simulation based on the λ model employing a monotonic shift in the EP in fast elbow movement.[3]

Additional arguments also reject the hypothesis that EP shifts first lead and then lag arm movements. First, the hypothesis is inconsistent with experimental estimation of the EP shift duration during fast movements (Feldman, Adamovich, & Levin, 1995). The experiment was based on the suggestion of the λ model that the motor response to the EP shifts in fast movements may substantially outlast the shifts because, in particular, of arm inertia and comparatively slow muscle and reflex reactions (see, e.g., Flanagan, Ostry, & Feldman, 1993). One can influence the interval between the end of the EP shifts and the end of the movement by using different loads. By applying an appropriate load, it is possible to estimate the duration of the EP shift. Experimentally, this was achieved by opposing the fastest 60° elbow flexion movement (peak velocity about 600°/s) with a springlike load in randomly selected trials (Feldman, Adamovich, & Levin, 1995). As a consequence, the limb moved less distance (10-15°) against the load. The movement and changes in torque were completed in about 100 ms rather than 300 ms in the absence of perturbation. It was unlikely that changes in control variables were responsible for the decrease in the movement time, since the latency of such changes is typically greater than 100 ms (e.g., Latash, 1993). Since the arm movement and changes in torques stopped after about 100 ms, so presumably did the EP: The EP shift is a motive force of movement and, if the EP had continued to change, a change in arm position and torques would have occurred after 100 ms. The experimental data suggest that the EP shifts are completed when the nonperturbed movement reaches its peak velocity.

Note that the demonstration of short-term EP shifts did not rely on any numerical model or such an assumption as the linearity of the system. This result is consistent with the λ model (Gribble et al., 1998; St-Onge et al., 1997) and incon-

sistent with the alternative hypotheses in which the EP shifts last until the end (Latash & Gottlieb, 1991) or, paradoxically, continue long after the end of the actual movement (Gomi & Kawato 1996).

Second, there have been no reports that a forward dynamic model based on the proposed complex EP shifts and simplified linear characteristics of force production may reproduce the empirical kinematic patterns of arm movements. In contrast, the endpoint trajectories, tangential velocity profiles, and stiffness data in single-joint or multijoint movements have been reproduced in a mathematical model based on a simple, monotonic pattern of EP shifts (Flanagan et al., 1993; Gribble et al., 1998). It was concluded from these simulations that the equilibrium and the actual endpoint trajectories for arm movements in a horizontal plane are close to each other (Figure 1A; see also Flanagan et al., 1993). Won and Hogan (1995) came to a similar conclusion after experimentally analyzing the effects of perturbations on arm movements.

Gottlieb's (1998) claim that the λ model incorrectly predicts the values of damping reported by Gomi and Osu (1996) is misleading. Gomi and Osu only reported damping values during the maintenance of a steady posture. These values (about 0.8 N · m · s/rad) match the values of the damping in the λ model for postural control (Gribble et al., 1998). By referring to the fast movement simulations by St-Onge et al. (1997), Gottlieb implied that the postural damping data (Gomi & Osu, 1996) are valid for dynamics, which is unlikely. Damping is affected, in particular, by the coactivation command in the λ model, which may be different for posture and movement (Gribble et al., 1998; St-Onge et al., 1997). For fast point-to-point movements, no empirical values of damping are known. Gomi and Kawato (1996) required movement damping data for their calculations of EP trajectories but did not report them. Latash and Gottlieb (1991) in their computations of EP trajectories assumed that joint torque varied with joint angle but was not dependent upon velocity (zero damping), a physiologically unrealistic assumption.

Nevertheless, based on two empirical studies (Feldman, Adamovich, & Levin, 1995; Gottlieb et al., 1989), it is not difficult to estimate damping values for fast point-to-point movements. The peak value of muscle torque during fast 60° elbow movements made with the instruction to "move as fast as possible" is about 15 N · m (Feldman, Adamovich, & Levin, 1995). In trials in which such movements are opposed by a stiff springlike load, the arm stops moving after 10-15°, about the time when the nonopposed movements reach their peak velocity (about 10 rad/s). In the opposed movements, the torque reaches 55 N · m (Feldman, Adamovich, & Levin, 1995). The difference between the two values of torque (40 N · m) is the torque lost due to muscle shortening. Because the changes in the joint angle and, consequently, muscle length in the opposed movements and in the nonopposed movements until peak velocity are similar, the loss in the joint torque at this point can be attributed to changes in velocity rather than in the amplitude of shortening. By dividing 40 N · m by movement peak velocity (about 10 rad/s), we get an estimate of the damping coefficient for fast movements: 4 N · m · s/rad.

With small modifications, this method allows us to estimate damping values using the data of Gottlieb, Corcos, and Agarwal (1989). They also measured peak values of velocity and torque during fast elbow movements, and did this for movements against different inertial loads. In their experiments, the smallest inertial load (0.18 kg · m^2) was substantially greater than in the experiment by Feldman et al. (1995; 0.05 kg · m^2). Therefore, their subjects typically moved slower and produced greater peak torques. For example, during 54° movement against the smallest inertial load, the mean peak velocity and torque were about 6 rad/s and 23 N · m, respectively (Figure 5A of Gottlieb et al., 1989). Unlike Feldman, Adamovich, and Levin (1995), they did not measure torques in arrested movements. Indirectly, however, the values of such torques may be estimated from their data. They showed that, for each movement distance, the movement peak velocity decreased and torque increased with the increasing load. The velocity/torque tradeoff was linear. By extrapolating this relationship to zero velocity, one can find the values of the muscle torques that subjects could develop if the movement were suddenly arrested. Using their Figure 5A, we found that the extrapolated, maximal torque for 54° movements was about 45 N · m. The torque lost due to muscle shortening was thus 22 N · m. Dividing 22 N · m by the movement peak velocity (6 rad/s) produces an estimate of the damping coefficient for fast 54° movements: 3.7 N · m · s/rad. The same method applied to other fast movements analyzed by Gottlieb et al. yields similar values of damping: 3.4 N · m · s/rad for 36° and 3.8 N · m · s/rad for 72° movements.

The estimates of damping based on different data are thus consistent and imply the existence of powerful damping mechanisms in the neuromuscular system. From a physical point of view, this is not surprising: Low damping is a recipe for system instability. Physiologically, damping mechanisms are well known: There is velocity-dependent force regulation at the level of sarcomeres as well as at velocity-dependent afferent feedback to motoneurons. These mechanisms are integrated in the λ model. In the model, damping depends on the μ command as well as the C command, which is likely scaled with the required movement speed (Gribble et al., 1998; St-Onge et al., 1997). Correspondingly, for slower, self-paced movements similar to those studied by Gomi and Kawato (1996), Gribble et al. (1998) obtained a lower damping value (2.5 N · m · s/rad) in their simulations using the λ model with a monotonic pattern of EP shifts.

There are several important implications of the suggested short-term, monotonic changes in control signals in simple point-to-point movements. First, this strategy relies on the capacity of the limb muscle mechanics and peripheral feedback to complete the movement without continuous supervision from control systems. Second, in the case of a movement error, the short-term control process may give the system time to prepare and initiate, if necessary, a corrective response before the end of the movement. Third, the system may rapidly generate a sequence of central commands for different movements without waiting for the end of the kinematic response to each movement component. This may be important for many motor behaviors, from piano playing to speech production. Fourth, the movement distance, speed, and duration can be easily coded by specifying the rate and duration of the monotonic control signal (see the section Central Commands, Reflexes, and EMG Patterns).

Movement Equifinality

The term *equifinality* has been used to describe a feature of the system occurring when the pattern of central commands remains unchanged (Asatryan & Feldman, 1965; Feldman & Levin, 1995). Under this assumption, a movement would end at the same final EP when unperturbed and following a transient (e.g., velocity-dependent) perturbation.

Reports of positional errors in arm pointing movements perturbed by velocity-dependent, Coriolis forces in a dark rotating room (Coello, OrLiaguet, & Prablanc, 1996; Lackner & Dizio, 1994) are another recent source of controversy associated with the λ model. Lackner and Dizio assumed that since the Coriolis force is velocity-dependent, it could not modify the final EP and thus no movement error would occur. Inconsistent with this assumption was Lackner and Dizio's (1994) finding of positional errors. This conclusion, however, only considers the mechanical aspects of the perturbation. We mentioned earlier that whereas mechanically, a velocity-dependent perturbation does not influence the system's EP, other aspects of the perturbation may force the control system to change the EP. According to the λ model, an essential requirement for equifinality is that the pattern of central commands underlying the movement remain the same regardless of the perturbation. Equifinality may thus not occur if perturbations alter the initial pattern of central commands.

A change in central commands may be responsible for the effects of perturbations elicited by Coriolis forces in Lackner and Dizio's experiments. Coriolis forces were likely sufficiently large to be perceived during movement and subjects could have reacted to the movement perturbations by changing central commands. Also note that the Coriolis force deflects the arm from the EP in proportion to movement velocity. This is in contrast to what happens when muscle forces react to deflections: They resist them. Coriolis forces thus belong to the family of antidamping, destabilizing perturbations. Control systems may be forced to actively react to such perturbations to preserve movement stability and restrict the arm deflections even though the price will be a positional error. From this point of view, positional errors resulting from Coriolis forces need not be inconsistent with the λ model (Feldman, Ostry, & Levin, 1995b; Figure 1). Thus, the finding of inequifinality does not justify the rejection of the λ model.

Figure 1 shows the effects of Coriolis force perturbations on arm movements in a dark rotating room, simulated in a forward dynamical model based on equations of Flanagan et al. (1993) modified to incorporate Coriolis forces. Dashed lines indicate the specified trajectories of shift in the equilibrium position of the hand. Solid lines indicate trajectories of hand movement resulting in the model from the shift. In the absence of Coriolis force (A), the shift in the equilibrium position along a straight line results in hand movement to the target. If the subject were able to produce the same shift during the application of Coriolis force without correction (B), the resulting hand trajectory would be substantially deflected from the straight line but eventually the hand would arrive at the target (filled square). It is assumed that subjects may not tolerate such deflections of the arm from the intended movement direction and produce corrections in the first (C) and subsequent (D) trials during rotation of the room. These corrections are made by changing the direction of the shift in the equilibrium position. In the first trial during rotation (C), the subject changes the sagittal direction of the shift to the frontal, coinciding with the direction of Coriolis force. As a result, the arm stops before it arrives at the target but this strategy introduces a positional and a directional movement error. In the process of adaptation (D), the directional error is eliminated by trial-to-trial incremental rotations of the equilibrium trajectory as a whole in a counterclockwise direction. In the first post-rotation trial (E), the subject initially reproduces the remembered direction of the equilibrium trajectory used during room rotation after adaptation and then produces a correction making

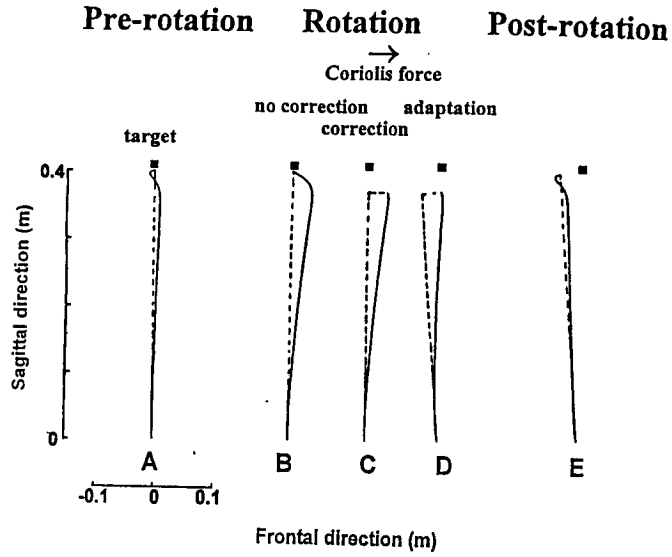

Figure 1 — Effects of Coriolis force perturbations on arm movements in a dark rotating room.

the trajectory straight. Compared to the first trial during rotation (C), this results in a reversal of positional and directional errors. Movement adaptation in subsequent post-rotational trials eliminating these errors is produced by rotation of the equilibrium trajectory clockwise (not illustrated). The model makes the testable prediction that after adaptation during rotation (D), subjects may still undershoot the target (compare with A) and thus, despite movement corrections, the compensation of errors may be incomplete. An undershoot can actually be seen in the empirical data by Lackner and Dizio (1994).

The case in which a transient perturbation results in the loss of equifinality was reported long ago in a Russian book by Feldman (1979; reproduced in Feldman & Levin, 1995, Figure 4). In this case, active elbow flexors were unloaded and then abruptly reloaded. The elbow did not return to the initial position even though the initial and the final loads were the same. This finding showed that subjects are more sensitive to loading and less able not to intervene than in the case of unloading, as was also noticed by Crago, Houk, and Hasan (1976). Indeed, this observation has not been considered as inconsistent with the λ model. The conclusion was that regardless of the instruction given to the subject, control systems might not tolerate perturbations when loading speed is above a specific threshold. In addition, Van Emmerik (1992) analyzed drawing movements perturbed by forces created by an electromagnet and found that equifinality may not occur when the perturbation force is above a specific threshold value.

Muscle Asymmetries in the λ Model

Gottlieb (1998) argued that the λ model should be rejected because of problems with the definition of the coactivation (C) command. Specifically, he stated that

the C command, as it is presently defined, affects the EP of the system because of muscle asymmetries. He also claimed that the problems associated with these asymmetries have never been addressed. Actually, it was demonstrated in several publications how the R and C commands may be defined for groups of muscles in the arm, jaw, and even tongue systems having complex geometry and asymmetric actions (Feldman, 1993; Feldman & Levin, 1995; Gribble & Ostry, 1996; Gribble, Ostry, & Laboissière, 1997; Gribble et al., 1998; Laboissière, Ostry, & Feldman, 1996; Ostry, Gribble, & Gracco, 1996; Sanguineti, Laboissière, & Payan, 1997).

The C command in the λ model is defined as an independent, control variable not influencing the existing EP of the system or active shifts in the EP elicited by the R command. This definition is based on experimental observations. For example, ask a subject to hold a heavy book at a given elbow position. This task is associated with a strong asymmetry in the action of agonist and antagonist muscle groups (agonists are active whereas antagonists are slack). Then, tell the subject to produce agonist/antagonist coactivation. While doing this, the subject usually maintains the same arm posture. This is easily done at different arm configurations despite changes in muscle actions. Another observation is that after fast arm movements to a final position, strong tonic agonist/antagonist coactivation occurs, gradually diminishing without changes in the arm position. These observations are consistent with the idea that subjects can modulate the magnitude of a C command that increases muscle coactivation without affecting the existing EP of the limb, even in the presence of muscle asymmetries.[5]

Gottlieb's criticism of the inadequacy of the C command has been offered in reference to simplified models in which we have explicitly assumed that muscle moment arms and force-generating abilities are equal. However, the simple relationship between the R and C command and thresholds valid for this case (the R command as the sum of, and the C command as the difference between, the agonist and antagonist thresholds) should not be regarded as the definition in the systems with muscle asymmetries.

The important question of how the nervous system specifies the thresholds of agonist and antagonist muscles to produce the C command without modification of the existing EP despite muscle asymmetries has been addressed in the context of the λ model. In our arm and jaw models (Gribble & Ostry, 1996; Gribble et al., 1997, 1998; Laboissière et al., 1996; Sanguineti, Laboissière, & Ostry, in press), control signals to muscles were organized to ensure the functional independence of R and C commands. In these models, we explicitly accounted for muscle force-generating asymmetries and asymmetrical muscle moment arms in the definition of the R and C commands. Central commands were defined as specifying different relationships between the activation thresholds of all muscles. For example, in the jaw-hyoid system, one R command produces jaw rotation and the other jaw translation. There are also R commands that produce hyoid motion. By combining these commands, one can specify any configuration of the jaw-hyoid system. In our recent multi–degree of freedom models, the C command was defined as the set of lambda shifts that result in equal increases in agonist and antagonist torques at a given limb configuration. This formulation as well as alternate assumptions about the organization of control signals underlying the R and C commands is testable on the basis of EMG activity patterns.

Although in our arm and jaw models we have assumed that muscle and moment arm asymmetries are accounted for in establishing control signals

subserving R and C commands, it is also possible that control systems influence agonist and antagonist muscles as if they were symmetrical structures. Segmental interneurons may adjust influences to motoneurons, depending on proprioceptive signals carrying information about muscle asymmetries and may prevent changes in the system's EP. In this context, a neural network was developed to explore these ideas (Feldman, 1993). The network integrates afferent inputs from muscle spindle and tendon organ afferents, presynaptic and recurrent inhibition, and supraspinal inputs to motoneurons. The phenomenon of "reflex reversal" is also a feature of the network. The neural network may be verified by electrophysiological methods.

Finally, in some cases, the effect of mechanical asymmetries of flexor/extensor muscles may be small. For example, for arm movements in a horizontal plane, the influence of gravity is minimal. If active movements are produced, the arm, despite flexor and extensor asymmetries, arrives at a final position at which the action of these muscle groups is symmetrical: They create equal and opposite torques. Moreover, numerical simulations show that the mechanical asymmetries may have little effect on rapid single-joint movements against zero loads. For example, St-Onge et al. (1997) reported that changes in the velocity profiles of fast elbow movements elicited by the same central commands in different biomechanical structures (one with symmetrical flexor/extensor properties and the other with asymmetrical properties originating from position-dependent changes in the flexor moment arms when the extensor moment arm is constant) were insignificant.

Two factors contribute to the decrease of the effects of biomechanical asymmetries on movement in the model. First, the natural tendency of the system is to balance the action of agonist muscles, antagonist muscles, and loads. Second, the C command employed in fast movements tends to add equivalent agonist and antagonist torques. The decreasing effect of muscle asymmetries is an interesting aspect of the model, implying that the nervous system in some cases may control muscles as if they were symmetrical structures.

Central Commands and Movement Characteristics

Central Commands, Reflexes, and EMG Patterns

Gottlieb (1998) claimed that the existing version of the λ model does not explain "movement features such as distance, speed, and load." The specification of movement distance, speed, and muscle forces was explained, in a straightforward way, in the framework of the λ model based on a monotonic shift in the arm EP (Feldman & Levin, 1995; Feldman et al., 1990; Flanagan et al., 1993; St-Onge et al., 1997). The movement distance is defined by the magnitude of the EP shift. The shift is defined by two parameters: the velocity and duration of the shift. These parameters as well as the C and μ commands also influence movement speed. The velocity of EP shift is a vector. The control systems may change the direction of the EP shift while its rate and time may remain the same. The resulting movement direction but not distance will be changed. On the other hand, the system may only change the rate of EP shift. In this case, only movement distance and not its direction will be affected. Thus, the possibility of independent coding of arm movement distance and direction (Messier & Kalaska, 1997) is also integrated in the model.

Similarly, the model accounts for "speed-sensitive" and "speed-insensitive" strategies described by Gottlieb et al. (1989), phenomena initially observed by Wadman, Denier van der Gon, Geuze, and Mol (1979). To produce arm movements that differ in terms of distance, control systems in the model may vary the time of the EP shift while preserving its direction and rate and values of other control variables. The control patterns for different movements thus are initially identical and then diverge. As a consequence, the trajectories of resulting movements and EMG patterns will also be identical in an initial phase. This is a "speed-insensitive" strategy. On the other hand, suppose it is necessary to make a movement to the same target but at different speeds. Then the rate and duration of the EP shift should be varied in a reciprocal manner. The resulting trajectories will diverge from the start of movement ("speed-sensitive" strategy). The λ model suggests that these strategies are not unique in movement production and are consequences of the more basic phenomenon of a monotonic EP shift (Feldman et al., 1990; St-Onge et al., 1997).

A number of additional criticisms of the model have been reported, but they are not consistent with recent experimental findings.

First Example. Muscle spindles in the jaw-opener muscles are few in comparison with the jaw-closer muscles (see Rowlerson, 1990, for review), and it has been suggested that the stretch reflex plays a minimal role in regulating their activity (see Luschei & Goldberg, 1981, for summary). This suggestion conflicts with the λ model, in which central control processes are associated with changes in reflex parameters such as the threshold of the stretch reflex. The conclusion may follow that the model is inapplicable to the jaw system.

Alternatively, one can reinvestigate the reflex behavior in the jaw system using the "do not intervene" paradigm (Asatryan & Feldman, 1965). Using this method, robust phasic and tonic stretch reflexes perhaps mediated by afferents other than muscle spindles have recently been revealed in jaw opener muscles (Ostry, Levin, & Feldman, 1997). Thus, the applicability of the λ model to the jaw system is not in question.

There are experimental data implying that the stretch reflex is absent in extrinsic eye muscles despite the presence of a sufficient number of muscle spindles (Robinson, 1981). Since we know that a similar suggestion regarding the jaw-opener muscles has proven to be incorrect, additional experimental analyses of stretch responses in extrinsic eye muscles would seem appropriate before deciding on the applicability of the λ model to the oculomotor system.

Second Example. It was observed that the onset time of the first agonist burst in fast arm movement does not depend on the stretch reflex; that is, the onset is insensitive to external perturbations that lengthen or shorten the muscle (Brown & Cooke, 1986). This finding supports the notion that EMG activity may be directly programmed in the nervous system. In contrast, the λ model suggests that central control signals do not carry information on the timing and magnitude of EMG bursts and that these patterns emerge from the interaction of central control signals, reflex components, and biomechanical components of the system. As a consequence, the model predicts that the onset time of the first agonist burst is stretch reflex–dependent. However, to observe this effect, specific characteristics of perturbation are required. Since the model shows that the rate of central changes in the stretch-reflex threshold underlying fast movements is high (700°/s; see, e.g., St-Onge et al., 1997), changes in the onset time of the first EMG bursts can only be obtained with high-speed perturbations (>100°/s). Recently, experimental data consistent with this prediction have been reported for the fastest movement (Adamovich, Levin, & Feldman, 1997). Rapid perturbations applied 50 ms before the onset of movement elicited robust stretch-reflex–mediated modifications of the onset time of the first agonist as well as other EMG bursts in the fastest elbow flexion movements.

Third Example. A triphasic EMG pattern characteristic of fast arm movement can be found in trials in which the movement is suddenly arrested (Ghez & Gordon, 1987; Latash, 1993). This finding is usually also considered as evidence that the EMG pattern is basically preprogrammed.

The model actually predicts that a triphasic EMG pattern may be found in arrested movements whose amplitude is less than about 50°. If the amplitude is greater than that, the antagonist EMG burst will be suppressed, whereas the first agonist burst will not be terminated (Feldman, Adamovich, & Levin, 1995). This effect has been demonstrated in a recent empirical study by arresting the elbow movements with a strong springlike load created by a torque motor (Feldman, Adamovich, & Levin, 1995). Consistent with the model, it simultaneously promotes the notion of the absence of central programming of EMG bursts.

Neuromuscular Systems Are Fundamentally Nonlinear

Mathematically, the well-established threshold properties of motoneurons and other neuronal elements make the motor system fundamentally nonlinear. In other words, it cannot be considered linear even locally, for small changes in variables. The reflex delay is also a nonlinear element of the system. The invariant characteristics of muscles are also substantially nonlinear. In contrast, not only local but also global linearity of the system was assumed in Gomi and Kawato's (1996) computations of equilibrium trajectories in pointing movements. These simplifications contribute to their paradoxical finding (shifts in the EP end after the end of movement).

There are additional problems with Gomi and Kawato's computational method. They assumed that muscle torques are only functions of position, velocity, and control signals. They thus ignored time- and history-dependent generation of muscle torques, that is, gradual changes of muscle torques in response to muscle activation or deactivation that occurs even if other variables are fixed.

In addition, the physical definition of stiffness implies computation of partial derivatives of muscle torques with respect to position for zero values of velocity and fixed values of control signals. Gomi and Kawato estimated these derivatives but did not extrapolate them to the values resembling zero velocity. Each experimental point was obtained by averaging kinematic and torque data over a period of 300 ms. The change in control variables during this time could affect the measurement. Gomi and Kawato thus obtained some quantities that cannot be called stiffness and damping without reservations. The error in the computation of the EP shifts based on these data may be substantial, as was demonstrated by Gribble et al. (1998). In a model employing a simple, monotonic EP shift, Gribble et al. produced computations according to the algorithm of Gomi and Kawato (1996) and obtained nonmonotonic trajectories resembling those considered by Gomi and Kawato as EP trajectories. The nonmonotonic shape of the EP shifts may thus be considered an artifact of the algorithm.

The measurement of stiffness and damping in motor systems is presently based on analogies between the nonlinear motor system and linear springlike sys-

tems. The λ model not only questions these analogies but shifts the focus from these biomechanical variables to variables (R, C, and μ commands) more directly controlling the system's dynamic properties (see, e.g., Gribble et al., 1998, and St-Onge et al., 1997, for analysis of the sensitivity of the model's dynamics to these commands).

Concluding Remarks

There are different options for testing the model's validity, as described in several sections of this paper. Recent publications by Ostry's group (Gribble & Ostry, 1996; Gribble et al., 1998) in which they reproduced stiffness and damping data reported in other studies are also examples of testing of the model. In our response to the commentaries on our target article (Feldman & Levin, 1995), we suggested some critical tests of the model. If they were invalid, it would be a challenge to come up with something better than the existing model. If the predictions are justified, it is reasonable to keep the model and to formulate and test other predictions. The development of the model is not finished, and some aspects of the model can only be elaborated upon based on the results of appropriate experiments. Indeed, testing and developing the model require a complete understanding of its fundamentals and the ability to use its language to explain different motor phenomena. This language may be helpful in the systematic analysis of motor control. One might even say that rumors of the model's "death" have been slightly exaggerated.

References

Adamovich, S.V., Levin, M.F., & Feldman, A.G. (1997). Central modification of reflex parameters may underlie the fastest arm movements. *Journal of Neurophysiology*, **77**, 1460-1469.

Asatryan, D.G., & Feldman, A.G. (1965). Functional tuning of the nervous system with control of movement or maintenance of a steady posture: I. Mechanographic analysis of the work of the limb on execution of a postural task. *Biophysics*, **10**, 925-935.

Bigland, B., & Lippold, O.C.J. (1954). The relationship between force, velocity and integrated electrical activity in human muscle. *Journal of Physiology (London)*, **123**, 214-224.

Bizzi, E., Hogan, N., Mussa-Ivaldi, F.A., & Giszter, S. (1992). Does the nervous system use equilibrium-point control to guide single and multiple joint movements? *Behavioral and Brain Sciences*, **15**, 603-613.

Brown S., & Cooke, D. (1986). The initial agonist burst is modified by perturbations preceding movement. *Brain Research*, **377**, 311-322.

Coello, Y., OrLiaguet, J.P., & Prablanc, C. (1996). Pointing movements in artificial perturbing inertial field: A prospective paradigm for motor control study. *Neuropsychologia*, **34**, 879-892.

Crago, P., Houk, J.C., & Hasan, Z. (1976). Regulatory action of human stretch reflex. *Journal of Neurophysiology*, **39**, 925-935.

Feldman, A.G. (1979). *Central and reflex mechanisms in the control of movement* (in Russian). Moscow: Nauka.

Feldman, A.G. (1980) Superposition of motor programs: I. Rhythmic forearm movements in man. *Neuroscience*, **5**, 81-90.

Feldman, A.G. (1986). Once more on the equilibrium-point (λ model) for motor control. *Journal of Motor Behavior*, **18**, 17-54.

Feldman, A.G. (1993). The coactivation command for antagonist muscles involving Ib interneurons in mammalian motor control systems: An electrophysiologically testable model. *Neuroscience Letters*, **155**, 167-170.

Feldman, A.G., Adamovich, S.V., & Levin, M.L. (1995). The relationship between control, kinematic and electromyographic variables in fast single-joint movements in humans. *Experimental Brain Research*, **103**, 440-450.

Feldman, A.G., Adamovich, S.V., Ostry, D.J., & Flanagan, J.R. (1990). The origin of electromyograms—Explanations based on the equilibrium point hypothesis. In J.M. Winters & S.L-Y. Woo (Eds.), *Multiple muscle systems: Biomechanics and movement organization* (195-213). New York: Springer-Verlag.

Feldman, A.G., & Levin, M.L. (1993). Control variables and related concepts in motor control. *Concepts in Neuroscience*, **4**, 25-51.

Feldman, A.G., & Levin, M.L. (1995). The origin and use of positional frames of reference in motor control. *Behavioral and Brain Sciences*, **18**, 723-806.

Feldman, A.G., Ostry, D.J., & Levin, M.F. (1995). Velocity-dependent Coriolis force perturbations: An explanation of the positional errors and adaptation. *Neuroscience Abstracts*, **21**, 681.

Flanagan, J.R., Ostry, D.J., & Feldman, A.G. (1993). Control of trajectory modifications in target-directed reaching. *Journal of Motor Behavior*, **25**, 140-152.

Ghez, C., & Gordon, J. (1987). Trajectory control in targeted force impulses: I. Role of opposing muscles. *Experimental Brain Research*, **67**, 225-240.

Gielen, C.C.A.M., & Houk, J. (1986). Simple changes in reflex threshold cannot explain all aspects of rapid voluntary movements. *Behavioral and Brain Sciences*, **25**, 605-607.

Glansdorf, P., & Prigogine, I. (1971). *Thermodynamic theory of structure, stability and fluctuations*. London, New York: Wiley.

Gomi, H., & Kawato, M. (1996). Equilibrium-point control hypothesis examined by measured arm stiffness during multi-joint movement. *Science*, **272**, 117-120.

Gomi, H., & Osu, R. (1996). Human arm stiffness and viscosity in interaction with environment on a horizontal plane. *Nippon Telegraph and Telephone* (ISRL-96-3). Kanigawa: Atsugi.

Gottlieb, G.L. (1995). Shifting frame of reference but the same old point of view. *Behavioral and Brain Sciences*, **18**, 758.

Gottlieb, G.L. (1998). Rejecting the equilibrium-point hypothesis. *Motor Control*, **2**, 10-12.

Gottlieb, G.L., & Agarwal, G. (1988). Compliance of single joints: Elastic and plastic characteristics. *Journal of Neurophysiology*, **59**, 937-951.

Gottlieb, G.L., Corcos, D.M., & Agarwal G.C. (1989). Organizing principles for single joint movements: I. A speed-insensitive strategy. *Journal of Neurophysiology*, **62**, 342-357.

Gribble, P.L., & Ostry, D.J. (1996). Origins of the power law relation between movement velocity and curvature: Modeling the effects of muscle mechanics and limb dynamics. *Journal of Neurophysiology*, **76**, 2853-2860.

Gribble, P.L., Ostry, D.J., & Laboissière, R. (1997). Control of human arm and jaw motion: Issues related to musculo-skeletal geometry. In P.G. Morasso & V. Sanguineti (Eds.), *Self-organization, computational maps and motor control* (Advances in Psychology Series, vol. 119). Amsterdam: Elsevier-North Holland.

Gribble, P.L., Ostry, D.J., Sanguineti, V., & Laboissière, R. (1998). Are complex control signals required for human arm movement? *Journal of Neurophysiology*, **79**, 1409-1424.

Laboissière, R., Ostry, D.J., & Feldman, A.G. (1996). Control of multi-joint muscle systems: Human jaw and hyoid movements. *Biological Cybernetics*, **74**, 373-384.

Lackner, J.R., & Dizio, P. (1994). Rapid adaptation to Coriolis force perturbations of arm trajectory. *Journal of Neurophysiology*, **72**, 1-15.

Latash, M.L. (1993). *Control of human movement*. Champaign, IL: Human Kinetics.

Latash, M.L., & Gottlieb, G. (1991). Reconstruction of shifting elbow joint characteristics during fast and slow movements. *Neuroscience*, **47**, 357-365.

Luschei, E.S., & Goldberg, L.J. (1981). Neural mechanisms of mandibular control: Mastication and voluntary biting. In V.B. Brook (Ed.), *Motor control* (Handbook of physiology, Section 1. The nervous system, Vol. II, Part 2, pp. 1237-1274). Bethesda, MD: American Physiological Society.

Matthews, P.B.C. (1959). The dependence of tension upon extension in the stretch reflex of the soleus muscle in the decerebrated cat. *Journal of Physiology (London)*, **147**, 521-546.

Messier, J., & Kalaska, J. (1997). Differential effect of task conditions on errors of direction and extent of reaching movements. *Experimental Brain Research*, **115**, 469-478.

Ostry, D.J., Gribble, P.L., & Gracco, V.L. (1996). Coarticulation of jaw movements in speech production: Is context-sensitivity in speech kinematics centrally planned? *Journal of Neuroscience*, **16**, 1570-1579.

Ostry, D.J., Gribble, P.L., Levin, M.F., & Feldman, A.G. (1997). Phasic and tonic stretch reflexes in muscles with few muscle spindles: Human jaw-opener muscles. *Experimental Brain Research*, **116**, 2999-3008.

Robinson, D.A. (1981). Control of eye movements. In V.B. Brook (Ed.), *Motor control* (Handbook of physiology, Section 1. The nervous system, Vol. II, Part 2, pp. 1237-1274). Bethesda, MD: American Physiological Society.

Rowlerson, A.M. (1990). Specialization of mammalian jaw muscles: Fibre type composition and the distribution of muscle spindles. In A. Taylor (Ed.), *Neurophysiology of the jaw and teeth* (pp. 1-51). London: Macmillan.

Sanguineti, V., Laboissière, R., & Ostry, D.J. (in press). A dynamic biomechanical model for the neural control of speech production. *Journal of Acoustical Society of America*.

Sanguineti, V., Laboissière, R., & Payan, Y. (1997). A control model of human tongue movements in speech. *Biological Cybernetics*, **77**, 11-22.

St-Onge, N., Adamovich, S.V., & Feldman, A.G. (1997). Control processes underlying elbow flexion movements may be independent of kinematic and electromyographic patterns: Experimental study and modeling. *Neuroscience*, **79**, 295-316.

Van Emmerik, R.E.A. (1992). Kinematic adaptation to perturbations as a function of practice in rhythmic drawing movements *Journal of Motor Behavior*, **24**, 117-131.

Wadman, W.J., Denier van der Gon, J.J., Geuze, R.H., & Mol, C.R. (1979). Control of fast goal-directed arm movements. *Journal of Human Movement Studies*, **5**, 3-17.

Won, J., & Hogan, N. (1995). Stability properties of human reaching movements. *Experimental Brain Research*, **107**, 125-136.

Zatsiorsky, V.M. (1997). On muscle and joint viscosity. *Motor Control*, **1**, 299-309.

Notes

[1]This is a major point of disagreement between the λ model and another version of the EP hypothesis—the α model (Bizzi, Hogan, Mussa-Ivaldi, & Giszter, 1992) in which EP shifts are determined by changes in the magnitude of muscle activation.

[2]In some studies it was mistakenly assumed (e.g., Gottlieb & Agarwal, 1988) that the phrase "invariant characteristic of the joint" implies that the shape of the characteristic remains invariant when the characteristic is shifted by control systems. The change in the shape is basically related to the differences in muscle moment arms and passive characteristics in different parts of the biomechanical range (Feldman, 1979).

[3]Note that the above consideration also implies that despite monotonic changes of activation thresholds by the R command, the overall changes in the thresholds are nonmonotonic, an experimentally testable consequence of the λ model. Gielen and Houk (1986) also noticed that the explanation of the triphasic EMG pattern in the model presupposes a nonmonotonic change in the stretch reflex thresholds.

[4]The physical definition of stiffness implies measurement of partial derivatives of muscle torques with respect to position for zero values of velocity and fixed values of control signals. The definition of damping implies measurement of partial derivatives of muscle force with respect to velocity, also for fixed values of control signals. Whether the quantities measured by Gomi and Kawato (1996) are consistent with these definitions is discussed in the section Neuromuscular Systems Are Fundamentally Nonlinear. In addition, we replace their term *viscosity* with *damping*. According to Zatsiorsky (1997), viscosity refers to friction between the layers of fluid, a property that may not be entirely applicable to the neuromuscular system.

[5]A change in the EP may be elicited not only voluntarily, by an R command, but also involuntarily, by a change in the external force or load. The involuntary change in the EP, indeed, depends on the existing C command since this command restricts the arm displacements elicited by external forces. However, the new EP may be preserved if a new C command is applied. One may thus say that the C command represents an adaptive mechanism discriminating between voluntary and involuntary actions (Feldman, 1993).

Acknowledgments

We thank Mark Latash for helpful comments on the manuscript. This research was supported by NSERC-Canada and FCAR-Québec-Canada.

Accepted for publication: February 18, 1998