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Article *in* Journal of Motor Behavior · August 2013

DOI: 10.1080/00222895.1986.10735369

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Journal of Motor Behavior
1986, Vol. 18, No. 1, 17-54

Once More on the Equilibrium-Point Hypothesis (λ Model) for Motor Control

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ABSTRACT. The equilibrium control hypothesis (λ model) is considered with special reference to the following concepts: (a) the length-force invariant characteristic (IC) of the muscle together with central and reflex systems subserving its activity; (b) the tonic stretch reflex threshold (λ) as an independent measure of central commands descending to alpha and gamma motoneurons; (c) the equilibrium point, defined in terms of λ, IC and static load characteristics, which is associated with the notion that posture and movement are controlled by a single mechanism; and (d) the muscle activation area (a reformulation of the "size principle")—the area of kinematic and command variables in which a rank-ordered recruitment of motor units takes place. The model is used for the interpretation of various motor phenomena, particularly electromyographic patterns.

The stretch reflex in the λ model has no mechanism to follow-up a certain muscle length prescribed by central commands. Rather, its task is to bring the system to an equilibrium, load-dependent position. Another currently popular version defines the equilibrium point concept in terms of alpha motoneuron activity alone (the α model). Although the model imitates (as does the λ model) spring-like properties of motor performance, it nevertheless is inconsistent with a substantial data base on intact motor control. An analysis of α models, including their treatment of motor performance in deafferented animals, reveals that they suffer from grave shortcomings. It is concluded that parameterization of the stretch reflex is a basis for intact motor control. Muscle deafferentation impairs this graceful mechanism though it does not remove the possibility of movement.

1.0 Introduction

THE EQUILIBRIUM-POINT (EP) HYPOTHESIS suggests that active movements may be the result of shifts in the equilibrium state of the motor system. The hypothesis in its original formulation associates the shifts with setting the threshold (λ) of the stretch reflex. Originally the λ model emerged as a result of a number of experimental studies

This article was written as the result of comments by Drs. David Cooke, Scott Kelso, and Elliot Saltzman concerning another article (Adamovich & Feldman, manuscript in preparation) and numerous discussions with my colleagues, Mike Berkinblit, Serge Adamovich, Nina Burlachkova, Rustam Abdusamatov, and Alex Chernavsky among others. I would like to express my gratitude to all of them.

(Asatryan & Feldman, 1965; Feldman, 1966 a, 1966 b). Later it was presented as a complete system (Feldman 1974 a, 1974 b, 1976, 1979). The model attracted investigators' attention but was unevenly interpreted (cf. Bizzi, Polit, & Morasso, 1976; Bizzi, 1980; Cooke, 1980; Enoka 1983; Hogan, 1984; Houk, 1976; Kelso, 1977; Kelso & Holt, 1980; Nichols & Houk, 1976; Schmidt, 1980; Simmons & Richardson, 1984; Vincken, Gielen, & Danier van der Gon, 1983).

A number of ideas and concepts provided the underpinnings for the λ model, in particular: (a) the concept of invariant characteristics (ICs) associated with the fact that the stretch reflex threshold is an independent measure of central commands descending to alpha and gamma motoneurons; thus, the nervous system uses the reflex to perform movements; (b) the idea that muscle together with reflex and central control mechanisms behaves like a nonlinear spring, zero length of which is a controllable parameter; (c) the EP concept and its consequence that posture and movement are controlled by a single mechanism; (d) the idea that the nervous system specifies the velocity of EP shifts to alter movement speed (Feldman, 1974 b, 1979, 1981; Adamovich & Feldman, 1984; Adamovich, Burlachkova, & Feldman, 1984); and (e) the notion of muscle activation area—the area of kinematic and command variables in which the muscle undergoes activation (Feldman, 1974 a, 1974 b, 1979). It seems to me (and the argument will be elaborated in this article) that (a) and (b) were adequately perceived and further developed by Houk, Nichols, and other research workers (Davis & Kelso, 1982; Houk, 1976; 1979; Kelso, Holt, Kugler & Turvey, 1980; Kugler, Kelso & Turvey, 1980; Nichols, 1982; Nichols & Houk, 1976; Simmons & Richardson, 1984; Vincken et al., 1983), (b) and (c) were popularized by Bizzi and colleagues working originally with monkeys (Bizzi, 1980; Bizzi, Accornero, Chapple & Hogan, 1982; Bizzi et al., 1976; Hogan, 1984), and independently by Kelso and others working with humans (e.g., Kelso, 1977; Kelso & Holt, 1980; Schmidt, 1980; Cooke, 1980; see also Keele, 1981 for review). In numerous elegant experiments they have illustrated, in particular, the spring-like behavior of the motor system. Bizzi, however, elaborated his own version of the EP hypothesis based, instead of (a), on the notion of muscle length/force characteristics each corresponding to an invariant level of muscle α innervation. The model was represented in such a form as if the α were an independent control variable. Rendering Bizzi his due, I, nevertheless, intend to show that his α model, as far as intact motor control is concerned, gives rise to a misinterpretation of the spring analog and EP concept. In addition, concept (d) concerning the timing of central commands was not noticed, and a similar concept was formulated anew but in terms of the α model (Bizzi et al., 1982; Hogan, 1984).[1]

Concept (e) that seems to be a key to the explanation of EMG patterns has not been perceived by investigators at all. Essentially, the λ model is a logically consistent system of motor control principles (a)-(e), with attention focused only on a part of the system, thus narrowing the extent of its explanatory and predicative power.

The aim of the present article is to clarify the main aspects of the λ model and to illustrate its capacity for the interpretation of various motor phenomena, in particular, EMG patterns. The model is formulated gradually, firstly addressing static, postural aspects and then more complex, dynamic aspects of motor control. Unless otherwise specified, a muscle together with operating central-reflex mechanisms is considered. All the forces that counteract the muscle are considered to constitute its "load." Antagonist muscles can contribute to the load but for the aim of simplicity the load is considered a passive component of the system. This reservation is somewhat weakened by the fact that various sorts of loads are considered. The control of agonist-antagonist coupled muscles is described in a more direct form elsewhere (Adamovich & Feldman, manuscript in preparation).

Some aspects of the equilibrium control hypothesis and its alternative version have recently been discussed in a brief outline (Berkinblit, Feldman, & Fukson, in press), and the reader may also wish to consult that article.

2.0 Two Classes of Motor Processes

The theory presented below concerns chiefly the principles underlying voluntary and involuntary control over muscle force and length (or torque and joint angle) in intact organisms. These control processes can be distinguished on the basis of a rigorous, mathematical definition.

Voluntary movements are elicited by a modification of muscle activity and force. However, the central nervous system provides the effects indirectly, via regulation of some other variables (e.g., Bernstein, 1967). A number of such variables influence muscle force in statics. At least for certain type of motor tasks, only those that are or can be independent of each other—as well as of muscle force—are of interest since it is desirable: (i) to exclude an indirect duplication of the variables (e.g., variables related in one-to-one fashion constitute, in essence, a single variable) and (ii) to discern clearly cause (independent variables) and effect (dependent variables) in motor processes.

The static muscle force (F) depends on the muscle length x because of the stretch reflex (since motoneuron activity and muscle force increase with muscle length) and because of elasticity of active muscle fibres. If the length were the only independent variable affecting static muscle force, the muscle would behave like an ordinary spring changing its equilibrium length strictly dependent on the magnitude of the load. For comparison, stretching a muscle in the decerebrate cat results in the recruitment of motor units, thus producing an increase in muscle force and stiffness in accordance with the mechanism of the stretch reflex (Feldman & Orlovsky, 1972; Hoffer & Andreassen, 1981; Houk, 1976; Matthews, 1959). However, despite the control complexities, the behavior of the decerebrate preparation is fairly primitive from a functional point of view: For example, changing the muscle length for a particular load is not possible. Thus, any voluntary change in muscle length

in intact organisms is a manifestation of an independent control process, that is, independent of both the force and length, over some variable or parameter (λ) essentially influencing muscle force. Consequently, in statics

$$F = f(x,\lambda) \quad (1)$$

In contrast, an involuntary regulation of muscle length and force is elicited by external load perturbations. Thereby the yet unknown variable or parameter λ may remain constant (cf. section on "Trigger reactions").

In dynamics, the muscle force depends on additional variables, for example, on the velocity v of muscle lengthening (Hill, 1938) and, possibly, on one more controllable variable, μ, affecting the dependence of F on v (see below). However, the principle distinguishing voluntary and involuntary motor processes remains the same: the former manifest themselves in independent centrol control over the "internal" variables λ and μ, whereas the latter are elicited by changes in the "external," kinematic variables x and v.

Although the above classification may seem somewhat trivial, it provides initial criteria for accepting or rejecting a model of motor control. For example, models in which voluntary motor control in intact organisms is associated with the specification of overall level (α) of muscle efferent activity do not seem to meet these criteria since there is no indication that the nervous system can select this variable without regard to muscle force or length (Bernstein, 1967; Stein, 1982). In fact, the stretch reflex excludes such a possibility in intact organisms. Nevertheless, a fairly popular α model (Bizzi, 1980; Cooke, 1980; Crossman & Goodeve, 1983; Schmidt, 1982) is based on the tacit assumption that α or k is an independent variable. As a consequence, the model may lead to erroneous conclusions concerning the properties of intact motor control (Berkinblit et al., in press; see also below).

3.0 Invariant Characteristics

When parameter λ remains constant, a single-valued dependence of the muscle force on the length takes place (see Equation (1)), that is, the muscle together with operating central and reflex mechanisms is constrained to act like an ordinary spring. This suggestion has been corroborated by the recording of static spring-like relations between the exerted elbow muscle torque and the joint angle in unloading experiments when subjects were instructed "not to intervene voluntarily when deflections of the elbow were elicited" (Asatryan & Feldman, 1965; Feldman, 1966; 1980). Figure 1 shows the relations termed invariant characteristics (ICs), which have also been recorded for other limb muscles (Feldman, 1979; Davis & Kelso, 1982; Vincken et al., 1983). For what follows, it is convenient to accept that ICs are transformed into relations between the muscle force exerted and muscle length.

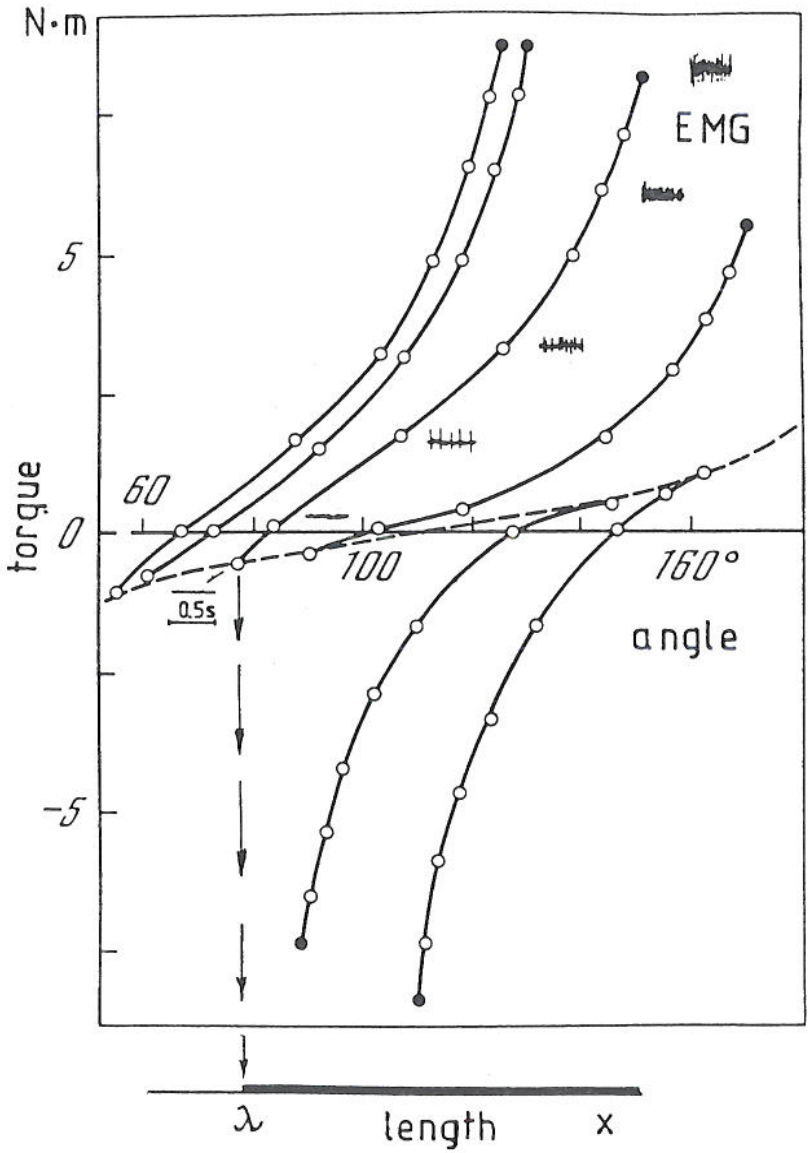
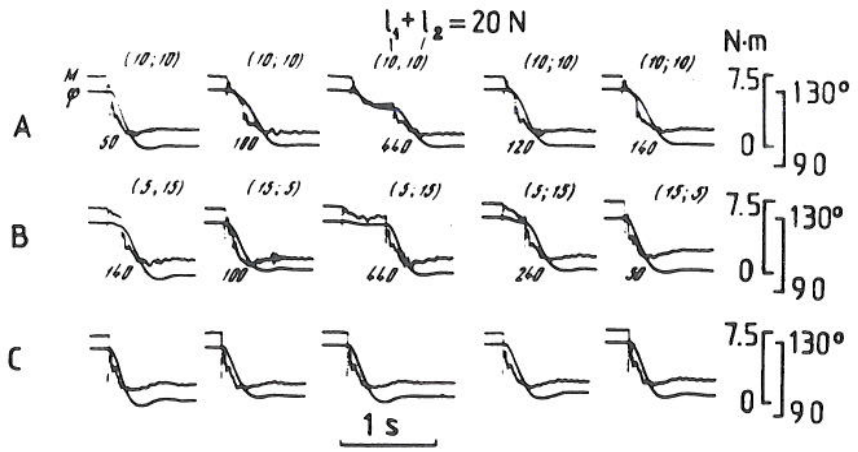


Fig. 1—A family of invariant characteristics (ICs) of elbow flexors (upper curves) and extensors (lower curves), measured experimentally by the unloading method. The instruction was "not to intervene voluntarily to forearm deflections when the load is removed"; solid circles: initial combinations of muscle torque and elbow angle; open circles: combinations of the same variables when they have settled after the unloading trials. For one IC, tonic EMG activity of biceps is shown (see also Figure 12). The activity decreases with muscle shortening, so the IC finally merges with the characteristic of passive elbow muscle (dashed line). The confluence point makes it possible to find (arrows) the parameter λ, invariant for the IC, on the scale of muscle lengths (modified from Feldman 1980).



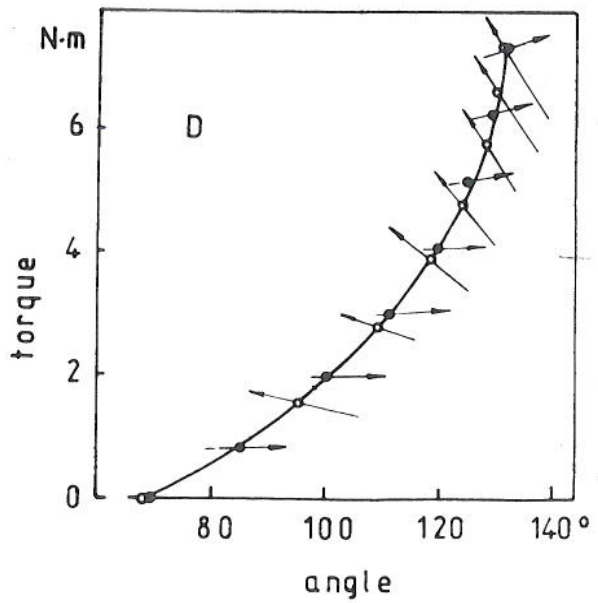


Fig. 2—Two tests of invariance of ICs. A-C: dynamic variations of unloading procedure do not affect final forearm position; M: load torque; φ: elbow angle; one part (l_2) of load 20 N to the elbow flexors was removed after another (l_1) in various combinations (in brackets) with various time delays (numbers under M and φ curves, in milliseconds). In control trials (C) the load 20 N was removed at once. The mean final position settled in A, B differs insignificantly from that in C ($P > .05$). D: another test; two flexor ICs beginning from one and the same initial (top) point were measured by unloading method with the use of loads either increasing (arrows directed to the right) or decreasing (the rest arrows) with joint angle. Note that experimental points despite the essential variations in load gradients form one IC (modified from Feldman, 1979).

Mathematically, the existence of a family of ICs for a muscle means that static muscle force is a function of one more independent variable or parameter that is constant for a given IC, but different for different ICs. The conclusion that some parameter remains invariant for every IC has been corroborated by special tests showing that space-time variations of the unloading procedure, within reasonable limits, leave the ICs unchanged (Figure 2). The variable or parameter that is constant for all the points on the IC should not be identified with the level of muscle activation alone, since EMG changes are evident in association with various IC points (Figure 1). On the other hand, ICs depart from various points of the torque/angle curve for passive elbow muscles (Figure 1, dashed line). This means that tonic recruitment of motor units for every IC begins from a specific threshold length (λ). Being constant for a given IC, the λ varies from IC to IC and consequently plays the role of the additional independent variable affecting muscle force. Thus, one may accept that λ is identical with the variable λ in Equation (1).

Experimentally, the transfer from one IC to another was evident when the subject voluntarily changed the position of the elbow. Thus, it was concluded that the independent central change in the parameter λ underlies voluntary control over arm position. Since neighboring ICs travel almost in parallel, the static muscle force is, to a first approximation, an increasing function of the difference between the current muscle length and its threshold value:

$$F = F(x - \lambda) \tag{2}$$

The symmetry of the variables x and λ is a remarkable property of motor control: although the nervous system is incapable of direct controlling the muscle length, x, it can control some other variable, λ, having the dimension of length and affecting the static muscle force as effectively as the muscle length itself. The same symmetry is expressed in the condition of muscle activation: the muscle is active if its absolute length (x) exceeds the threshold length λ:

$$x - \lambda > 0 \tag{3}$$

The inequality defines the *static muscle activation area* and can be read in two ways: not only can muscle stretching to a length $x > \lambda$ elicit muscle activation but so can a central bias of the parameter λ to a value $\lambda < x$. Consequently, inequality (3) takes into account both reflex and central means of activating the muscle (Figure 3).

The change of tonic EMG in association with points of IC shows that the level of muscle activity (α) in the suprathreshold area (3) is an increasing function of $s = x - \lambda$:

$$\alpha = \alpha(s) = \alpha(x - \lambda) \tag{4}$$

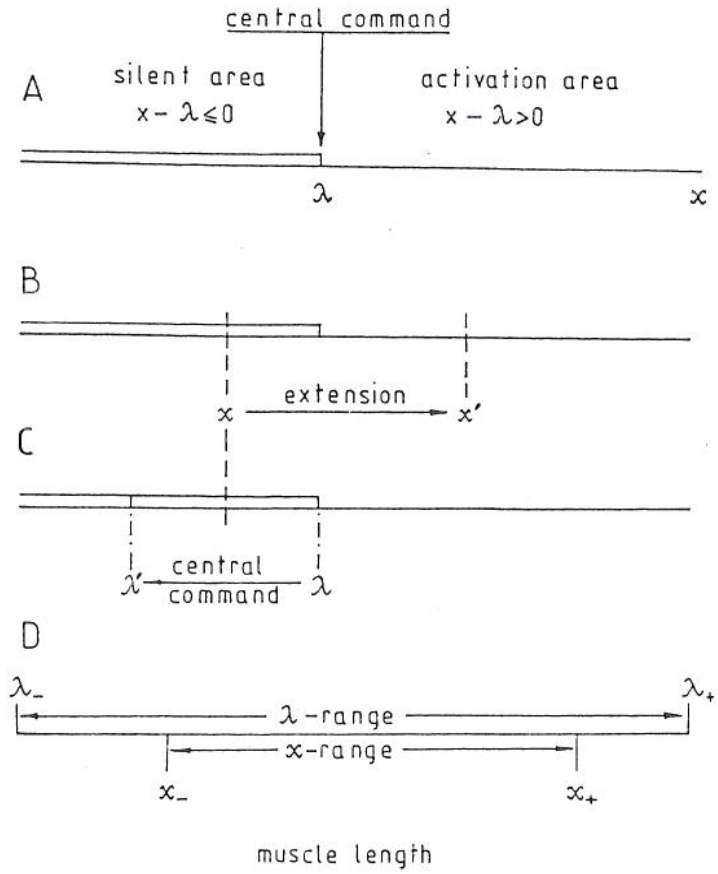


Fig. 3—Muscle activation area $x - \lambda > 0$ (statics). A. Central commands descending to α and γ motoneurons specify a lower border (λ) of the range of muscle lengths x, in which the muscle undergoes activation. B, C. The condition $x - \lambda > 0$ takes any ways of muscle activating into account. B. Reflex activation by muscle extension ($x \rightarrow x'$). C. Central command ($\lambda \rightarrow \lambda'$) shifts the border to the left and as a result the initial muscle length (x) turns out in the activation area. D. Natural range of parameter λ attainable in situ (λ_-, λ_+) is suggested to exceed that of muscle length (x_-, x_+).

The IC has a dual (central-reflex) nature. Reflex properties of IC become apparent from the dependence of muscle activity on length. In this respect ICs are similar to tension-extension curves for muscles in the decerebrate cat (Feldman & Orlovsky, 1972; Hoffer, & Andreassen, 1981; Houk, 1976; Matthews, 1959). On the other hand, the range of muscle lengths in which the IC operates is essentially dependent on central commands or their overall measure λ as relations (2) – (4) show. In fact, the IC should be associated with the central fixation of two independent variables one of which specifies its position and the other its form (e.g., Nichols, 1983). The corresponding modification of the λ model is fairly obvious and is not considered here.

4.0 The λ Model

4.1 *Neurophysiological interpretations*

Neurophysiologists often concentrate on the neuronal interactions that form the basis of motor control. The λ model, in the main, deals with common, biophysical principles underlying the motor control. However, the two approaches have begun to be integrated in the λ model due to the association of the IC with the tonic stretch reflex and the λ with its threshold. It seems to be useful to develop the model in this direction. On the other hand, at present, it seems impossible to do so unambiguously. An additional simplification of possible neurophysiological interpretations of the model might look as follows (Figure 4). There are three levels of the regulation of muscle activity in the proposed model. *The lowest level* includes loads or forces counteracting the muscle. *The middle level* or the level of the stretch reflex is a servosystem of muscle control and includes the muscle, its receptors, afferents, homonymous α and γ motoneurons, and interneurons mediating proprioceptive influences upon the motoneurons. *The highest level* includes neuronal (e.g., descending) systems exerting central, independent influences upon the α and γ motoneurons. The term "independent" here is relative: each of the descending systems does not always function in an open-loop fashion, that is, they may be influenced by afferent signals from the muscle. If so, the middle level is the appropriate system description. The λ model indicates an integral measure (λ) of the central commands of the highest level. This measure indicates simultaneously a concrete IC from the family of ICs. The middle

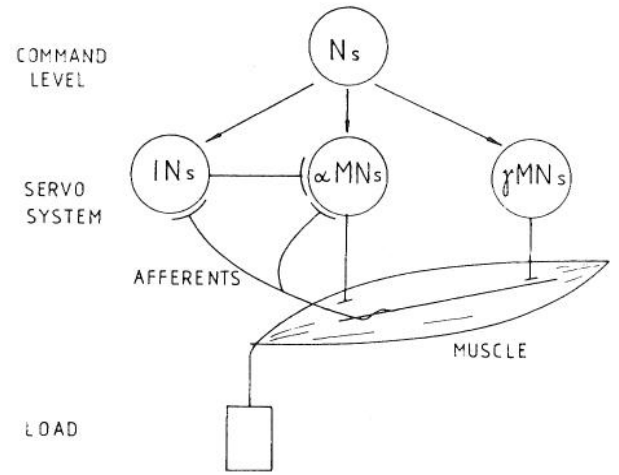


Fig. 4—A simplified diagram to show three levels of motor control. Each circle is a population of neurons (Ns) interneurons (INs) and motoneurons (αMNs, γMNs). Each level brings about a certain function in the λ model (see text).

level generates muscle force, the static component of which is determined by the IC. The lowest level (load) interacts with the middle level, and they—in combination—establish the system's final, equilibrium state.

4.2 *Loads and tonic electromyograms*

Central commands only specify the border (λ) of muscle activation area (Figure 3A). Neither the level of muscle activation, nor the force or length are directly specified by the commands. Only the interaction between the muscle and the load finally specifies these variables. Consequently, load properties have an essential influence on motor behavior. Among the ones worth considering are: static characteristics (load magnitude versus muscle length), the slope or force gradient and the so-called EMG-gradient. The static characteristic of isotonic, constant load is a horizontal line on the coordinate plane of force against muscle length; that of isometric load is a vertical line on the same plane (Figure 5A). There are loads with positive, negative and variable force gradients (Figure 5B).

The above classification of loads can be combined with a consideration of EMG levels needed to balance the load at various muscle lengths. Let α be the level of EMG for a point of IC (Figure 5C). Another IC has a point associated with the same EMG level and points with higher or lower EMG levels. The line $\alpha\alpha$ passing through points associated with an identical EMG level for a set of ICs is a characteristic of isoelectric load, that is, a load, any equilibrium position of which corresponds to one and the same tonic muscle activation. Experimentally, the measured isoelectric load characteristic can be realized, for example, with the help of a torque motor. In general, loads have negative (Figure 5C, line $\alpha\alpha'$) or positive (line $\alpha\alpha''$) EMG-gradients and their equilibration is associated with a decrease or increase in tonic muscle activity as a function of muscle length. The analysis shows that although muscle activity

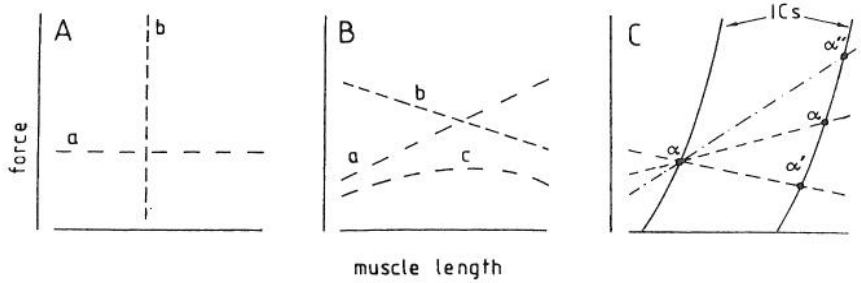


Fig. 5—Static load characteristics (dashed). A: isotonic (a) and isometric (b) loads. B: loads with positive (a), negative (b) and variable (c) force gradients. C: loads with various EMG gradients. Isoelectric load characteristic ($\alpha\alpha$) intersects muscular invariant characteristics (ICs) in points associated with one and the same level (α) of muscle activation, that is, EMG levels needed to balance the load in various positions are identical; $\alpha\alpha'$,$\alpha\alpha''$: loads with negative and positive EMG gradients, respectively.

strictly depends on the muscle length (Equation (4)), the dependence can be masked when the second variable (λ) is modified as well. In such cases, the load characteristic plays a considerable role in determining the correlation between the muscle activation level and the length: selecting loads with various gradients may cause positive, negative and even zero correlations between the two variables. Strictly speaking, such observations do not justify popular hypotheses that the participation of the stretch reflex in motor control is non-systematic.

The implications of various loading influences indicated above are crucial for various theories of motor control. For example, the isotonic load shows that muscle force cannot be a specific determinant of final muscle length. Thus, no correlation between the two variables can be observed. Bizzi's α model (1980) suggests that central specification of the level of muscle activity underlies motor control (Figure 6A). However, the model gives no explanation of how a final muscle length is specified in the case of an isoelectric load, when the system is constrained to demonstrate a zero correlation between EMG level and muscle length. The equilibrium point concept, as advocated in terms of the α model, cannot help resolve the problem. The fact is that the muscle length-force characteristics in the model are themselves isoelectric (α is invariant for each of them) and consequently neither of them can intersect an isoelectric load characteristic to specify a concrete equilibrium point (Figure 6B).

Moreover, the α model does not cope with even a small load having a positive EMG-gradient. Figure 6C illustrates the notion. Two α-invariant muscle characteristics (α_1 and α_2; $\alpha_2 > \alpha_1$) intersect a load characteristic

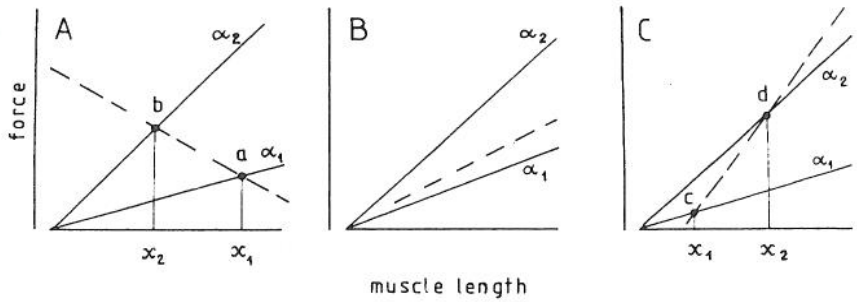


Fig. 6—Bizzi's model (A) and its consequences: the muscle is unable to balance isoelectric loads (B) or those having positive EMG gradients (C). A. The nervous system specifies a level (α_1) of muscle activation and as a result muscle stiffness associated with the slope of the static muscle force/length function (solid line α_1); dashed line: static load characteristic; a: equilibrium point; x_1: equilibrium position. A new level of muscle activation (α_2) sets a new equilibrium point (b) and thus elicits a movement to still another final position x_2 (see Bizzi, 1980). B. Any α – invariant muscle characteristic can at best coincide with an isoelectric load characteristic (dashed line) but cannot intersect it to specify a concrete equilibrium point. C. The muscle counteracts a load having a positive EMG gradient (dashed line). Equilibrium points (c,d) and positions (x_1,x_2) are non-functional since the system is instable there (see text).

at two points associated with equilibrium positions x_1 and x_2. The load has a positive EMG-gradient, that is, the greater the muscle length the greater the muscle activation necessary to balance the load. Let the system's initial conditions correspond to position x_1 and the task require a change to x_2. Bizzi's model suggests that to do so, the muscle activation has to be changed to α_2. However, an increase in muscle activation in position x_1 elicits muscle shortening but not the necessary muscle extension. Thus, the α model provides for *non-functional* EPs when the muscle deals with positive EMG-gradient loads. In contrast, this flaw is absent in the λ model (see below).

4.3 *A reformulation of the "size principle"*

The reader should notice that the λ model expresses the idea of monoparametric control of tonic activity in the motoneuron pool and thus is similar (but not identical) to the idea of the orderly recruitment of motoneurons according to their size (Henneman & Mendell, 1981). One can correspondingly hypothesize that the tonic recruitment of motoneurons occurs in the order of their threshold lengths: $\lambda^{(1)}, \lambda^{(2)}, \ldots, \lambda^{(i)}, \ldots, \lambda^{(n)}$ (Figure 7). The threshold $\lambda^{(1)}$ of the most sensitive motoneuron coincides with that of the tonic stretch reflex ($\lambda^{(1)} = \lambda$). By definition, the motoneuron of a rank i is tonically active if $x - \lambda^{(i)} > 0$. Those

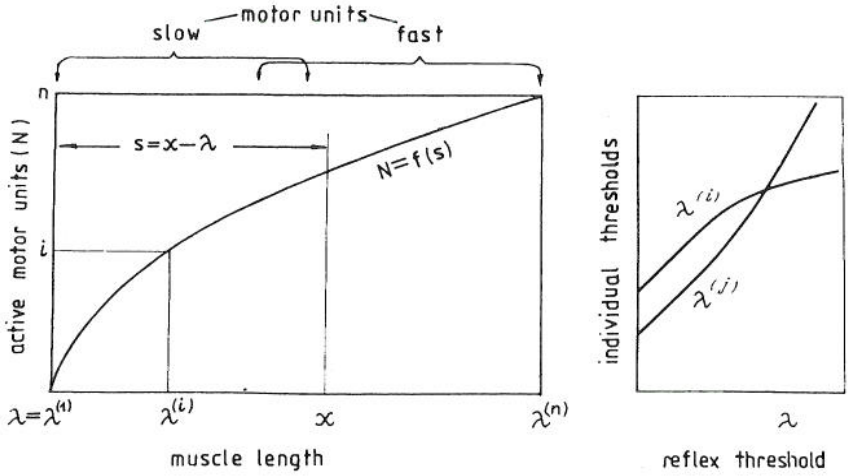


Fig. 7—A reformulation of the "size principle" in terms of the λ model (statics; for dynamics see Figure 13). Recruitment of motor units of a muscle is suggested to occur in the order of their reflex thresholds ($\lambda^{(1)}, \ldots, \lambda^{(i)}, \ldots, \lambda^{(n)}$; $\lambda^{(1)}$ coincides with threshold length λ of the tonic stretch reflex). Fast motor units are in the main involved after slow ones. The number N of motor units recruited at a suprathreshold muscle length x depends on its distance s to the threshold λ. The thresholds of motor units are unambiguously interrelated and thus any central command (λ) affects simultaneously thresholds of all motor units ("monoparametric control"). Right diagram shows that the λ-control may bring to an inversion of the recruitment order for motor units if their thresholds (e.g., $\lambda^{(i)}$, $\lambda^{(j)}$) depend on the λ non-linearly, as is illustrated.

motoneurones are recruited at a muscle length, $x > \lambda$ and have thresholds between λ and x such that their number N is an increasing function of one variable $s = x - \lambda$ if $s > 0$:

$$N = f(s) \tag{5}$$

In addition, the monoparametric hypothesis claims that all the thresholds are unambiguously interrelated so that for any i

$$\lambda^{(i)} = f_i(\lambda) \tag{6}$$

It is of interest that the monoparametric hypothesis admits, to a certain degree (as does the size principle), reversals of the recruitment order with changes in the λ. An increase in the threshold of one motor unit may advance that of another since a priori the functions f_i can be of various gradients (Figure 7B). The monoparametric hypothesis will be generalized to dynamics in a later section on Muscle activation area in an effort to explain the various types of EMG pattern.

4.4 *Physiological range of* λ

There are normal operating ranges of the muscle length attainable in situ, say, from x_- to x_+ and of the static muscle force, from $F_- = 0$ to F_+. The ranges are provided with the respective control over parameter λ. How is the λ-range (λ_-, λ_+) correlated with the x-range? Based on simple arguments one may show that the λ-range exceeds the x-range, or more precisely,

$$\lambda_- < x_-;\ \lambda_+ > x_+ \tag{7}$$

(Figure 3C). Actually, the λ-control has to provide muscle relaxation in the total x-range. To do so, the IC must be shifted to the right, beyond the maximum physiological length (x_+). Hence, it follows that $\lambda_+ > x_+$. On the other hand, the nervous system must provide muscle activation at any physiological length. In particular, to activate the muscle at the minimum physiological length (x_-) the IC must be shifted to the left, beyond the x_-. Hence $\lambda_- < x_-$.

This consideration can be used to classify pathologies of central regulation of muscle tonus. For example, if the first inequality in (7) is broken, no muscle force can be exerted in a range of low lengths ("hypotonia"). If the second inequality is broken, it is impossible to relax the muscle at high lengths ("spasticity").

In its present form Bizzi's model (Figure 6A) does not provide regulation of muscle length and force in the muscle's physiological range. All the muscle characteristics in that model start from a point at which the muscle is relaxed and its length (x) coincides with the parameter l called rest length (Figure 6A). Thus, the l is suggested to be constant whatever the muscle activation. It is important to indicate clearly whether the rest

length is situated inside or outside the normal operating range of muscle lengths. Passive limb muscles in vertebrate (e.g., human forearm flexors; Ralston, Inman, Strait, & Shaffrath, 1947) usually sit inside the operating range and, consequently $x_- < l < x_+$. Since the l is constant in Bizzi's model, the muscle is unable to exert a nonzero force for lengths between x_- and l (as muscle characteristics do not operate in this part of range). The "dead zone" of motor control in this model is reminiscent of motor pathologies (see above). However, even in the remaining part of the physiological range, the Bizzi model is inadequate. For example, a load close to zero cannot be lifted at all despite intensive variations of muscle stiffness (for comparison, stiffness control in a spring is ineffective when the load is small). In contrast, only a heavy load can present difficulties in natural conditions. The chief flaw of the model is a consequence of postulating that l is constant irrespective of muscle activation. In this respect, the model is not consistent with the classical sliding-filament theory (Huxley, 1974) nor with data showing that when sarcomeres or whole muscles are tonically stimulated without a load they contract to a new steady state length (Rack & Westbury, 1969; Partridge & Benton, 1983).

4.5 *The functional characteristic of the α motoneuron*

Whether a single α motoneuron is active or not depends on its synaptic inputs and its intrinsic properties. The inputs can be interpreted as information about current muscular state and central commands. In this connection, there must exist a relation between basic electrical parameters of the motoneuron (the membrane potential and its threshold) and such variables as x and λ. This section is an attempt to establish such a relation which will be termed the functional motoneuron characteristic.

Consider a single motoneuron with the lowest threshold λ coinciding with the stretch reflex threshold. Let V be a membrane potential of the motoneuron when the muscle is relaxed at a length $x < \lambda$. The potential increases with x owing to the stretch reflex (Figure 8A). The threshold potential V_+ is reached at a muscle length $x = \lambda$, and the motoneuron begins its tonic firing. For simplicity, let us suppose that V increases linearly with x in the subthreshold range (Figure 8A, dashed line), then the functional characteristic of the motoneuron is given by

$$V = V_+ + a(x - \lambda) \tag{8}$$

where the positive parameter, a corresponds to an "electromechanic" motoneuron gradient. Similar relations can be written for any α motoneuron with respective particular values of variables and parameters. It should be noted that

$$\Delta V = -a\Delta\lambda \tag{9}$$

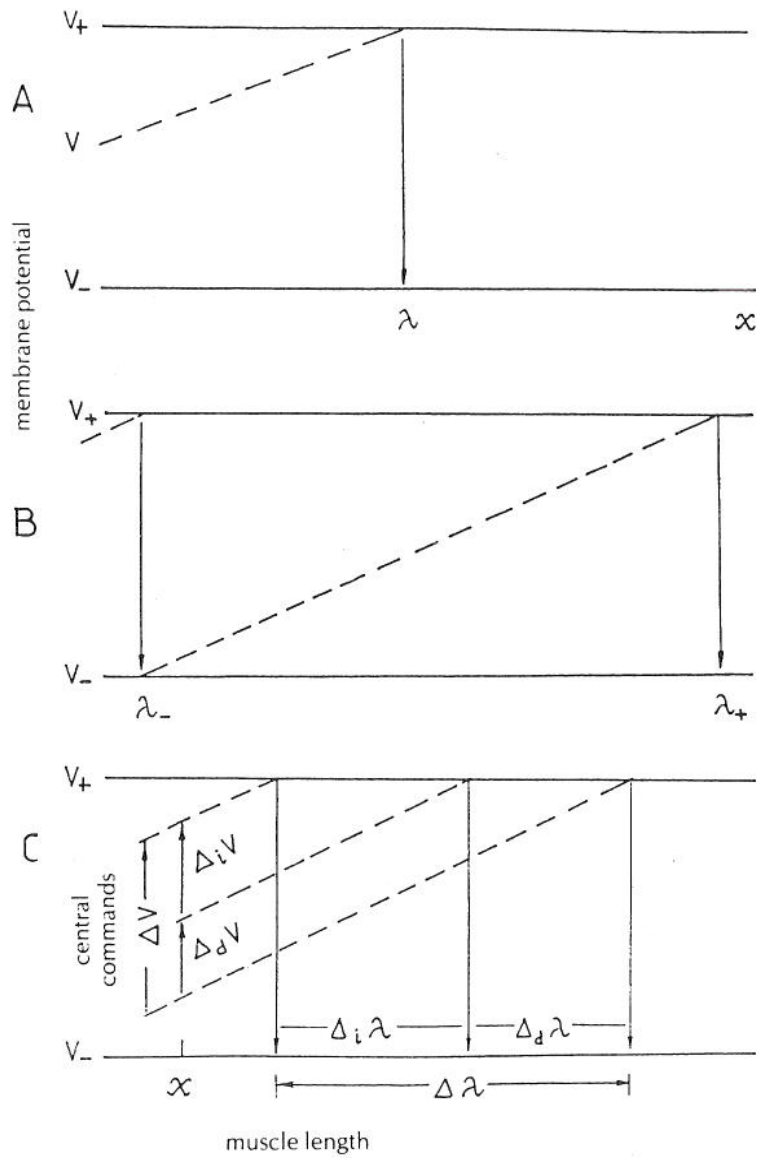


Fig. 8—Supposed reflex characteristics of α motoneuron (dashed lines) and their descending control by direct and indirect (mediated by γ motoneurons or interneurons of reflex loops) inputs. V_+,V_-: threshold and minimal membrane potentials of a motoneuron. A: current potential (V) increases with muscle length x; the threshold V_+ of motoneuron firing is achieved at a muscle length λ that for the most sensitive motoneuron coincides with the threshold of the tonic stretch reflex. B: two extremal motoneuron characteristics to show that normal operational range (λ_-,λ_+) of λ attainable in situ corresponds to that of membrane potential (V_-,V_+). C: additivity of direct (d) and indirect (i) central commands in terms of λ ($\Delta\lambda = \Delta_i\lambda + \Delta_d\lambda$) is the result of a linear summation of potentials Δ_iV, Δ_dV elicited by the commands.

where ΔV is an increment of membrane potential conditioned by independent central commands on motoneurons, and $\Delta\lambda$ is an increment of the tonic stretch reflex threshold. Consequently, the two measures of central motor commands are interrelated with each other.

Equation (9) allows us to find factor a. Actually, maximal change of membrane potential from its minimal level (V_-) to threshold level is $V_+ - V_-$. In terms of λ, the corresponding central command is $\lambda_- - \lambda_+$ where λ_- and λ_+ are physiological limits for λ (Figure 8). From (9) it follows

$$a = -\frac{\Delta V}{\Delta\lambda} = \frac{V_+ - V_-}{\lambda_+ - \lambda_-} \qquad (10)$$

4.6 $\alpha - \gamma$ – *linkage*

To illustrate the use of the motoneuron characteristic, let us compare the effects of direct central influences upon α motoneurons with the effects of indirect influences upon them elicited by central activation of γ motoneurons. If an initial potential V is altered as a result of direct (d) central influences upon the α motoneuron, its threshold potential V_+ is reached in the course of muscle extension at a smaller length than the λ by a value $\Delta_d\lambda$ as is shown in Figure 8C. On the other hand, the initial potential may be shifted by indirect (*i*) influences upon the motoneuron, and thus the λ is shifted by a value $\Delta_i\lambda$. When the two inputs act in combination, the overall shift $\Delta\lambda$ is given by

$$\Delta\lambda = \Delta_d\lambda + \Delta_i\lambda \qquad (11)$$

provided that the factor a in equation (8) is constant. This relation shows that tonic central inflows to α and γ motoneurons result in additive shifts in the λ and thus are mutually replaceable. In addition, the two inputs in combination bring about the regulation of threshold λ (and, consequently, of muscle force and length) in a broader range than each central input in isolation. This is a very essential function of central inputs to α and γ motoneurons given the necessity to provide λ – control in the overall physiological range (see above).

The suggestion that both direct and indirect central commands on α-motoneurons are reducible to shifts in the reflex threshold is consistent with data that (a) show selective influences upon γ motoneurons, and (b) that tonic stimulation of different descending systems in the decerebrate cat with complex action on α and γ motoneurons affects only the stretch reflex threshold (Feldman & Orlovsky, 1972; Matthews, 1959).

4.7 *Equilibrium points*

In contrast to parameter λ characterizing the state of command neuronal levels, and to the IC conditioned by reflex and command levels, the EP is an integral property of the interaction between all the levels including load. The EP concept is something more than the

mechanical condition of balance between the muscle and the load. This is a deep dynamic concept in the sense that the EP essentially (though by no means completely) determines not only the static but also dynamic behavior of the system.

Nominally, the EP in the λ model is a point of intersection between the static load characteristic and the IC determined by a current value of parameter λ (Figure 9). The point can be found for various loads (isotonic, isometric, isoelectric or having other force or EMG-gradients). It has two coordinates and thus indicates both the system's equilibrium position and the muscle force necessary to balance the load. The isometric load predetermines an equilibrium position whereas the isotonic load acts as an equilibrium muscle force. In general, a relation between the two equilibrium variables is predetermined by the static load characteristic.

The definition of EP proceeds from the fact that the parameter λ is an independent measure of central commands descending to α and γ motoneurons. Consequently, not muscle activation *per se*, but λ is the important determinant of the system's equilibrium state. In contrast, the EP in Bizzi's model is defined in terms of muscle α activity which specifies stiffness (Figure 6A). At first sight, the two definitions of the EP are identical: the EP in the λ model could also be associated with a certain level of α activity necessary to achieve equilibrium, as in the α model. However, the following example illustrates a distinction between the two definitions.

Let a load be pushed by a short external tap from an equilibrium position to a subthreshold position $x < \lambda$. In accordance with the λ model, the initial muscle activity will temporarily disappear (see (3)) but the system's EP, by definition, remains the same provided that λ remains constant. In contrast, the α model could associate the EMG modifications with meaningful shifts in the EP.

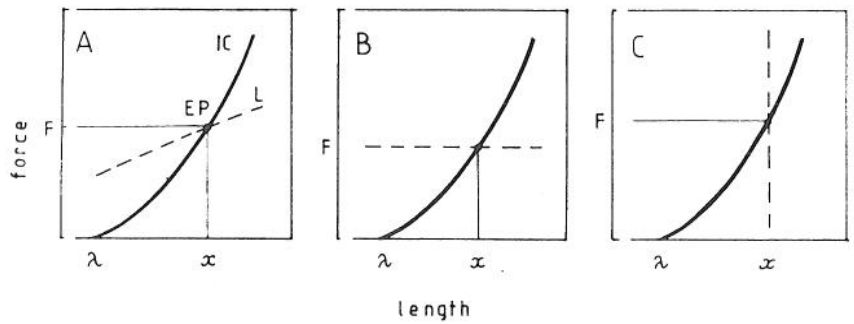


Fig. 9—Equilibrium point (EP in the λ model as a point of intersection of muscle invariant characteristic (IC) and static load characteristic (L). Three types of loads are shown: with a positive force gradient (A), an isotonic (B) and an isometric (C); x, F: equilibrium position and force; λ: stretch reflex threshold.

One could say that an *a priori* selection between the two definitions of EP is a matter of taste. However, the fact is that the EP concept must be defined in accordance with the notion of equilibrium position used in dynamical systems theory (e.g., Andronov & Chaikin, 1949). Using this theoretical framework as a guide, let us define the EP concept for a muscle-load system in terms of muscle activation. Let our system be described by a Newtonian differential equation for muscle length x, its time derivatives $\dot{x}$ and $\ddot{x}$ and α:

$$m\ddot{x} = L(x,\dot{x}) - F(x,\dot{x},\alpha) \tag{12}$$

where m is mass, F is muscle force and L is load. Parameter α is also a function of x and its derivative: $\alpha = \alpha(x,\dot{x})$. Generally speaking, the L can also be a function of kinematic variables. In an equilibrium state, all interactions among the system's components are balanced and the system can remain in that state for an indefinitely long time in the absence of perturbations. More precisely, to find an equilibrium position x_0 in a moment t_0 one must nullify all the derivatives in the differential equations for x and α. The task is then reduced to the resolution of two algebraic equations, one for x_0:

$$L(x_0) - F(x_0,\alpha) = 0 \tag{13}$$

and another for α, that is, taking into account (4):

$$\alpha = \alpha(x_0 - \lambda_0) \tag{14}$$

where $\lambda_0 = \lambda(t_0)$.

Equation (13) relates equilibrium muscle force and length at a given static level α of muscle activation and consequently describes an isoelectric muscle force-length characteristic. Thus, although one can in principle define an equilibrium position in terms of muscle activation, other factors must be taken into account. Among these are the dependence of activation on muscle length, the fact that only the static component (i.e., not all) of α activity has to be considered at every moment in order to judge veridically the time course of the equilibrium point.

Since the form of isoelectric muscle characteristics postulated in Bizzi's model is not consistent with the theory of muscle contraction (see section 4.4 on the "Physiological range of λ") and, in addition, that α is considered an independent variable (see also Hogan, 1984), means that both the relation (13) and (14) are misrepresented in Bizzi's definition of EP. Consequently, his definition is unacceptable for intact motor control.

Even a correct definition of the EP concept in terms of muscle activation has the drawback that it implies that EMG changes give rise to EP shifts. In reality, the causal chain of events is exactly the inverse: *shifts in EP give rise to EMG modifications in intact organisms*. This is an essential aspect of the EP concept (see below) crucial to its understanding.

It is important to notice that the complex variable, α, can be excluded from (13) by substitution of (14) into (13). The second term in (13) describes then an IC specified by threshold λ_0, and equation (13) as a whole means that equilibrium position x_0 is a point of intersection between the IC and a load static characteristic as initially defined in the λ model (Figure 9).

Essentially, the equilibrium position is defined: (a) for any movement t_0, although the system can be in movement (for comparison, the equilibrium position of a pendulum is defined both for statics and dynamics); (b) in dynamics, the current and equilibrium states of the system, generally speaking, do not coincide ($x \neq x_0$); (c) by the static characteristics the system possesses at that moment; (d) by the current measure λ_0 of central commands. Other aspects of the EP concept will be considered later on in this paper.

4.8 *Postural stability*

The EP concept is associated with the idea that any deflection from an equilibrium state gives rise to an active dynamic process—both at the neuronal and at the mechanical level of muscle-load interaction—returning the system to that state if certain postural stability conditions are met. Such conditions can be elucidated from dynamic equations of movement linearized in a range of deflections from the system's equilibrium position. Such equations have been proposed and the analysis is available in Russian (Feldman, 1979). However, some essential conditions of postural stability may be formulated in English from rather simple, qualitative considerations. First of all, if the system's state differs from the equilibrium state, there is a difference between the muscle force and the load. The difference causes an acceleration of the system if there is no isometric fixation. The acceleration is directed to the equilibrium position if muscle force increases (decreases) more effectively during muscle extension (shortening) than the load magnitude, that is, if

$$k > k' \tag{15}$$

where k is slope or stiffness of IC and k' is load gradient in the equilibrium point (Figure 10).

Inequality (15) is a necessary condition for stable equilibrium: it provides the force pushing the system to the equilibrium position that is reached if additional criteria are fulfilled. In particular, the system must be damped sufficiently, which means that muscle force is rapidly reduced with the velocity of muscle shortening—a characteristic property of active muscle (Hill, 1938) and of the stretch reflex as a whole (see below). If the above criterion is broken, the system will oscillate about its equilibrium position. In what follows it is accepted that such is not the case (i.e., additional criteria have been met), so inequality (15) is the only condition of stable equilibrium.[3]

In contrast to Merton's hypothesis (Merton, 1953), the stretch reflex has no mechanism to restore one and the same muscle length in response to different loads, that is, their positions vary in accordance with the IC (see also Houk, 1976). A meaningful gain or stiffness of the stretch reflex is correspondingly unnecessary for postural stability in the λ model. For example, inequality (15) shows that muscle stiffness k must be slightly positive to stabilize an isotonic load ($k' = 0$) and the IC meets the requirement with a meaningful reserve. The monotonous increase of muscle activation with muscle length for any IC guarantees that it intersects any isoelectric load characteristic at a superior gradient. Thus, not only is a certain equilibrium point specified in this case, but also the condition of stability (15) is fulfilled. Moreover, to a certain degree, the surplus IC gradient makes it possible to stabilize loads with positive EMG-gradients.[4] In contrast, in the α model only loads with negative EMG-gradients can be stabilized (otherwise condition (15) is broken since muscle characteristics in the α model themselves have zero EMG-gradients). Thus, muscles in the λ model have broader capabilities as far as balancing loads is concerned than in the α model. If one posits that the α model is applied only to deafferented muscles, one may conclude that intact motor control is more effective than after muscle deafferentation.

In isometric conditions, the system aspires to reach an equilibrium value of muscle force. To explain this tendency, the interaction between the active contractile and passive series elastic components of the muscle must be considered. In short, one of them plays the role of a load for the other. The equilibrium force is achieved at a certain equilibrium length of the contractile component. In a non-equilibrium state,

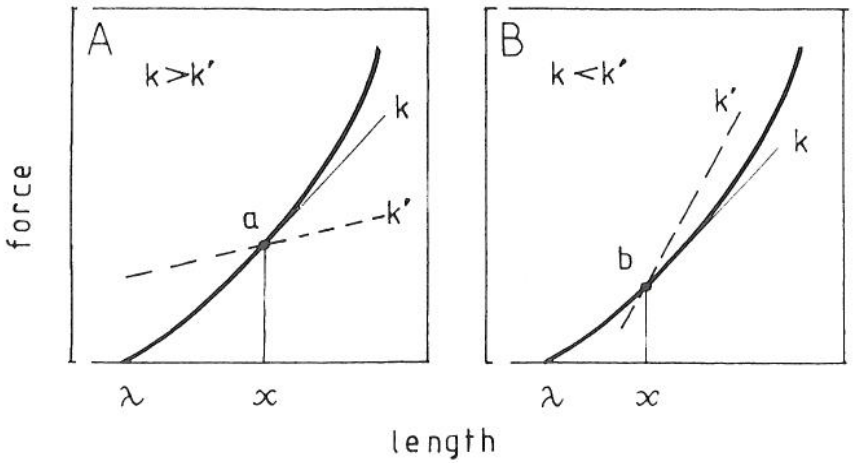


Fig. 10—A necessary condition of postural stability: $k > k'$. A. In equilibrium point a and position x, stiffness k of invariant characteristic specified by reflex threshold λ exceeds that k' of static load characteristic (dashed line); thus, the condition $k > k'$ is observed. B. Postural fixation is impossible ($k < k'$) despite the existence of equilibrium point (b).

one component contracts at the expense of the other's extension. When the tension of the contractile component increases with its length (i.e., $k > 0$), that of the elastic component decreases with the length of the *contractile* component (i.e., $k' < 0$). Thus, the necessary condition (15) of stable equilibrium is observed in isometric conditions as well.

Usually, two components of muscle stiffness are distinguished in physiological studies. One component is associated with an initial tonic activation and muscle elasticity, and the other is associated with an additional enhancement of activity through muscle extension. Only the latter component is usually believed to have a reflex origin. This component often proves to be small in certain motor tasks, and the conclusion follows that the stretch reflex plays an insignificant or non-systematic role in motor control. This conclusion is grounded in the suggestion that the first component of muscle stiffness is conditioned by an independent central activation of motoneurons. However, one can just as easily admit that the initial tonic excitation and the corresponding stiffness component are an integral result of preliminary muscle extension from λ to x. From this point of view, the first component is not less reflexive than the second one. It is better to recognize the two components as integral and differential stiffnesses, with the understanding that both are equally conditioned by central and reflex mechanisms (cf. Equation (4)).

4.9 *Active movements*

Both voluntary and involuntary movements belong to a class of active movements and both are elicited by shifts in the system's EP. Figure 11A shows an involuntary step-like shift of equilibrium point ($a \rightarrow b$) evoked by a sudden unloading of the muscle as in a trial performed during the recording of ICs. As a consequence, the initial position of the system looks like a defection (Δx) from the new equilibrium position—the excess muscle force provides an initial impulse for the movement. However, the muscle shortening gives rise to a decrease in muscle activation and force (see Equations (2) and (4)), although the system's EP does not change any more provided that the λ, an independent measure of central commands, is constant. The velocity-dependent properties of active muscle (Hill 1938) as well as of the stretch reflex (see following section) intensify the drop of muscle force. Finally, the residual part of the load begins to exceed the muscle force and decelerates the movement to a new EP.

The nervous system can intentionally perform shifts in the EP. To do so, command neuronal levels (Figure 4) alter central influences upon the α and/or γ motoneurons to bias the threshold λ (Figure 11 B,C). Initially, the system is in a non-equilibrium state, and, again, a dynamic transfer to a new EP occurs.

A shift in λ under isotonic conditions, elicits a movement to a new equilibrium muscle length, whereas a similar shift in isometric conditions results in a new equilibrium force (Figure 11C). In other cases both variables are modified due to the λ bias (Figure 11B). The α innervation

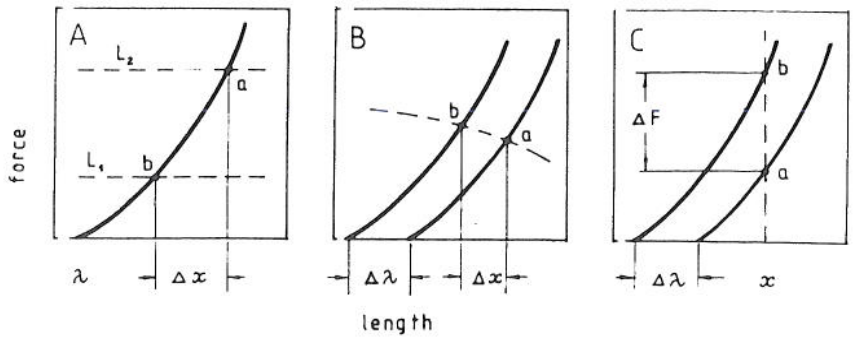


Fig. 11—Shifts in the system's equilibrium point ($a \rightarrow b$), position (Δx) and muscle force (ΔF) elicited either by changes in load L (in A) or central command $\Delta\lambda$ (B, C) that specifies a new position of muscle invariant characteristic (solid curve); dashed lines: static load characteristics. Note that a single mechanism (shift in the threshold λ) provides isometric force generations (C) and postural modifications (B).

is specified in accordance with the EMG-scale determined by the IC and dynamic properties of the servo-control level. On the whole, the λ model suggests that both muscle length and force are controlled by a single mechanism—by shifts in the stretch reflex threshold. In addition, the model is associated with the idea (not new perhaps but vaguely formulated in the past) that posture and movement are also controlled by a single mechanism—a shift in the equilibrium (postural) state of the system gives rise to movement.

4.10 *Muscle activation area*

The EP concept is associated with a dynamic transfer process that occurs every time the system is found in a non-equilibrium state. It would be useful to represent the dynamic processes elicited by EP shifts in more detail. In particular, it is necessary to explain how the shifts give rise to recruitment and derecruitment of motor units and how the movement trajectory is formed. In this connection, one needs a concept that describes the dependence of muscle activation on "peripheral," kinematic variables (muscle length and its derivatives) as well as on variables (λ and other possible parameters) characterizing independent, central commands descending to alpha and gamma motoneurons. In fact, such a concept has been introduced (Feldman, 1974 a, 1974 b, 1979)—the concept of muscle activation area (AA).

The AA is a generalization of static condition (3) for muscle activation ($x - \lambda > 0$) to dynamics. In addition, the AA is a further reformulation of the size principle discussed above.

Let λ^* be the dynamic threshold length for the stretch reflex, that is, by definition, the muscle is active if its length x exceeds λ^*:

$$x - \lambda^* > 0 \qquad (16)$$

In dynamics, the λ^* differs from static threshold λ. In statics, however, they coincide:

$$\lambda^* = \lambda \text{ if } v = 0 \qquad (17)$$

where v is the speed of muscle lengthening. Consequently, condition (16) includes statics as a particular case.

Further specifications of condition (16) are founded on properties of two phasic reflexes—the tendon reflex and the unloading reflex (Figure 12). Let a muscle be initially relaxed at a length $x < \lambda$. The muscle can be excited phasically by a rapid extension ($v > 0$), for example, during the occurrence of the tendon reflex. This means that the threshold λ^* in dynamics can be lower than in statics:

$$\lambda^* < \lambda \text{ if } v > 0 \qquad (18)$$

On the other hand, if the muscle is tonically active at a length $x > \lambda$ a sudden muscle unloading elicits a silent period in the EMG (Figure 12). Hence

$$\lambda^* > \lambda \text{ if } v < 0 \qquad (19)$$

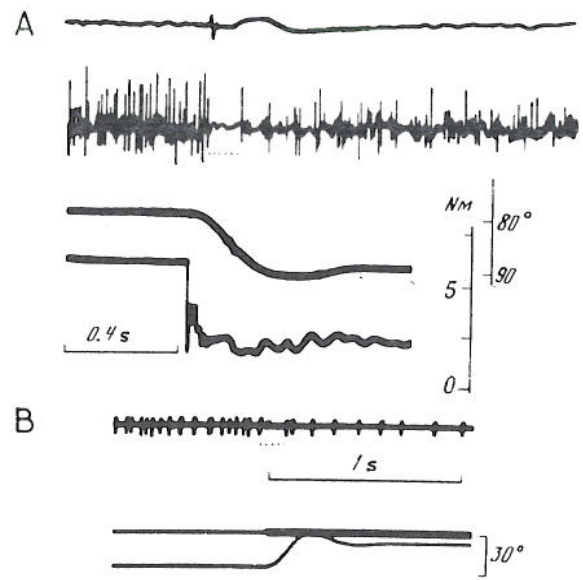


Fig. 12—Unloading trials during measurement of ICs (as in Figure 1). A, from top to bottom: EMGs of mm. biceps and triceps brachii, elbow angle, load torque. B: activity of several motor units of m. biceps brachii, unloading mark, elbow angle (flexion is up). Notice the occurrence of EMG silent periods (dotted) in dynamic phase of the reflex and diminution of tonic EMG levels in the result of unloading (modified from Feldman, 1979).

Taken together, the relations (17) – (19) show that the dynamic reflex threshold is a decreasing function of speed.[5] For purposes of simplicity, this function is assumed to be linear:

$$\lambda^* = \lambda - \mu v \tag{20}$$

where μ is a dynamic parameter having the dimension of time and playing the role of a damping factor (Feldman, 1974 a). The AA is a domain of the points (x,v) that meet the condition (16) of muscle activation. The border of AA is defined by the equation $x - \lambda^* = 0$ or, taking into account (20),

$$x + \mu v - \lambda = 0 \tag{21}$$

A simple geometric representation explains the above relations (Figure 13). The border of AA is a straight line on the phase plane of variables x and v. The λ indicates the point of intersection of the line with the x-axis, whereas the μ shows the slope of the line. The AA is situated to the right of the border line. It has been shown that tonic muscle activity increases with the difference between muscle length and its threshold length (see (4)). It is natural to use the same rule for dynamics, that is, that muscle activity increases with difference, s:

$$s = x - \lambda^* = x + \mu v - \lambda \tag{22}$$

that is equal to the horizontal distance from the current point (x,v) to the AA border. Rule (5) can be also generalized to dynamics: the number N of recruited motor units and their firing frequencies for any suprathreshold point increases with distance s. Consequently, there are isoelectric lines inside the AA each associated with a certain level of muscle α activation. They are parallel to the AA border. In accordance with the "size principle," one may accept that slow motor units are recruited at a shorter distance, s from the AA border than fast ones.[6]

Not only can the AA be represented on the phase plane but so can movement trajectories. This allows us to formulate the following rule for EMG bursts and silent periods. If the movement trajectory enters the AA, muscle activity appears and increases the more the trajectory penetrates inside the AA. When the trajectory leaves the AA, the EMG activity disappears.

The concept of AA is universal in the sense that it takes into account both central and reflex ways of muscle activating. Central commands manifest themselves in the motion of the border line due to changes in λ (Figure 13B). The horizontal distances from the current point of the movement trajectory to the border line alters thereby, and, in accordance with the above rule EMG varies correspondingly.

It is important to notice that the slope of AA depends on parameter μ. As a speed factor, it can affect muscle activity only in dynamics (when

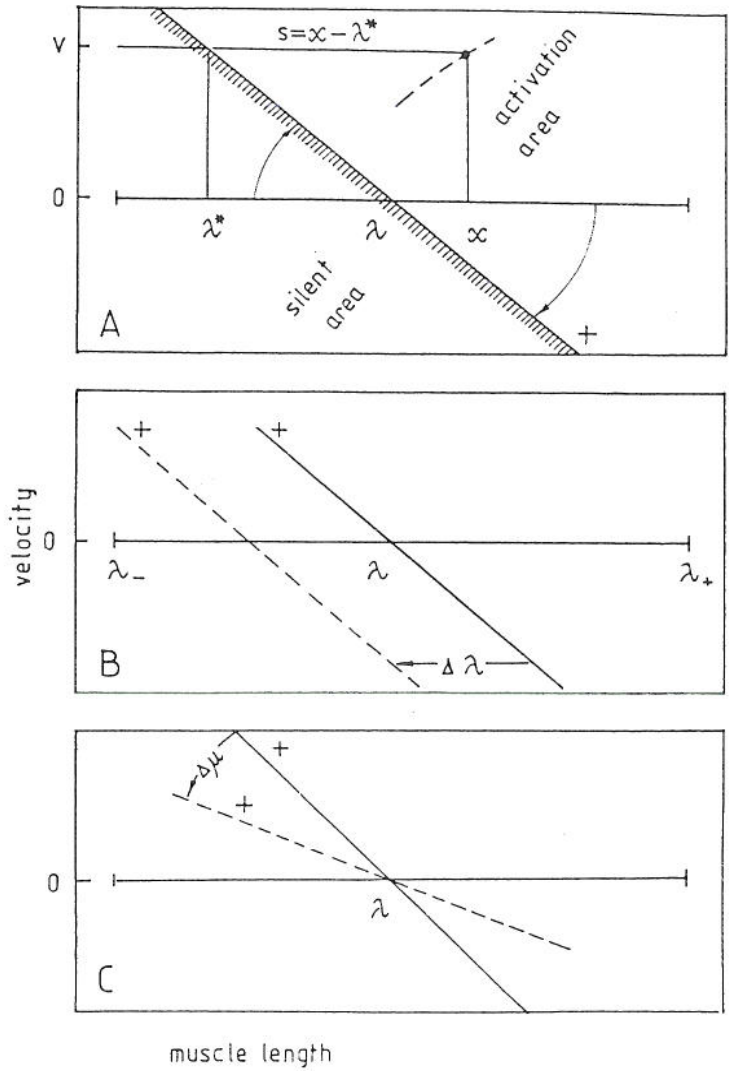


Fig. 13—Muscle activation area (dynamics)—the domain of kinematic and command variables at which the muscle undergoes activation. A. Phase plane (muscle length x versus its drivative v). Recruitment of motor units occurs to the right of the borderline (hatched) and increases as a function of excess $s = x - \lambda^*$ of muscle length over its threshold value. The s is a horizontal distance from current point (x,v) of movement trajectory (dashed) to the borderline; λ^*: dynamic, speed-dependent threshold of muscle activation ($\lambda^* = \lambda - \mu v$); central command λ specifies the position of the borderline, another command, μ, does its slope. Left arrow: a movement trajectory to show that a rapid stretching elicits muscle activation before achieving the static threshold λ (a "tendon reflex"). Right arrow: another movement trajectory; rapid shortening results in muscle deactivation at a length higher than λ (an "unloading reflex"; cf. Figure 12). B, C: geometrical interpretations of central commands (shift $\Delta\lambda$ of the border in B and its turn $\Delta\mu$ in C).

$v \neq 0$). On the whole, the μ plays the role of a damping coefficient. It is clear, that sensitivity of α motoneurons to the muscle speed is finally conditioned by dynamic sensitivity of muscle spindle afferents. Since the sensitivity is modified by dynamic γ motoneurons (Boyd, 1981; Matthews, 1964), it is natural to suggest that factor μ is a controllable variable (Feldman, 1974 a). Geometrically, the control over the μ is interpreted as a turning of the AA border (Figure 13C). Whether the μ and λ parameters can be controlled in isolation or only in combination is not known. It should also be noted that the influence of various variables and parameters on muscle activity occurs with certain time delays. For example, when a movement trajectory enters the AA, the muscle activity begins somewhat later because of a reflex delay.

A further reservation is necessary with respect to isometric conditions. Being isometric for the muscle as a whole, the conditions are not isometric for its active contractile component. To make more exact judgments of muscle activity in such conditions it is expedient to represent the AA not in the space of variables x and v but in the space of the corresponding variables for the contractile component (Figure 14C). One may, to a first approximation, neglect a slight deformation of the AA borderline and its inner structure in these variables. The force of the series elastic component is viewed as a load for the contractile component.

4.11 *Electromyographic patterns*

The geometric concept of muscle activation area is used below for a qualitative account of EMG-patterns arising in the muscle-load system during active motor performance such as rapid isotonic or isometric flexions. An extension against a load with positive EMG-gradient is also considered,—i.e., the case for which the α model does not work (see section on "Loads and tonic electromyograms") but the λ model does. In the discussion below, some additional dynamic properties of the system are worth bearing in mind such as the double dependence of muscle force on the speed v. Moreover, in dynamics, not static (λ) but dynamic threshold λ^* (dependent on v) affects the muscle force. On the other hand, the muscle fibres' tension drops with the speed of their contraction (Hill 1938). A computer simulation of various EMG patterns during one-joint movements will be presented elsewhere (Abdusamatov & Feldman, manuscript in preparation).

Voluntary changes in muscle length are performed by shifts of λ or, geometrically, by displacement of the AA border. It has been suggested that the velocity of the displacement ($d\lambda/dt$) is a centrally controllable parameter essentially affecting movement speed (Adamovich & Feldman, 1984; Feldman 1974 b; 1979; 1981). Recent studies have shown that the fastest one-joint movements to a final position and performed by a gradual, but very rapid shift of the EP at a constant velocity (Adamovich & Feldman, manuscript in preparation), and thus the central control signal achieves its final value long before the end of the mechanical movement (e.g., the shift for a 20° movement takes about

40 ms whereas the movement ends about 250 ms later). For reasons of simplicity, only initial and final positions of the AA borderline are considered during qualitative explanations of fast active motor performance.

Consider an isotonic flexion. Figure 14A shows initial equilibrium muscle length (x_1) and an initial position of the AA border. The primary tonic activity that supports equilibrium is determined by the distance s_1 from the x_1 to the border line. An intentional shift ($\lambda_1 \rightarrow \lambda_2$) of the border to the left results in an increase of the distance and consequently muscle activity. The first EMG burst provides the initial impulsive force for the movement. Its phase trajectory (the oval curve) is constrained to come towards the new AA border and leave the AA soon. Thus, the first EMG burst ends giving place to a "silent period" in the EMG. The load begins then to decelerate the movement. In this phase, the trajectory enters into the AA again, and the EMG resumes (the second EMG burst) and, finally, tonic EMG activity provides the balance of the load at a new position (x_2).

Figure 14B shows how the λ model functions in the case of positive EMG-gradient load. The load is initially balanced at a muscle length x_1 whereas the tonic stretch reflex threshold is equal to λ_1. Its rapid bias ($\lambda_1 \rightarrow \lambda_2$) gives rise to a shift of the AA border to the right. As a consequence, the initial tonic EMG activity disappears and the load begins to

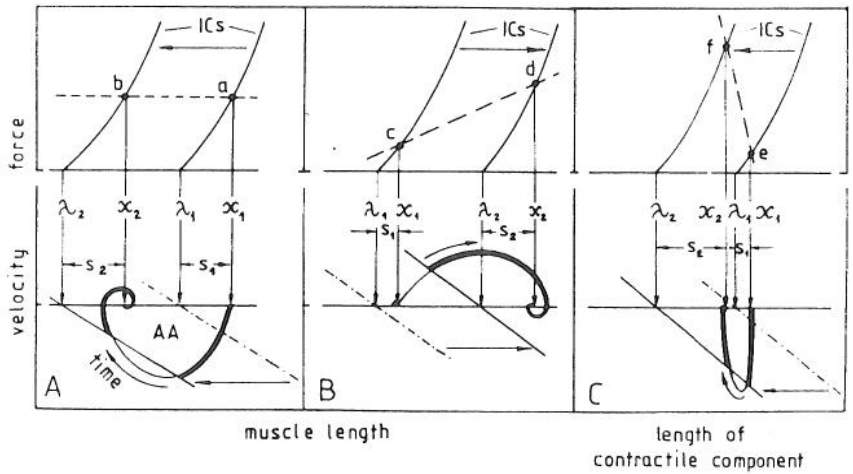


Fig. 14—The generation of EMG bursts during rapid motor performance: schematic representation with the use of force/length and velocity/length diagrams. A: isotonic flexion. B: extension against a load having a positive EMG gradient. C: isometric contraction. Horizontal arrows show shifts of: ICs; reflex thresholds ($\lambda_1 \rightarrow \lambda_2$); equilibrium positions ($x_1 \rightarrow x_2$); AA borders from an initial position (dashed-point line) to a final one (solid); equilibrium points (*a* to *b* in A, *c* to *d* in B and *e* to *f* in C). Horizontal distances s_1 and s_2 determine an initial and a final level of EMGs. Oval curves are phase movement trajectories; their thick sections show periods of muscle activation. Dashed lines: static load characteristics (A, B) and a force/length function for the series elastic component of the muscle (C). Note that the horizontal axis in C is the length of contractile component. See text.

stretch the muscle. The movement trajectory is directed towards the new AA and enters it soon. The EMG activity gradually increases during the muscle extension and finally the load is balanced at a new position x_2. Thereby the final tonic EMG level exceeds that at the initial position x_1 since $s_2 > s_1$.

Similar principles underlie the explanation of EMG patterns arising during isometric force exertion. To take into account the interaction between the two muscle components (passive series elastic and active contractile) the AA will be represented on the plane of variables contractile component length versus its derivative. Thereby, the threshold λ, the form of AA border and ICs, are slightly modified. For a qualitative consideration, however, we neglect the modifications. The series elastic component plays the role of a load for the active contractile component. An approximate characteristic of the load is shown in Figure 14C. Given a threshold λ_1, the load is balanced at a length x_1 of the contractile component. Central commands perform a rapid shift of the AA border, so that the distance from the initial length x_1 to the AA border increases, giving rise to additional α activation and muscle force. It is natural to suggest that the isometric force essentially exceeds that exerted during an isotonic flexion performed with the same central commands, and thus the active muscle component can contract at a rather high speed despite a small absolute contraction magnitude. Its trajectory first leaves and then enters again the new AA and, finally, comes toward a final EP—a new isometric muscle force is achieved (Figure 14C). On the whole the rapid isometric force exertion is associated with two EMG bursts, the first being more intensive than the second, a result consistent with experimental data (Gordon & Ghez, 1984).

For the above qualitative account, it has been assumed that the phase trajectories and EMGs are mutually coordinated: the first EMG burst provides movement acceleration while the "silent period" allows the load to execute a deceleration of the movement. Such a process is imaged on the phase plane by a bell-shaped curve as is shown in Figure 14A. The second EMG burst adds a small terminal overshoot to the curve.

To obtain deeper insight into the diagramatic method for explaining EMG patterns, the reader may wish to consider other examples (e.g., isotonic extensions or an unloading reflex).

4.12 *Trigger reactions*

The fact that the nervous system can specify parameter λ disregarding peripheral afferent signals does not mean that this kind of motor control is the only one constantly used. The independent (open-loop) central strategy may be inadequate in some cases, for example, in the task of postural fixation irrespective of load. The stretch reflex is unable to support a certain muscle length when the load magnitude varies, and the nervous system has, therefore, to adjust central commands to restore one and the same position. The use of peripheral information about the load is necessary for this process. Experimental data have suggested that

the nervous system estimates the necessary shift ($\Delta\lambda$) beforehand and performs the corrective reaction as soon as the load changes (Crago, Houk, & Hasan, 1976; Houk, 1976). Figure 15 illustrates the so-called "trigger reaction" strategy (Houk, 1976) in terms of the λ model. Command λ_1 provides position x_1 when the load is L_1, and command λ_2 does the same position when the load is L_2. The diagram predicts possible mistakes in the task of postural fixation if a subject triggers off inappropriate commands. For example, when command λ_1 is used when the load is L_2, a position x' instead of x is achieved. Similar mistakes have been observed (Crago et al., 1976).

The use of trigger reactions depends on the kind of motor task discussed above (one need not use them when postural fixation is unnecessary). However, sometimes they appear irrespective of the instruction given to the subject if, for example, external perturbations are excessive. Such reactions were observed during recording of ICs (Feldman, 1979; Gielen & Houk, 1984; Vincken, 1983), in particular, when an abrupt loading was used (Feldman, 1979). The arm took then a position that differs from that prescribed by an IC determined previously by the unloading method. The use of a soft gradual loading usually illiminated the trigger effects during recording of ICs (see Figure 42 of Feldman, 1979; cf. Vincken, 1983).

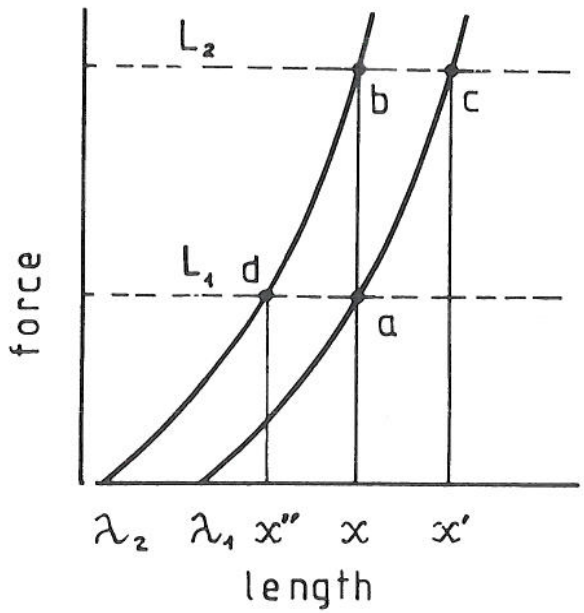


Fig. 15—Trigger strategy during postural fixation. To provide posture x when the load varies from L_1 to L_2 or vice versa it is necessary to shift correspondingly the reflex threshold (λ_1 to λ_2 or vice versa) and invariant characteristics (solid curves). Otherwise postural mistakes occur (x',x''); $a - d$: equilibrium points of which only two (a,b) correspond to correct performance.

There are plenty of neuronal structures that affect muscle activity, and always the λ model compels us to decide whether a given structure belongs to the muscle servo-loop, or executes its parameterization (e.g., modifies the λ). Probably skin reflexes and other non-proprioceptive reflexes are mediated by structures of the second class, which also includes the Ia-interneuron system of reciprocal inhibition of motoneurons (i.e, stretching a muscle can bias the stretch reflex threshold of antagonist muscle in the decerebrate cat (Feldman & Orlovsky, 1972; Matthews, 1959).

Muscle stretch effects called the "functional stretch reflex" (Phillips, 1969) and mediated by spinal interneurons and pyramidal tract cells (Wiesendanger & Miles, 1982) are probably trigger reactions, judging by their dependence on instruction (Hammond, 1960). Consequently, one may predict that pyramidal tract cells will sustain their activity during an unloading reflex under the instruction "do not intervene," but not if a restoration of the initial posture is required. The "klasp-knife" phenomenon (Rymer, Houk, & Crago, 1979) may also be referred to as a trigger reaction.

As a mass-spring model, the λ model has predicted the property of "equifinality," that is, in certain types of task temporal perturbations may not affect the final equilibrium position. The property has been found constant for a number of motor tasks (Bizzi, 1980; Feldman, 1966 b; Kelso, 1977; Kelso & Holt, 1980; Kelso & Tuller, 1983; Schmidt, 1982). Violations of equifinality in some cases (Schmidt, 1982) are, indeed, consistent with the model—trigger reactions or voluntary modifications of central commands in response, for example, to excessive perturbations, do not promote equifinality.

5.0 General Discussion

5.1 *Dynamic principles of the model*

The above considerations illustrate, in particular, the following aspects of motor performance.

(a) *The causal chain of events underlying voluntary motor control* (Figure 16). Central commands express themselves by subthreshold changes (ΔV) of motoneuron membrane potentials or, identically, in shifts of λ, IC and AA border. Generally speaking, this process is independent of the periphery (see, however, section on "Trigger reactions"). A consequence of the commands is an EP shift. This process is load dependent, that is, a change in the load also modifies the EP. EMG modifications are an effect of EP shifts but do not cause them. This shows an essential distinction between the λ model and the α models (Bizzi, 1980; Hogan, 1984) in which EP shifts are considered as a consequence of EMG modifications and thus cause and effect are inverted.

(b) *The absence of a central program for EMG bursts.* It is currently believed that EMG bursts and their parameters are an expression of a central program which undergoes a modification by afferent inputs. Such an idea underlies pulse-step or bang-bang control models for rapid eye, head and limb movements (Ghez & Martin, 1982; Hannaford, Lakshminarayanan & Stark, 1984; Robinson, 1964). The λ model claims the absence of a "central program" for EMG bursts or their time periods and amplitudes—an idea similar to that popularized by Kelso and Saltzman (1982). Central control signals only deal with shifts of the system's equilibrium. EMG bursts are a manifestation of dynamic processes that are evoked every time the current position of the system does not coincide with an equilibrium state. In the cases considered above, central control signals had a simple form (unidirectional changes in λ to certain final levels) but elicited complex EMG patterns. Neither amplitudes nor periods of EMGs were represented in the central control signals descending to motoneurons.

For comparison, it is appropriate to mention here the phenomenon of the generation of postsynaptic ion currents in nervous cells. The current depends on an electromotive force—the difference between actual membrane potential and equilibrium potential for a respective ion. There is no current at all if the potentials coincide. The direction of the current inverts when the membrane potential crosses its equilibrium value. These representations are, indeed, habitual for neurophysiologists, and it is unnecessary to believe that something else, instead of or apart from the electromotive force, evokes or produces a "program" for postsynaptic ion currents. The λ model suggests similar dynamic principles for motor processes: "muscular currents" or EMGs appear if the system's state differs from the equilibrium one. Central commands descending to α and γ motoneurons only affect the equilibrium state and thus execute an indirect control over muscle activity.

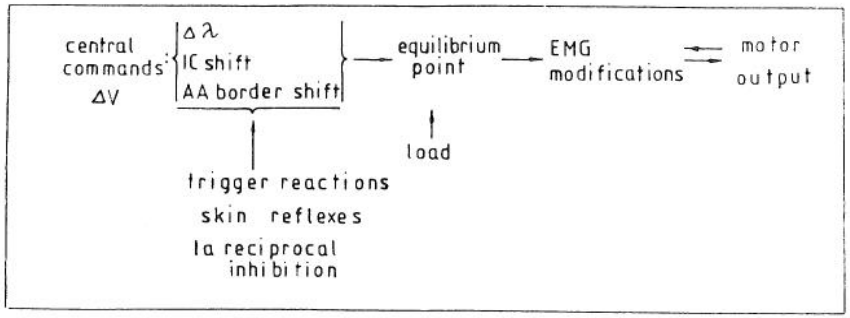


Fig. 16—The causal chain of events during intact motor performance. Voluntary movements are elicited by independent central commands biasing motoneurons' membrane potentials (ΔV). The commands are also expressed in shifts of the tonic stretch reflex threshold ($\Delta\lambda$), the invariant characteristic (IC) or the border of activation area (AA). The events result in a shift of the system's equilibrium point. Note that EMG modifications are the effect but not cause of the shift of the equilibrium point. In contrast, EMGs and motor output (kinematic variables) are mutually conditioned. Involuntary movements are elicited by load perturbations affecting the system's equilibrium point. There is an intermediate class of motor reactions conditioned directly or indirectly by peripheral afferent inputs that influence the same parameters the independent central commands do. It is suggested that trigger reactions to excessive perturbations, skin reflexes and effects of the interaction of flexor-extensor coupled muscles mediated by Ia-interneurons of reciprocal inhibition belong to the class.

(c) *Central parameterization of the stretch reflex as a basis for motor control*. The λ model does not exaggerate or underestimate the role of central and reflex (peripheral) factors in the origin of EMGs. Rather, the model takes all of them into account. For example, to exert an isometric muscle force, the nervous system decreases the λ, a measure of central commands. On the other hand, while doing so, it uses a reflex-conditioned IC and thus the isometric force exertion is, to the same degree, a reflex process.

Figure 14 shows motor effects which at first glance are contrary to the stretch reflex: the system generates a burst of EMG activity when the muscle is contracting (A) or elicits an EMG silent period when the muscle is stretching (B). Counterintuitive though it may seem, the model claims that these processes are not the result of suppression or elimination of the stretch reflex. To the contrary, the stretch reflex is *exploited* by its specific adaptation to central commands. This central strategy, reflex parameterization, is consequently considered a basis for motor control in the λ model, that is to say, reflex parameters (λ or μ), not a level of muscle activity, are the primary measure of central commands descending to motoneurons. It should be emphasized that the question of the measure of central motor commands cannot be resolved on the basis of currently popular methods of muscle deafferentation. The latter procedure destroys reflex systems: as a result, central commands are constrained to manifest themselves in unconditional activation of motoneurons, thus creating the impression that the same is true for intact motor control. The λ model claims that this impression is wrong (see also below).

5.2 *Comparison with Merton's Hypothesis*

There may be a tendency to equate the λ model with the γ drive hypothesis (Merton, 1953) which suggests that descending systems initially activate γ motoneurons to drive the movement. In this view, the γ-activity specifies the muscle length that the stretch reflex must follow-up irrespective, to a certain degree, of the load. Thus, there is the requirement (not corroborated experimentally) that the reflex has a significant gain or stiffness. In contrast, descending influences in the λ model can be directed to α and/or γ motoneurons to drive the movement; the central commands do not prescribe a concrete length or force to the muscle, but a stretch reflex threshold. Consequently only a relation between the variables, that is, an IC is specified. The IC has a moderate slope and thus the stretch reflex does not have to follow-up a certain muscle length. Rather, its task to bring the system to an equilibrium, load-dependent position (see also Houk, 1976).

Both models nevertheless proceed from the basic idea that the nervous system uses the stretch reflex to control movements. Concrete forms of the idea are quite different in the two models, not only because of the different roles played by the stretch reflex, but also because the λ model does not ascribe to the central-reflex dichotomy.

The servo-assistance hypothesis (Granit, 1970; Stein, 1974) stated the experimental fact that not only can γ motoneurons drive movements but so can direct inputs to α motoneurons (e.g., Vallbo, 1981). This step corrected one flaw in Merton's hypothesis. On the other hand, Merton's hypothesis had an elegance in that it provided a concrete principle for postural specification. Such elegance was lost in the later hypothesis.

5.3 *Deafferented muscles*

From the comments above, it follows that α models are inadequate for intact motor control organization. One may, however, create experimental α models by means of muscle deafferentation. No doubt, deafferented animals can satisfactorily perform many motor tasks (e.g., Bizzi et al., 1976). Nevertheless, an experimental α model is only a model: its mere existence, neither proves its relevance to intact motor control, nor rejects the λ model.

The two models belong to a class of mass-spring models that derives from the recognition that in certain tasks the motor system behaves in a way that is qualitatively similar to a simple physical system such as a spring with a load (cf. Kelso, Holt, Kugler & Turvey, 1980). The fact that spring-like properties remain, to a certain degree, after muscle deafferentation (e.g., Bizzi, 1980; see, however, Day & Mardsen, 1982) does not allow one to determine whether the α or λ model is a better account of intact motor control. At the same time, the experimental fact that the nervous system uses λ– (but not α–) invariant characteristics is a strong argument in favour of the λ model which posits that parameterization of the stretch reflex is a fundamental principle of intact motor control. Muscle deafferentation impairs the mechanism without removing the possibility of movement. Indeed, the analysis performed in this paper has predicted essential defects in the motor performance of deafferented animals. In particular, deafferented muscles are incapable of dealing with isoelectric loads or with those having EMG-positive gradients, because of stiffness deficits. In addition, there is a decrease in postural stability and a slowing of movement under deafferented conditions. In order to compensate for such stiffness deficit, the nervous system must co-activate agonist and antagonist muscles more often, even though the agonist muscles could perform the task sufficiently well alone if muscle innervation were intact. In all probability, the range of force and length attainable in deafferented muscles is narrowed. Thus, the conclusion reached as a result of deafferentation experiments, namely, that muscle afferent inputs are unnecessary for motor control (Hogan, 1984) seems wrong.

It has been illustrated above that the complex multiphasic EMG pattern (first EMG burst, then silent period followed by a secondary EMG burst) arising during rapid isotonic or isometric flexions (Figure 14) is the result, on one hand, of monophasic central commands (Δλ) descending to motoneurons and, on the other hand, of a significant dependence of EMG activity on kinematic variables (see also Ghez & Martin, 1982; Ter-

zuolo, Soechting, & Ranish, 1974). Hence, it follows that a part of the characteristic EMG pattern (silent period and secondary EMG burst) will be absent following muscle deafferentation, since only the monophasic central signal will activate motoneurons. Similar effects of muscle deafferentation have been observed by Terzuolo et al. (1974). In this connection, it is worth mentioning a recent report that multiphasic EMG patterns during human rapid wrist movements remain after functional (ischemic) muscle deafferentation as tested by the absence of phasic reflexes in *passive* muscles (Sanes & Jennings, 1984). In contrast, we found that at this stage of ischemia a sudden unloading of preliminary *active* muscles elicited a distinct reflex EMG reaction, a silent period (R. M. Abdusamatov, S. V. Adamovich, N. I. Burlachkova, & A. G. Feldman, unpublished observation). Thus, the functional deafferentation in the experiments of Sanes and Jennings was probably incomplete.[7]

5.4 *Phasic and tonic control*

A widespread opinion is that two independent systems perform motor control, one (phasic) producing movements and the other (tonic) specifying postures, with the whole movement pattern being a result of the combined action of the two control systems. The first pushes the limb to the desired position and the second holds it there (Ghez & Martin, 1982; Robinson, 1964). In contrast, the EP-hypothesis when understood correctly, expresses itself in the fact that there is a single mechanism for posture and movement: a shift in parameter λ with a subsequent EP—shift is a necessary and sufficient condition for eliciting movement.

Experimental data, especially those concerning the regulation of dynamic sensitivity of muscle spindle afferents (e.g., Matthews, 1964) make it indeed necessary to propose the existence of a phasic control system. The λ model associates phasic control with the regulation of dynamic sensitivity, μ, affecting the dependence of the stretch reflex threshold on velocity (see section "Muscle activation area"). The phasic system thus plays the role of modulating the postural shifts produced by the λ-control system. In other words, the phasic system may accelerate or decelerate transfers to equilibrium points but is itself unable to initiate movement.

6.0 Concluding Remarks

From the foregoing remarks, it does not follow that the λ model is the only possible motor control scheme and no other alternative exists. It goes without saying that many aspects of the model need further elaboration and detail. At the same time, the explanatory and predictive power of the model is rather high, and provides a stimulus to generating possible alternative hypotheses. Minimally, for intact motor control such hypotheses must (1) provide independent measure(s) of central control signals for driving the movement; (2) take into account the dependence of muscle activation on kinematic and command variables; (3) recognize a fundamental constraint in motor control, namely, that the nervous system produces EMG modifications, muscle contractions and limb movements only *indirectly*, through the control over parameters specifying the equilibrium state of the motor system; and (4) explain at least qualitatively, motor performance under isotonic, isoelectric and isometric loading conditions.

NOTES

1. The spring analogue in Bizzi's version was referred not to the integral system as specified in (b) but to the active muscle alone. Different versions of the EP concept are discussed in the section on "Equilibrium points." Concept (d) and its reformulations are discussed elsewhere (Adamovich & Feldman; Abdusamatov & Feldman; Adamovich & Feldman: manuscripts in publication).
2. This and the following section devoted to the role of motoneuronal membrane potentials and the $\alpha - \gamma -$ linkage in motor control in the context of the λ model may be missed during the first reading.
3. Stiffness k affects the system's damping (e.g., Partridge, 1972; Geilen & Houk, 1984), and thus the form and duration of motor transfer processes. Such issues are not much discussed here (see Feldman, 1979).
4. Rapid movements make heightened requirements on muscle stiffness especially if it is necessary to exclude meaningful terminal oscillations and overshoots. The nonlinear form of ICs may effectively meet the requirements: the nervous system can use segments of ICs having higher stiffness or co-activate agonist and antagonist muscles to increase its reserve (Feldman, 1980; cf. Partridge, 1984; see also Adamovich & Feldman, manuscript in preparation).
5. and, possibly, acceleration, which is not considered here.
6. The substitution of λ^* instead of λ allows one to generalize also relation (8) for the motoneruon to dynamics.
7. Incidentally, motoneurons as generators of impulses may reveal, even after deafferentation, a tendency to oscillations or burst activity in response to a monotonous increasing input but it is unlikely to call this phenomenon a "central program."

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Submitted August 15, 1985
Revision submitted December 19, 1985